Misunderstandings regarding the application of Granger causality in neuroscience

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August, 2017

Stokes and Purdon (1) raise several concerns about the use of Granger-Geweke causality (GGC) analysis in neuroscience. They make two primary claims:

1. That GGC estimates may be severely biased or of high variance, and
2. That GGC fails to reveal the full structural/causal mechanisms of a system.

Unfortunately, these claims rest, respectively, on an incomplete evaluation of the literature and a misconception about what GGC can be said to measure.

Stokes and Purdon explain how bias and variance in GGC estimation arise from the use of separate, independent full and reduced regressions. However, this problem has long been recognised (2, 3) and, moreover, has already been solved by methods which derive GGC from a single full regression. These methods effectively calculate reduced model parameters from the full model via factorisation of the spectral density matrix. Published approaches (also implemented in freely-available software) include Wilson's frequency-domain algorithm (4), Whittle's time-domain algorithm (3), and a state-space method involving solution of a discrete-time algebraic Riccati equation (5). Thus, the source of bias and variance discussed in (1) has already been resolved (see also 6). We note that (1) erroneously state that "Barnett and Seth [...] have proposed fitting the reduced model and using it to directly compute the spectral components ..." whereas, as mentioned, we derive GGC from a single full regression (3).

Stokes and Purdon then note that GGC reflects a combination of 'transmitter' and 'channel' dynamics, and is independent of 'receiver' dynamics. This independence has also been previously identified; it follows directly from the invariance of GGC under certain affine transformations (7). Stokes and Purdon argue that this runs "counter to intuitive notions of causality intended to explain observed effects" since, as they put it, "neuroscientists seek to determine the mechanisms that produce 'effects' within a neural system or circuit as a function of inputs or 'causes' observed at other locations". However, this perspective is more closely aligned with approaches such as dynamic causal modelling (DCM)—usually characterised as *effective connectivity*—which attempt to find the optimal mechanistic (circuit level) description that explains observed data. GGC, by contrast, models statistical dependencies among observed responses and is therefore a measure of (directed) *functional connectivity* (8). Essentially, the distinction is between making inferences about an underlying *physical causal mechanism* (DCM) and making inferences about *directed information flow* (GGC; 9). Both address valid questions.

Our view is that the real problems associated with GGC analysis of neurophysiological data reside elsewhere: with issues of stationarity, linearity and exogenous influences, as noted in (1), but also with the noise, sampling rates and temporal/spatial aggregation engendered by neural data acquisition (10).

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