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Detectability of Granger causality for subsampled continuous-time neurophysiological processes

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HIGHLIGHTS

- A "CTVAR" model for Neurophysiological processes is proposed.
- Subsampling analysis based on an exact analytic solution of the model is performed.
- Interactions between timescales of signal delay and sampling frequency are revealed.
- GC detectability decays exponentially for sample intervals beyond causal delay time.
- "Black spots" and "sweet spots" in GC detectability are discovered.

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ABSTRACT

Background: Granger causality is well established within the neurosciences for inference of directed functional connectivity from neurophysiological data. These data usually consist of time series which subsample a continuous-time biophysiological process. While it is well known that subsampling can lead to imputation of spurious causal connections where none exist, less is known about the effects of subsampling on the ability to reliably *detect* causal connections which *do* exist.
New method: We present a theoretical analysis of the effects of subsampling on Granger-causal inference. Neurophysiological processes typically feature signal propagation delays on multiple time scales; accordingly, we base our analysis on a distributed-lag, continuous-time stochastic model, and consider Granger causality in continuous time at finite prediction horizons. Via exact analytical solutions, we identify relationships among sampling frequency, underlying causal time scales and detectability of causalities.
Results: We reveal complex interactions between the time scale(s) of neural signal propagation and sampling frequency. We demonstrate that detectability decays exponentially as the sample time interval increases beyond causal delay times, identify detectability "black spots" and "sweet spots", and show that downsampling may potentially improve detectability. We also demonstrate that the invariance of Granger causality under causal, invertible filtering fails at finite prediction horizons, with particular implications for inference of Granger causality from fMRI data.
Comparison with existing methods: Our analysis emphasises that sampling rates for causal analysis of neurophysiological time series should be informed by domain-specific time scales, and that state-space modelling should be preferred to purely autoregressive modelling.
Conclusions: On the basis of a very general model that captures the structure of neurophysiological processes, we are able to help identify confounds, and offer practical insights, for successful detection of causal connectivity from neurophysiological recordings.

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1. Introduction

Neurophysiological recordings are generally obtained by sampling, at regular discrete time intervals, a continuous-time analogue signal associated with some underlying biophysiological processes. Thus, for example, electroencephalography (EEG) records electrical activity arising from ionic current flows in the brain, magnetoencephalography (MEG) records the weak magnetic fields produced by neuronal currents, while functional magnetic resonance imaging (fMRI) measures changes in blood oxygenation level associated with neural activity (Logothetis et al., 2001). Even spike train recordings are typically derived from a continuous analogue measurement of cellular membrane potentials.

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Wiener–Granger causality (Wiener, 1956; Granger, 1963, 1969, 1981; Geweke, 1982) – henceforth just Granger causality, or GC – is a popular technique for inferring directed functional connectivity of the underlying process in the neurosciences (Seth et al., 2015), from (discrete-time) subsampled[1] process. Granger causality is premised on a notion of causality whereby cause (a) precedes effect and (b) contains unique information about effect. This idea is commonly (but not exclusively) operationalised within a vector autoregressive (VAR) modelling framework. At this point, we recognise that the ascription of a "causal" interpretation to GC is clearly problematic to some. Our view is that Granger causality represents *a* rather than *the* notion of causality, an avowedly *statistical*, as opposed, e.g., to "interventionist" notions (Pearl, 2009). As such, its strengths and limitations have been widely discussed [see e.g. Valdes-Sosa et al. (2011) for a review of the issues involved with regard to biophysical modelling; also Chicharro and Panzeri (2014)], and we do not enter that debate here. We remark, however, that Granger causality also has a principled interpretation – through its intimate relationship (Barnett et al., 2009; Barnett and Bossomaier, 2013) with the information-theoretic *transfer entropy* (Schreiber, 2000; Paluš et al., 2001) – as a measure of *information transfer*, and we generally prefer this interpretation (Lizier and Prokopenko, 2010), particularly with regard to functional connectivity analysis.

Problems associated with Granger-causal inference from subsampled (or otherwise aggregated) time series have long been noted (Granger, 1969; Sims, 1971; Wei, 1981; Marcellino, 1999; Breitung and Swanson, 2002). Specifically, it has been observed that subsampling may distort GC values. This may be considered especially problematic in two distinct aspects:

(i) *Spurious causality*, where GC is absent at the finer time scale, but non-zero for the subsampled process (Comte and Renault, 1996; Renault et al., 1998; Breitung and Swanson, 2002; McCrorie and Chambers, 2006; Solo, 2007, 2016), and
(ii) *Undetectable causality*, where GC is present at the finer time scale, but zero (or too small to detect reliably) for the subsampled process (Barnett and Seth, 2011; Seth et al., 2013; Zhou et al., 2014).

Subsampling may, in addition, distort the *relative strengths* of causalities (Solo, 2016).

Solo (2007, 2016), drawing on previous work by Caines (1976), distinguishes between the conventional "weak" causality and "strong" causality (see Section 2.1), and concludes that only strong causality remains undistorted by subsampling. Seth et al. (2013) demonstrate that GC inference from fMRI data may be severely degraded by the sample rates, slow in comparison to underlying neural time scales, of fMRI recording technologies. More recently, Zhou et al. (2014) report oscillations in estimated causalities with varying sampling frequency, with causal estimates almost vanishing at some frequencies, as well as inference of spurious causalities.

Although Granger himself was clearly concerned about the detectability problem – in Granger (1969) he notes that "[...] a simple causal mechanism can appear to be a feedback mechanism[2] if the sampling period for the data is so long that details of causality cannot be picked out" – subsequent studies have concentrated mostly on spurious causality. Here we investigate detectability: specifically, we examine how the relationship between the underlying time scale of causal mechanisms and the sampling time scale mediates the distortion of (non-zero) Granger causalities, and how this distortion impacts on statistical inference of Granger causality from empirical data. We discuss the implications of our results with regard to the successful inference of Granger causalities at the structural (neural) level, from neurophysiological recordings.

1.1. Contributions of this study

A significant feature of the neuronal systems underlying such measurements is the potential range of signal propagation delays due to variation in biophysical parameters such as axonal length, diameter, conduction velocity and myelination (Miller, 1994; Budd and Kisvárday, 2012; Caminiti et al., 2013). Here we model the underlying analogue signal as a stochastic linear autoregression in continuous time. Unlike prevailing continuous-time stochastic process models in the neurosciences, our model accommodates distributed lags on arbitrary time scales, and is thus able to reflect variability of signal propagation delays. This leads, via consideration of prediction at *finite* time horizons, to a novel and intuitive definition of Granger causality at multiple time scales for continuous-time processes. In contrast to previous work on continuous-time Granger causality, in which various statistical (non)causality test criteria have been proposed, our definition is *quantitative*, furnishing a Granger–Geweke measure with an information-theoretic interpretation.

Using discrete-time VAR modelling, we then analyse the properties of processes obtained by subsampling the temporally multiscale continuous-time process, and relate the spectral and causal properties of the subsampled process to those of the underlying continuous-time model. Having defined continuous-time, finite-horizon GC – which represents a target for statistical analysis – we investigate the extent to which it may be inferred, and in particular *detected*, by discrete-time VAR analysis of the subsampled processes.

We focus on the practical questions of the feasibility and reliability of causal inference on sampling frequency and the (dominant) time scale of causal feedback in the generative process. We investigate in detail the relationship between sampling frequency and the quality of causal inference via a fully analytic solution of a minimal, but non-trivial, bivariate model in continuous time, with finite causal delay.

On the basis of our theoretical and empirical analysis, we identify critical relationships between causal delay, sampling interval and detectability of Granger causality. These include exponential decay of subsampled Granger causalities with increasing sampling interval, resonance between sampling frequency and causal delay frequency, potential detectability "black spots", and the existence of a non-zero optimal sampling interval (i.e., detectability may sometimes be improved by downsampling). We also discover a hitherto unremarked non-invariance of finite-horizon/multistep GC under causal, invertible filtering (in contrast with the known invariance of single-step discrete-time GC).

Finally, we discuss the implications of our findings for Granger-causal inference of neural functional relationships from neurophysiological recordings under various technologies – including fMRI, which continues to generate controversy.

1.2. Organisation

The paper is organised as follows: in Section 2 we review essential aspects of the theory of VAR processes and Granger causality in discrete time. In Section 3 we introduce CTVAR (continuous-time vector autoregressive) processes as

[1] The term "subsample" refers throughout to sampling of a discrete- or continuous-time process at *regular intervals*. We reserve the term "downsample" for the further subsampling of an already-sampled discrete-time process.

[2] Here, by "feedback mechanism", Granger refers to *contemporaneous* feedback between time series [Geweke (1982) terms this "instantaneous feedback"], as opposed to *time-delayed* feedback, which in his theory underpins "causal mechanism"; see Section 2.1 for details.

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