Is Granger Causality a Viable Technique for Analyzing fMRI Data?

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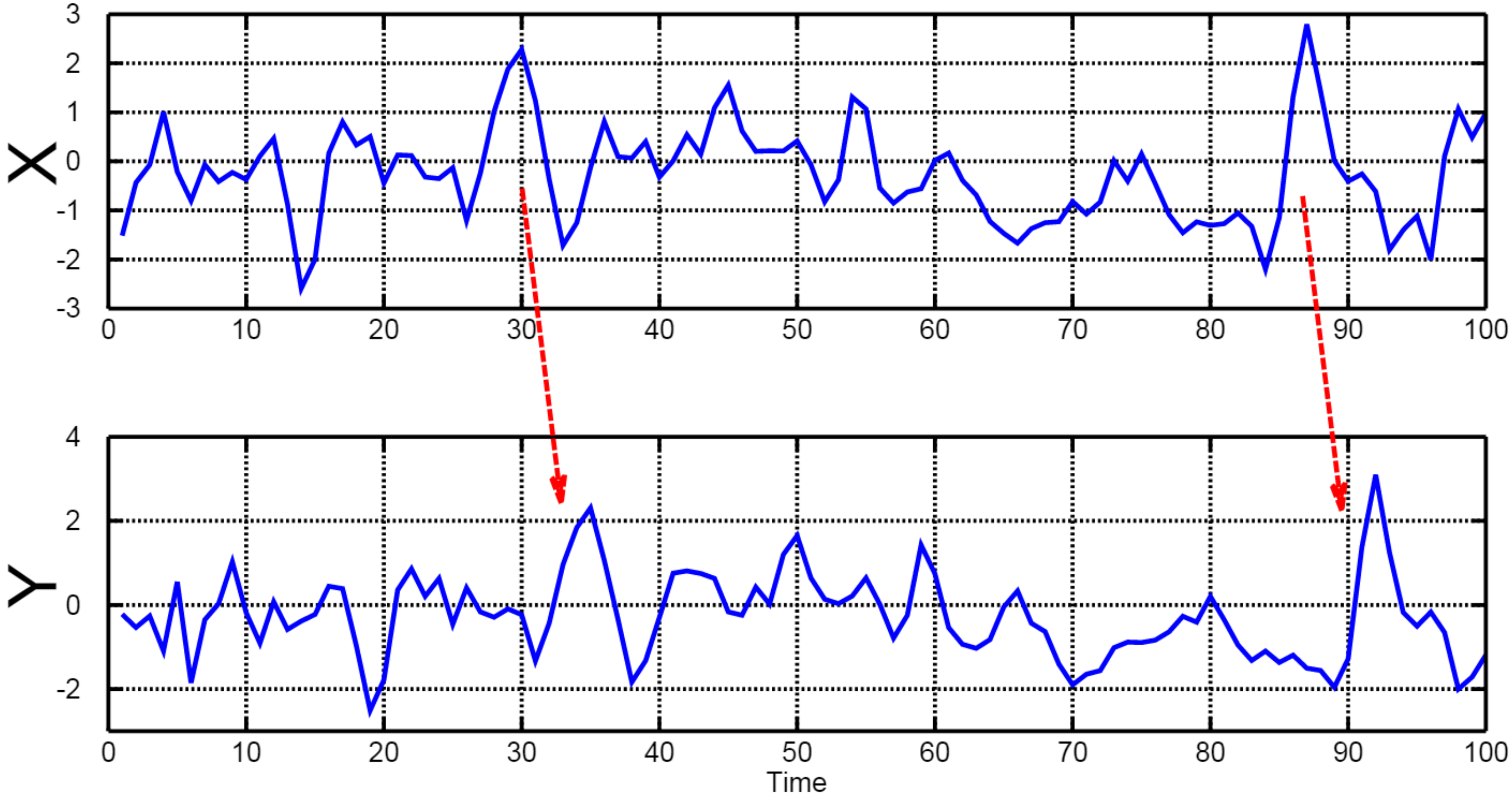
Background



- Granger causality
  - Time-delayed correlation of two time-series
- Problems
  - Could be reversed or common cause if latency
  - Not for nonlinear relationships
  - Isn't multivariable
  - True causality may not be so simple

Background

- Neural GC can be inferred from e-phys GC [6,7]
- At the group level, strong correlation between fMRI GC and neural GC [25]
- Rather than magnitude of GC, change across experimental conditions may be more important [11,26,27]

Goals

1. Is there a relationship between fMRI GC and neural activity GC?
2. How to infer neural GC from HRF GC?

- Whereas typical GC studies look at network inference, this attempts to check if fMRI level GC translates to neural level GC

(1.) Are changes in GC at fMRI level related to neural level?

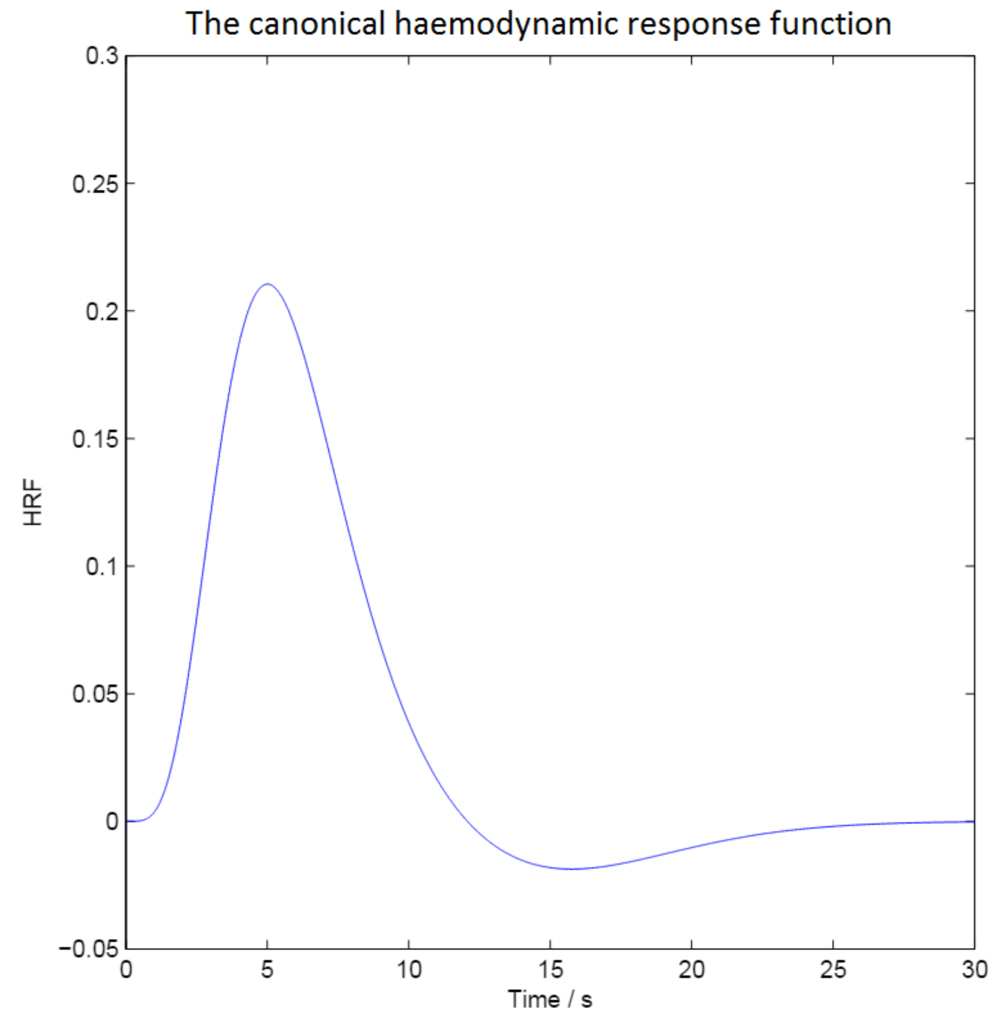
- Actual neural activity cannot be measured, so must be simulated
- Simulated neural time series

$$\begin{pmatrix} X_t \\ Y_t \end{pmatrix} = \begin{pmatrix} a & b \\ c & d \end{pmatrix} \begin{pmatrix} X_{t-1} \\ Y_{t-1} \end{pmatrix} + \begin{pmatrix} \varepsilon_t \\ \eta_t \end{pmatrix}$$

  - Noise terms ε_t and η_t are independent with unit variance
  - Unidirectional coupling: $b = 0$
  - Bidirectional coupling

(1.) Are changes in GC at fMRI level related to neural level?

- Simulated fMRI data
  - Neural data convolved with canonical hemodynamic response function (HRF) to simulate hemodynamic activity pattern
  - Downsampled to typical fMRI temporal resolution
  - Added Gaussian noise

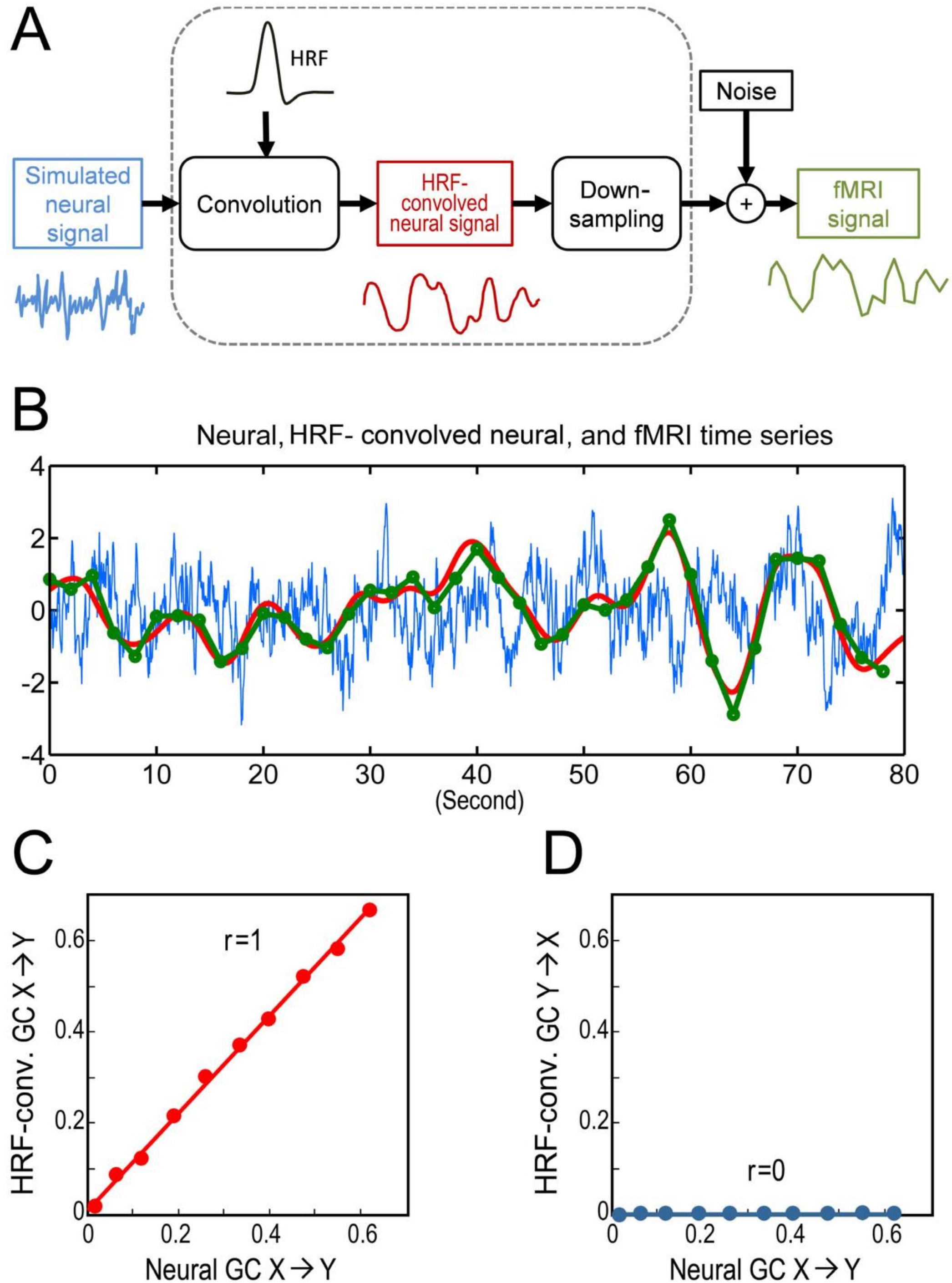


(1.) Are changes in GC at fMRI level related to neural level?

- For two time series X_t and Y_t, estimate GC with autoregressive models

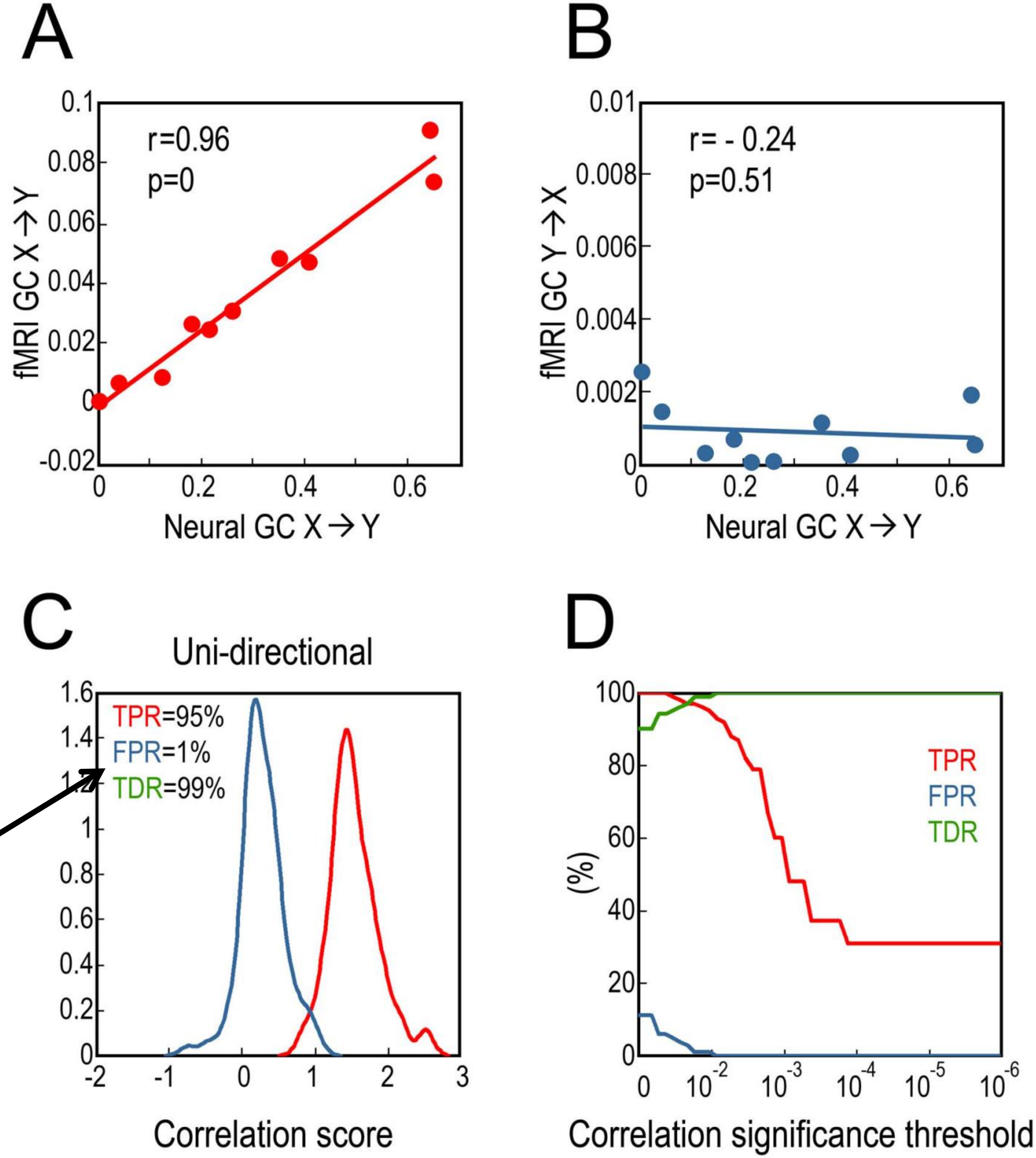
$$X_t = \sum_{j=1}^{\infty} a_{1j} X_{t-j} + \varepsilon_{1t}$$

$$Y_t = \sum_{j=1}^{\infty} d_{1j} Y_{t-j} + \eta_{1t}$$



Chose 50ms temporal resolution (TR); true delays range from 10s-100s of ms

Rates given for significance $p = 0.01$



Should be reported on other graph since colors misleading

Rates given for significance $p = 0.01$

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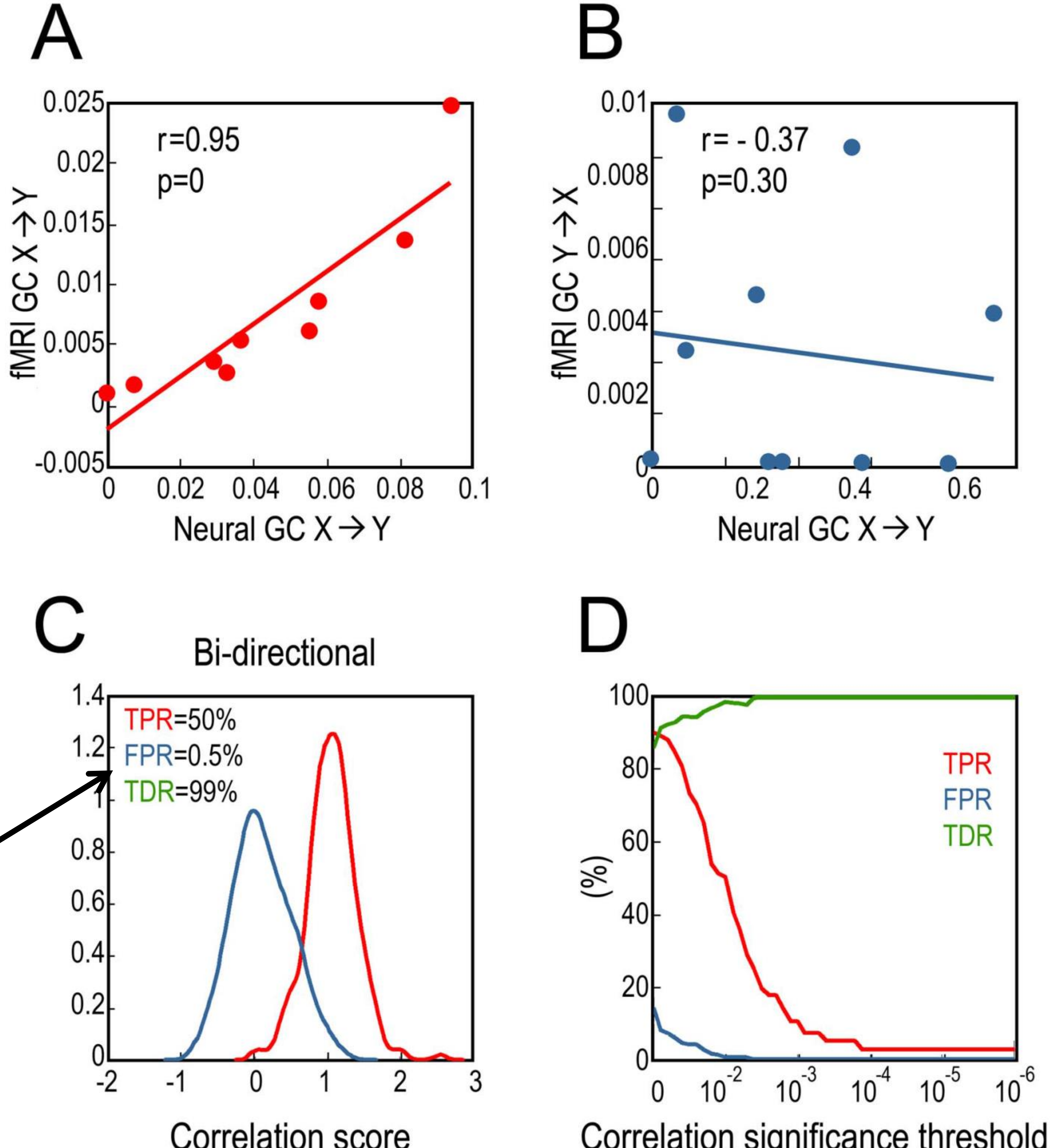


(1.) Are changes in GC at fMRI level related to neural level?

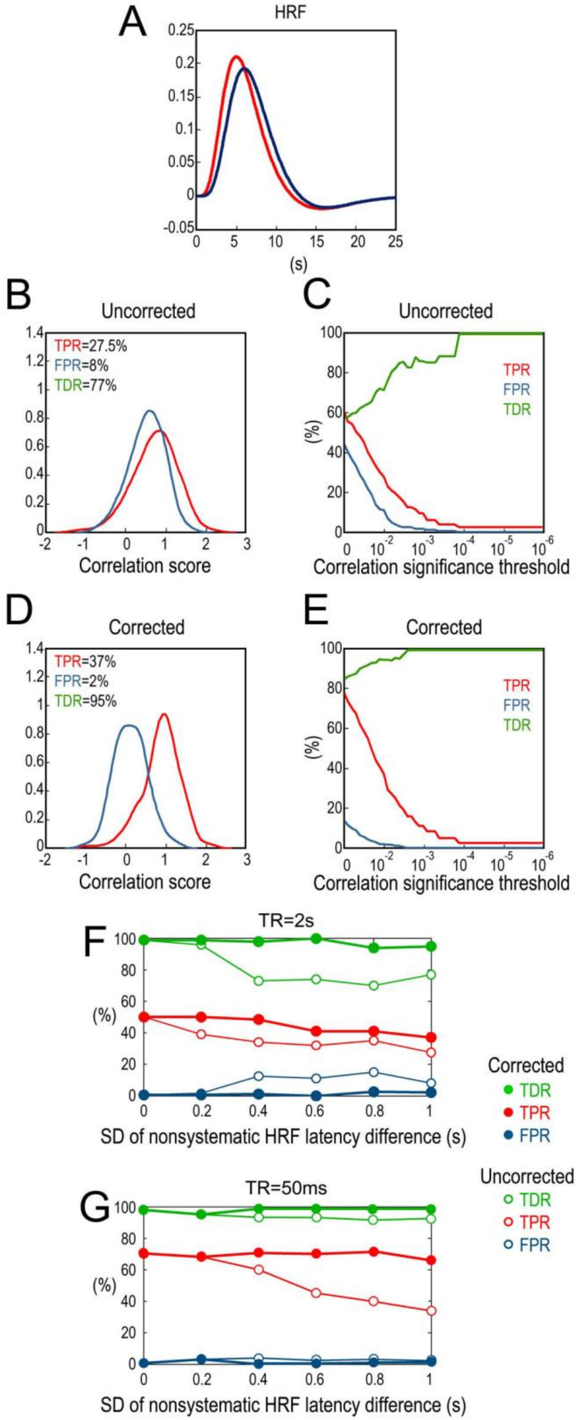
- Yes
  - For unidirectional coupling, fMRI level GC correlates with neural level GC, for any TR and measurement noise < 40% (20% is typical)
    - Slope near 1
    - $r = 0.95$
  - Similar results for bidirectional coupling

(2.) How to infer neural GC from HRF GC?

- Since detection is binary, spurious GC detected at fMRI level cannot be prevented
- Considering GC change given perturbation is more meaningful in neural terms

Problems with fMRI GC

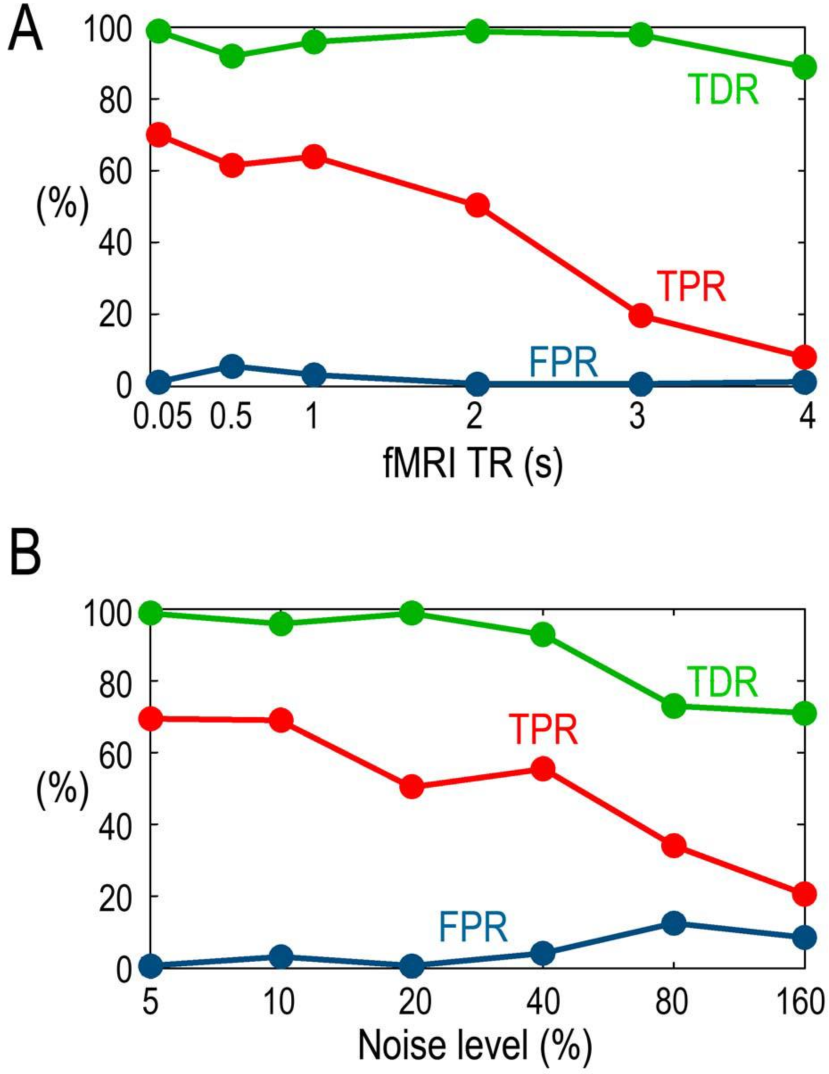
a) Latency in HRF
b) Low-sampling rates
c) Noise
d) “Third variable” problem



Problems with fMRI GC

a) Aforementioned problem about GC: if latency of X > Y, then direction of causality may be reversed
   - Latency difference increases FPR
   - Possible to preserve monotonicity by trading off FPR
   - Possibly estimate and correct for latency

Problems with fMRI GC

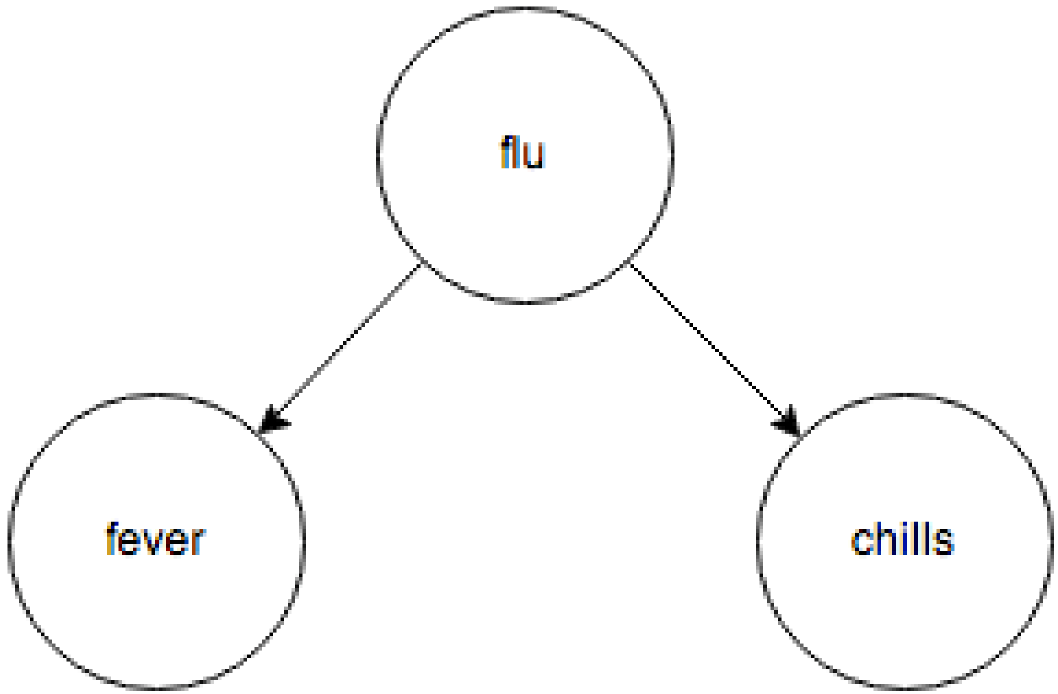


b) Even with 4s TR, TDR is 90%

c) TDR begins to drop with noise level > 40%
   - 20% noise is realistic [11,25]

Problems with fMRI GC

d) "Third variable" problem



Conclusions

- fMRI*HRF GC correlates monotonically with neural GC
- This monotonicity is positive correlation
- Decline in monotonicity due to HRF latency can be recovered with latency corrections