



EEG Connectivity Analysis

34th EEGLAB Workshop, UCSD

Nov 20, 2022

John Iversen

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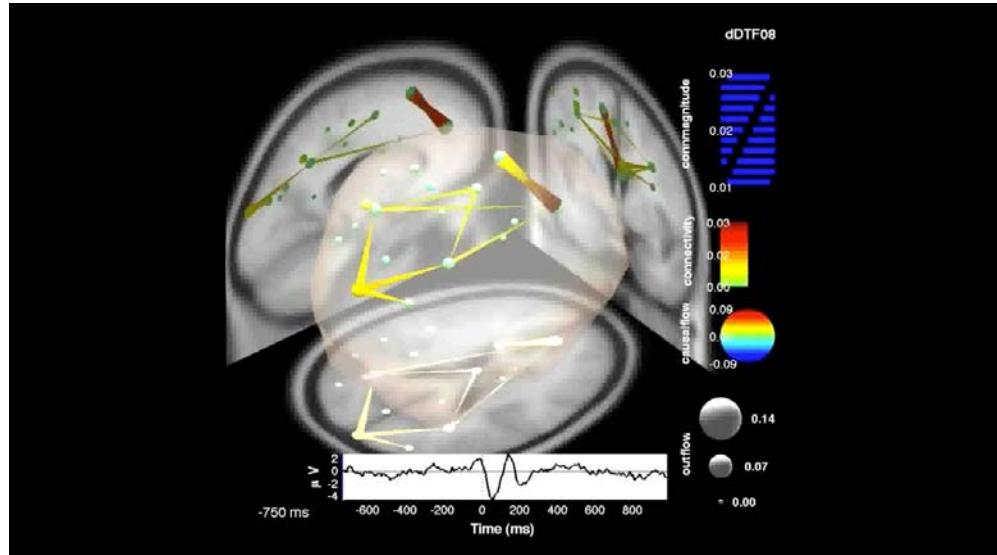
The Dynamic Brain

- A key goal: To model temporal changes in neural **dynamics** and **information flow** that **index** and **predict** task-relevant changes in cognitive state and behavior

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Modeling Brain Connectivity

- Model-based approaches mitigate the ‘curse of dimensionality’ by making some assumptions about the structure, dynamics, or statistics of the system under observation

Box and Draper (1987):

“Essentially, all models are wrong, but some are useful [...] the practical question is how wrong do they have to be to not be useful”

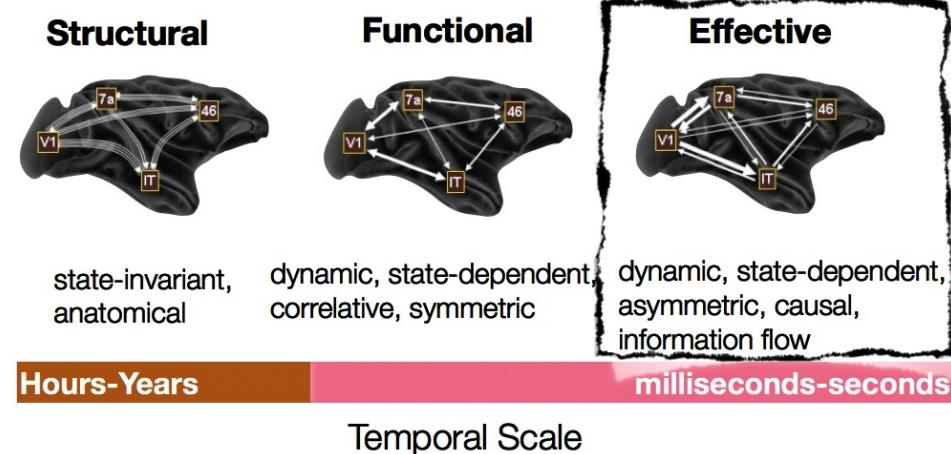


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Large-scale brain connectivity approaches

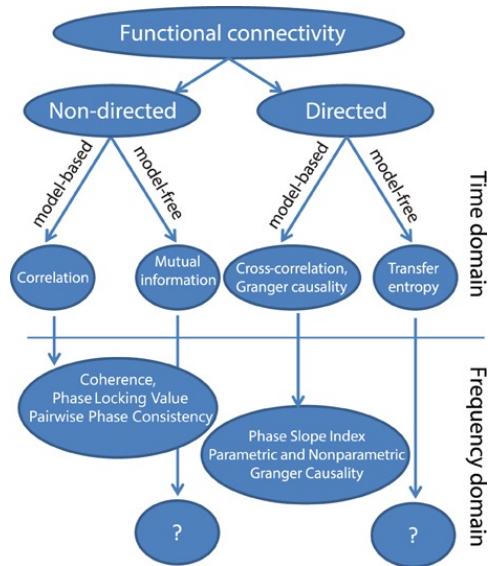
(Bullmore and Sporns, *Nature*, 2009)



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A taxonomy of connectivity measures



Bastos AM, Schoffelen J-M: A Tutorial Review of Functional Connectivity Analysis Methods and Their Interpretational Pitfalls. *Front Sys Neurosci* 2016, 9:413.

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Simple correlation approach:

Coherence Analysis

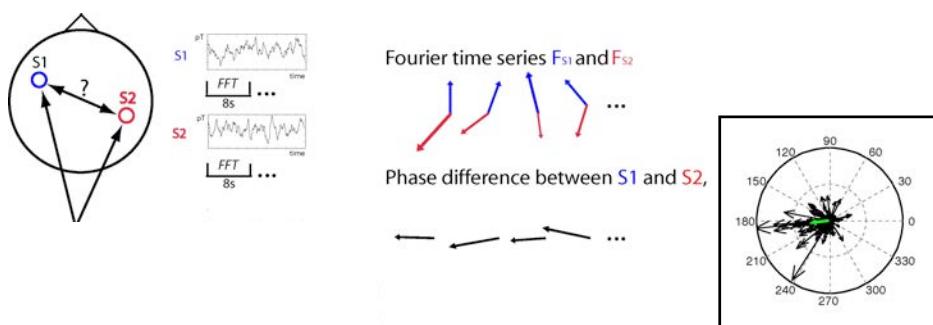
- Goal: How much do two signals resemble each other?
- Coherence = complex version of correlation: how similar are power and phase at each frequency?
- Variant: phase coherence (phase locking, etc.) considers only phase similarity, ignoring power
 - Regular coherence is simply a power-weighted phase coherence
 - Inter-trial coherence is useful!
- NOTE: For understanding connectivity between regions, *channel* coherence is a poor choice due to volume conduction. For IC connectivity, directional, 'causal' measures of connectivity have been developed (See SIFT lecture).

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Coherence

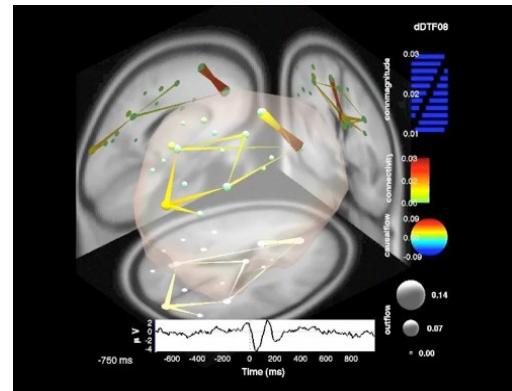
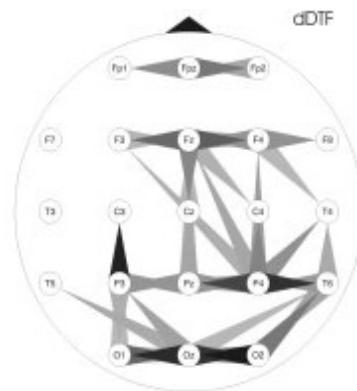
$$C(f,t) \propto \sum_{k=\text{trials}} F1_k(f,t) \overline{F2_k(f,t)} \\ a_1 e^{i\theta_1} a_2 e^{-i\theta_2} \propto e^{i(\theta_1 - \theta_2)}$$



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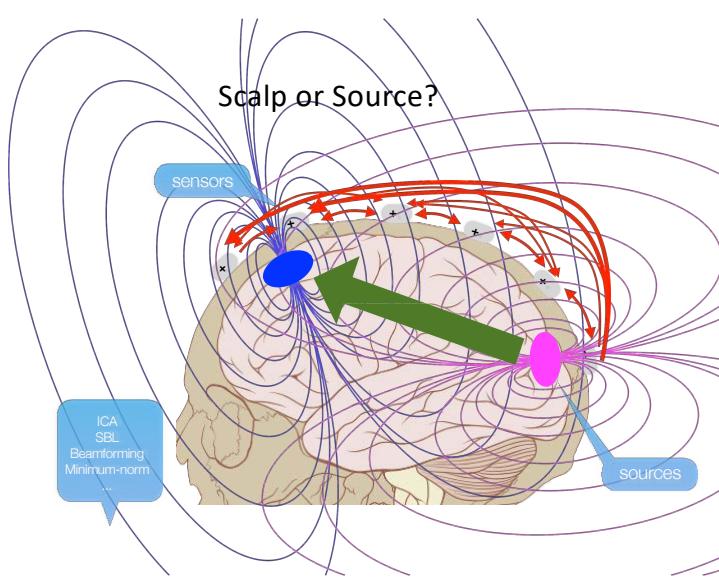
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Scalp or Source?



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Scalp or Source?



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SIFT
Source Information Flow Toolbox
<http://sccn.ucsd.edu/wiki/SIFT>
Mullen, et al., *Journal of Neuroscience Methods* (in prep, 2012)
Mullen, et al., *Society for Neuroscience*, 2010
Delorme, Mullen, Kothe et al., *Computational Intelligence and Neuroscience*, vol 12, 2011

- A toolbox for (source-space) electrophysiological information flow and causality analysis (single- or multi-subject) integrated into the EEGLAB software environment.
- Emphasis on vector autoregression and time-frequency domain approaches
- Standard and novel interactive visualization methods for exploratory analysis of connectivity across time, frequency, and spatial location

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Tim Mullen

QUSP is now **intheon**

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The problem of spurious connectivity

Coherency

$$C_{ij}(f) = \frac{S_{ij}(f)}{\sqrt{S_{ii}(f)S_{jj}(f)}}$$

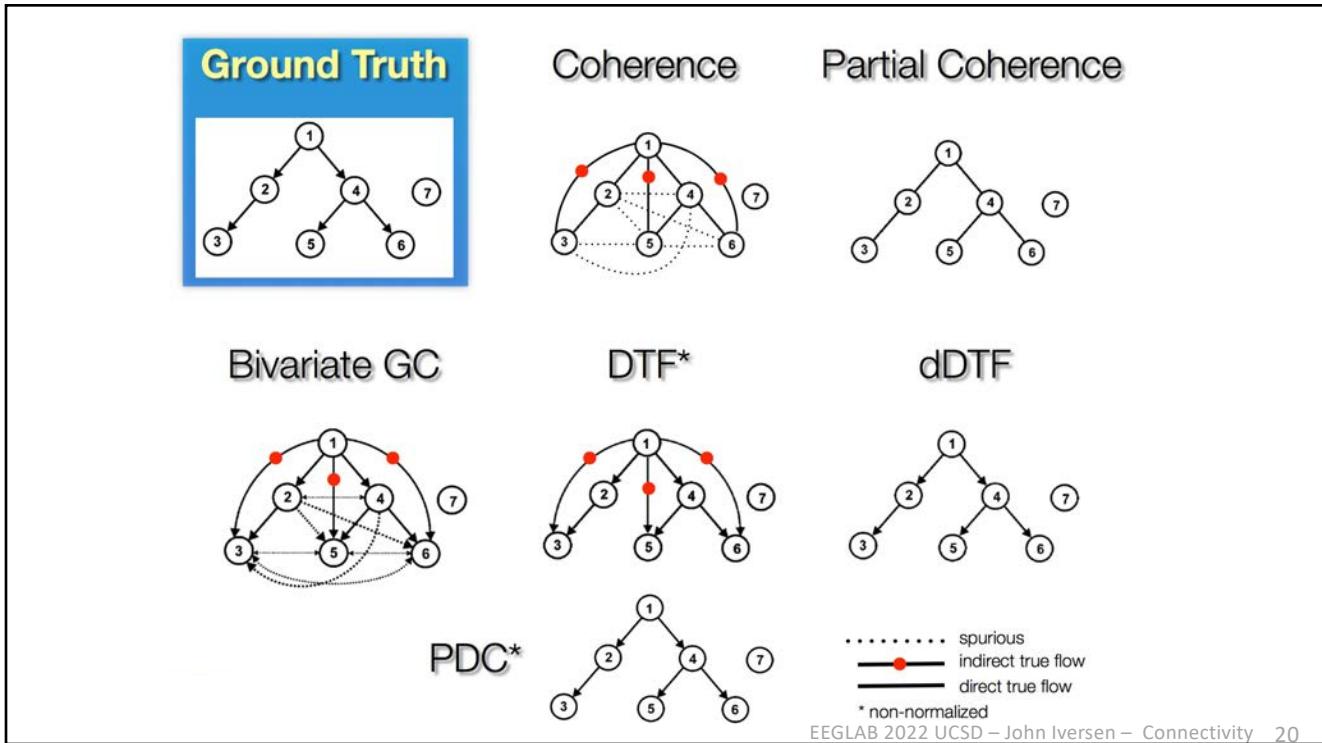
(Bendat and Piersol, 1986)

Kus, 2004

Bivariate measures, such as coherence (but also original GC), find spurious connections between nodes if they share a common input.

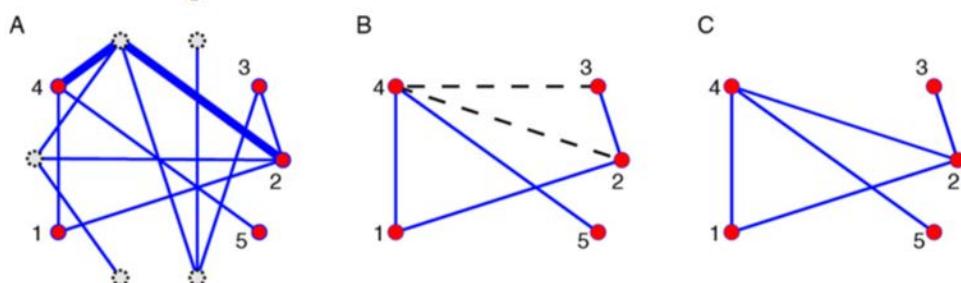
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A deeper problem – unobserved nodes



With EEG, it's unavoidable that there will be contributing network nodes (e.g. thalamus) that we cannot observe.

We also can't be sure ICA will identify all important sources...

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Granger Causality



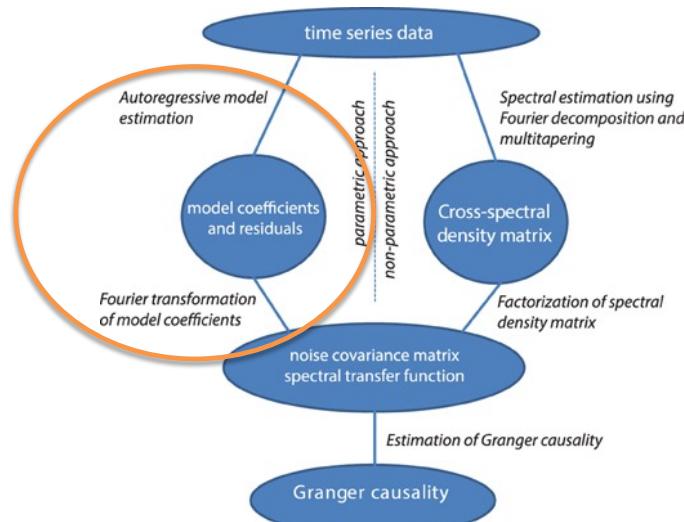
- A measure of *statistical causality* based on prediction.
- Widely used in time-series econometrics.
- Nobel Prize in economics, 2003.

If a signal A causes a signal B, then knowledge of the past of both A and B should improve the predictability of B, as compared to knowledge of B alone.

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Calculation of GC



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Granger Causality

- First introduced by Wiener (1958). Later reformulated by Granger (1969) in the context of linear stochastic autoregressive models
- Relies on two assumptions:

Granger Causality Axioms

- Causes should precede their effects in time (Temporal Precedence)
- Information in a cause's past should improve the prediction of the effect, above and beyond the information contained in past of the effect (and other measured variables)

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Granger Causality

- Granger (1969) quantified this definition for **bivariate** processes in the form of an F-ratio:

$$F_{X_1 \leftarrow X_2} = \ln \left(\frac{\text{var}(\tilde{E}_1)}{\text{var}(E_1)} \right) = \ln \left(\frac{\text{var}(X_1(t) | X_1(\cdot))}{\text{var}(X_1(t) | X_1(\cdot), X_2(\cdot))} \right)$$

full model

- Alternately, for a **multivariate interpretation** we can fit a single MVAR model to all channels and apply the following definition:

Definition 1

X_j granger-causes X_i conditioned on all other variables in \mathbf{X}
 if and only if $A_{ij}(k) > 0$ for some lag $k \in \{1, \dots, p\}$

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Fundamentals: Autoregressive Models

Goal: Predict future values of a data time series (EEG signal) from its past.

$$x(t) = \sum_{\tau=1}^p a(\tau)x(t-\tau) + e(t)$$

weighted sum of past values error

Example: order $p = 2$:

$$x(t) = a(1)x(t-1) + a(2)x(t-2) + e(t)$$

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AR Models (prediction of future of a signal by its past)

$$x_1(t) = \sum_{\tau=1}^p a(\tau)x_1(t-\tau) + e_1(t)$$

$$x_2(t) = \sum_{\tau=1}^p b(\tau)x_2(t-\tau) + e_2(t)$$

VAR Models (prediction of future of a signal by its past + the other signal's past)

$$x_{1|2}(t) = \sum_{\tau=1}^p c(\tau)x_1(t-\tau) + \sum_{\tau=1}^p d(\tau)x_2(t-\tau) + e_{1|2}(t)$$

$$GC_{2 \rightarrow 1} = \ln \frac{\text{var}(e_1)}{\text{var}(e_{1|2})}$$

$$\approx \ln(1)=0$$

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AR Models (prediction of future of a signal by its past)

$$x_1 \quad \text{blue wavy line} \quad x_1(t) = \sum_{\tau=1}^p a(\tau)x_1(t-\tau) + e_1(t)$$

$$x_2 \quad \text{blue wavy line} \quad x_2(t) = \sum_{\tau=1}^p b(\tau)x_2(t-\tau) + e_2(t)$$

VAR Models (prediction of future of a signal by its past + the other signal's past)

$$X_2 | X_1 \quad \text{red wavy line} \quad GC_{2 \rightarrow 1} = \ln \frac{\text{var}(e_2)}{\text{var}(e_{2|1})} \approx \ln(> 1) = > 1$$

$$x_{2|1}(t) = \sum_{\tau=1}^p u(\tau)x_2(t-\tau) + \sum_{\tau=1}^p v(\tau)x_1(t-\tau) + e_{2|1}(t)$$

Incorporating information about X_1 improves the prediction of X_2 !

We say " X_1 granger-causes X_2 "

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Granger Causality

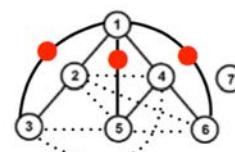
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full model

Problem: Pairwise GC for multichannel data has problems of spurious connectivity due to common inputs

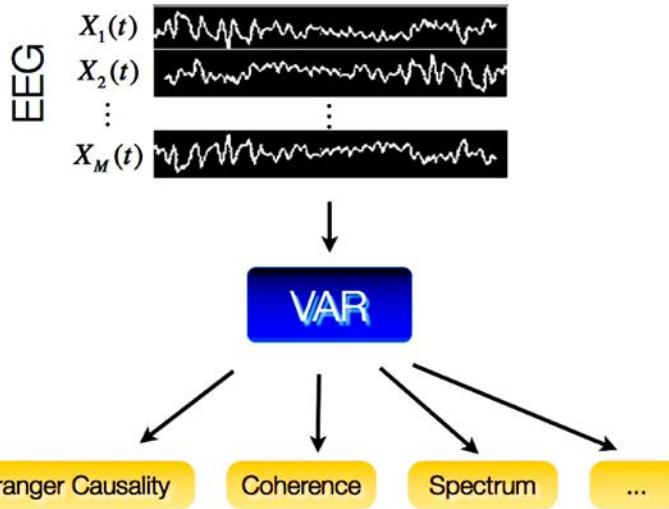
Solution: Multivariate VAR (MVAR)



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Vector Autoregressive (VAR / MAR / MVAR) Modeling



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The Linear Vector Auto-regressive (VAR) Model

Ordinary Least-Squares

$$X(t) = \begin{bmatrix} x_1(t) \\ x_2(t) \\ \vdots \\ x_M(t) \end{bmatrix}$$

VAR[p] model

$$X(t) = \sum_{k=1}^p A^{(k)}(t) X(t-k) + E(t)$$

Annotations for the VAR model equation:

- model order: p
- M-channel data vector at current time t : $X(t)$
- $M \times M$ matrix of (possibly time-varying) model coefficients indicating variable dependencies at lag k : $A^{(k)}(t)$
- multichannel data k samples in the past: $X(t-k)$
- random noise process: $E(t) \sim N(0, V)$

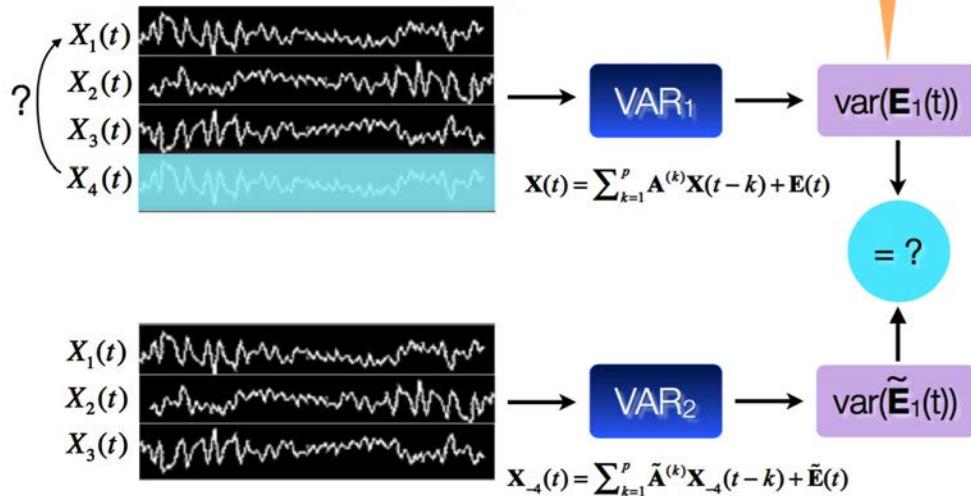
$$A^{(k)}(t) = \begin{pmatrix} a_{11}^{(k)}(t) & \cdots & a_{1M}^{(k)}(t) \\ \vdots & \ddots & \vdots \\ a_{M1}^{(k)}(t) & \cdots & a_{MM}^{(k)}(t) \end{pmatrix} \quad E(t) = N(0, V)$$

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Granger Causality

Does \mathbf{X}_4 granger-cause \mathbf{X}_1 ?
(conditioned on $\mathbf{X}_2, \mathbf{X}_3$)



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Granger-causality quiz

$$\begin{aligned} X_1(t) &= -0.5X_1(t-1) + 0X_2(t-1) + E_1(t) \\ X_2(t) &= 0.7X_1(t-1) + 0.2X_2(t-1) + E_2(t) \end{aligned}$$

Red arrows indicate causal relationships from X_1 to X_2 and from X_2 back to X_1 . A red 'X' is placed over the term $0X_2(t-1)$ in the first equation.

Which causal structure does this model correspond to?

- a) b) c)

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Granger Causality

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reduced model full model

- Alternately, for a **multivariate interpretation** we can fit a single MVAR model to all channels and apply the following definition:

Definition 1

X_j granger-causes X_i conditioned on all other variables in \mathbf{X}
if and only if $\mathbf{A}_{ij}(k) >> 0$ for some lag $k \in \{1, \dots, p\}$

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Granger Causality – Frequency Domain

$$\mathbf{X}(t) = \sum_{k=1}^p \mathbf{A}^{(k)} \mathbf{X}(t-k) + \mathbf{E}(t)$$

Fourier-transforming $\mathbf{A}^{(k)}$ we obtain

$$\mathbf{A}(f) = - \sum_{k=0}^p \mathbf{A}^{(k)} e^{-i2\pi fk}; \mathbf{A}^{(0)} = I$$

We can then define the spectral matrix $\mathbf{X}(f)$ as follows:

$$\mathbf{X}(f) = \mathbf{A}(f)^{-1} \mathbf{E}(f) = \mathbf{H}(f) \mathbf{E}(f)$$

Where $\mathbf{H}(f)$ is the *transfer matrix* of the system.

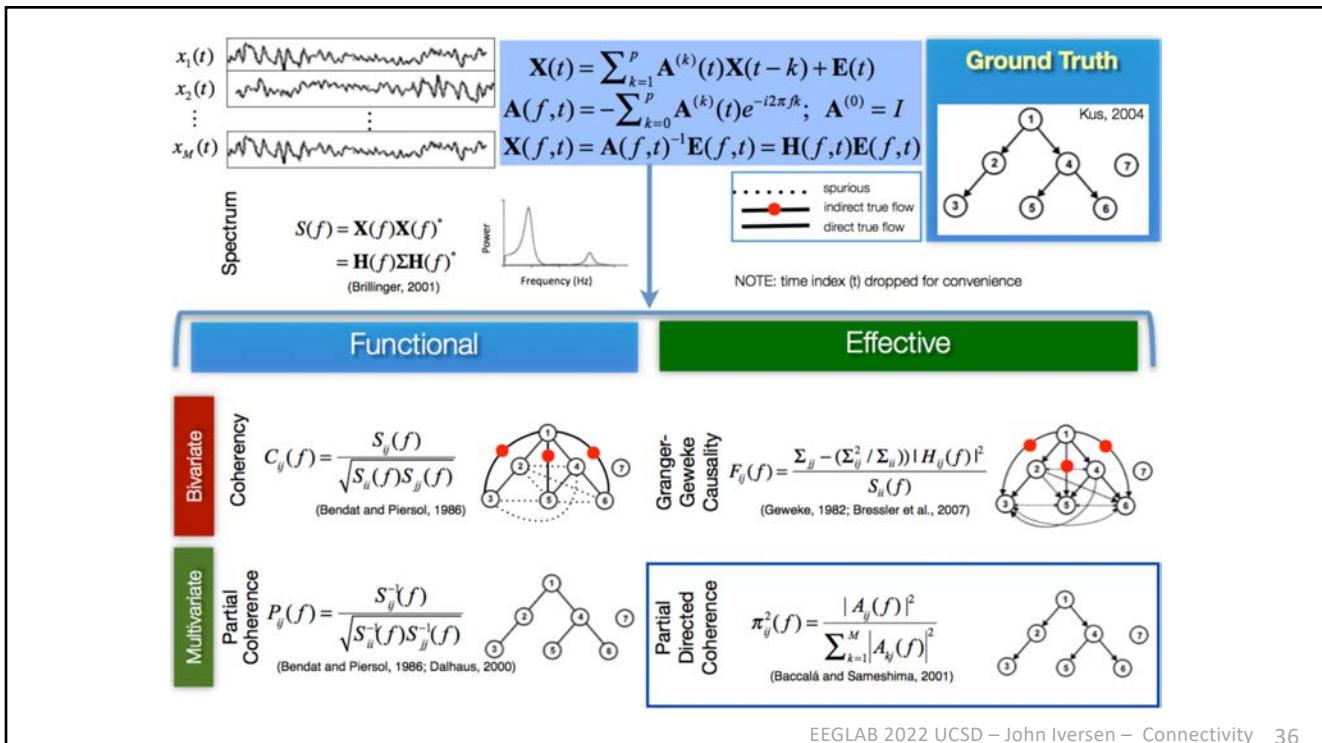
Definition 2

X_j granger-causes X_i conditioned on all other variables in \mathbf{X}
if and only if $|\mathbf{A}_{ij}(f)| >> 0$ for some frequency f

leads to
PDC

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Time-Frequency GC

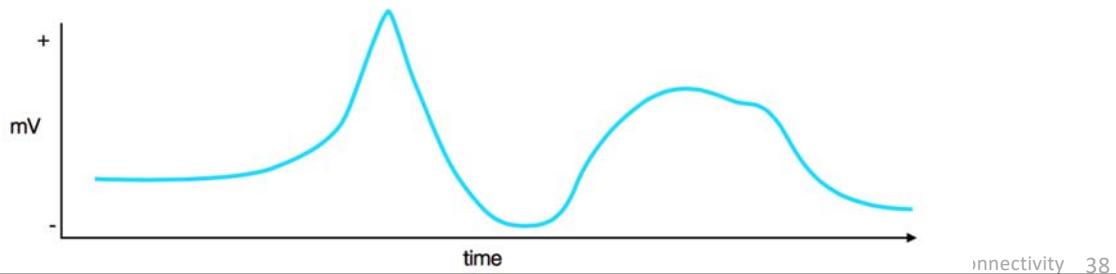
- Brain network dynamics often change rapidly with time
 - event-related responses
 - transient network changes during sequential information processing
- Electrophysiological processes often exhibit oscillatory phenomena, making them well-suited for frequency-domain analysis

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Adapting to Non-Stationarity

- The brain is a **dynamic system** and measured brain activity and coupling can change rapidly with time (non-stationarity)
 - event-related perturbations (ERSP, ERP, etc)
 - structural changes due to learning/feedback
- How can we adapt to non-stationarity?



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Adapting to Non-Stationarity

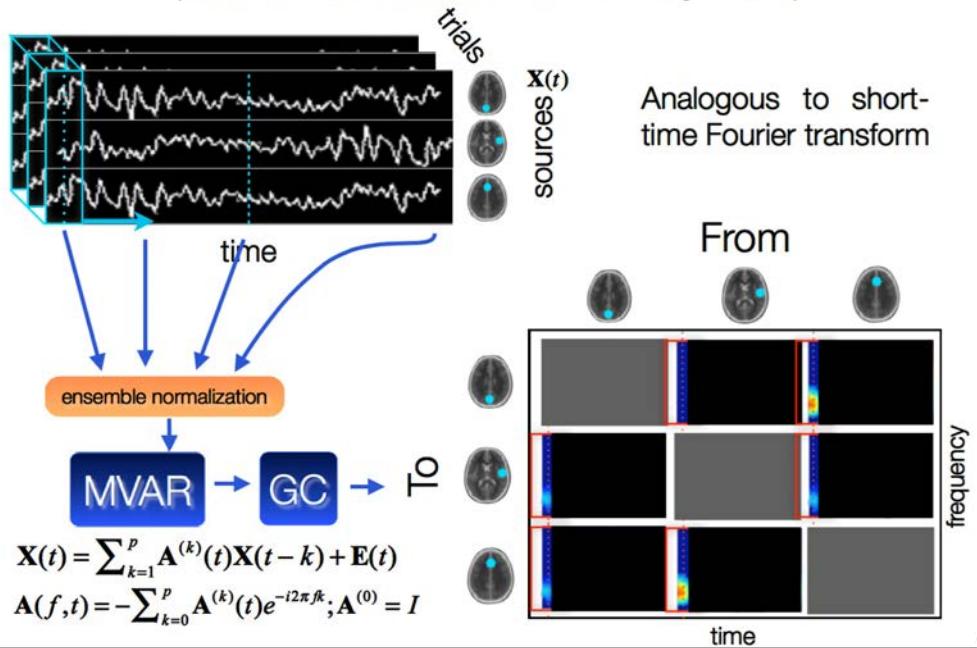
- Many ways to do adaptive VAR estimation
 - Segmentation-based adaptive VAR estimation
 - Factorization of time-varying spectral density matrices (e.g. from STFTs, Wavelets, etc)
 - State-Space Modeling
 - ...

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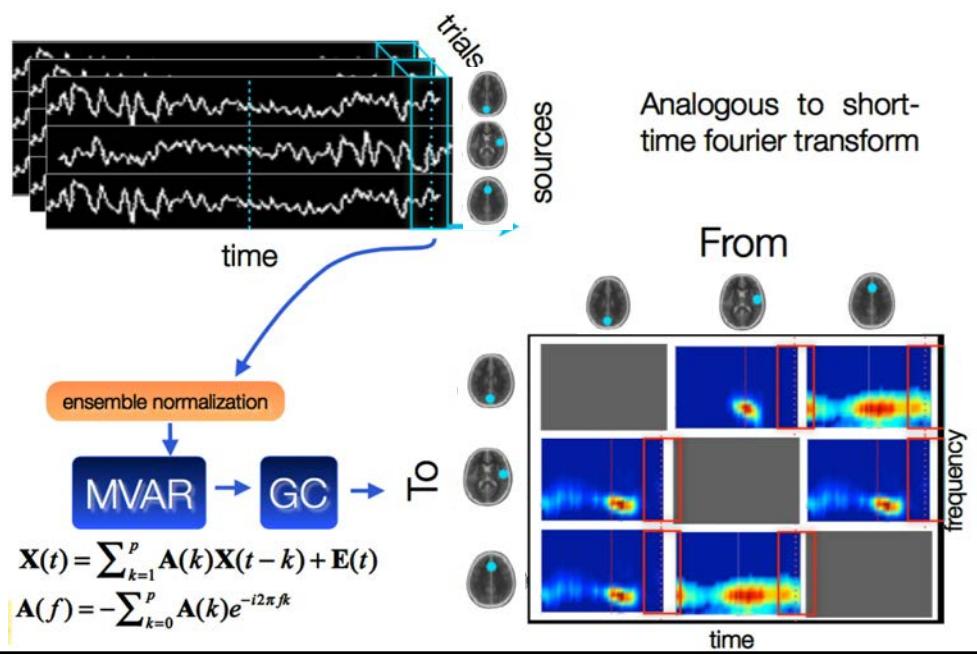
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Segmentation-based VAR

(Jansen et al., 1981; Florian and Pfurtscheller, 1995; Ding et al, 2000)



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Important Choices

- Model Order
 - Determines complexity of spectrum you can model
 - Larger orders need more data
- Window Length
 - Window must be long enough to contain sufficient data for your chosen model order
 - Must be long enough to encompass the time-scale of interactions
 - Yet not too long as to smear temporal dynamics or include non-stationary data

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Selecting a VAR Model Order

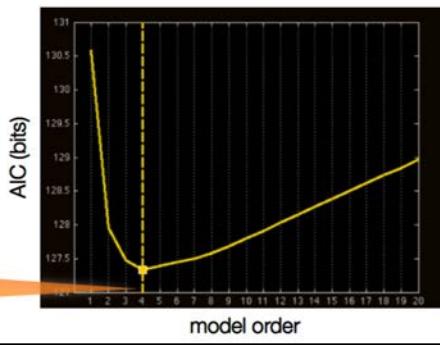
- Model order is typically determined by minimizing information criteria such as Akaike Information Criterion (AIC) for varying model order (p):

$$AIC(p) = 2\log(\det(\mathbf{V})) + M^2p/N$$

Penalizes high model orders (parsimony)

entropy rate (amount of prediction error)

optimal order



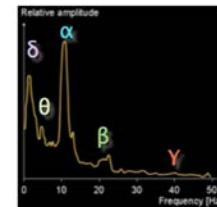
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Selecting a VAR Model Order

- **Other considerations:**

- A M -dimensional VAR model of order p has at most $Mp/2$ spectral peaks distributed amongst the M variables. This means we can observe at most $p/2$ peaks in each variables' spectrum (or in the causal spectrum between each pair of variables)
- Optimal model order depends on sampling rate (higher sampling rate often requires higher model orders)



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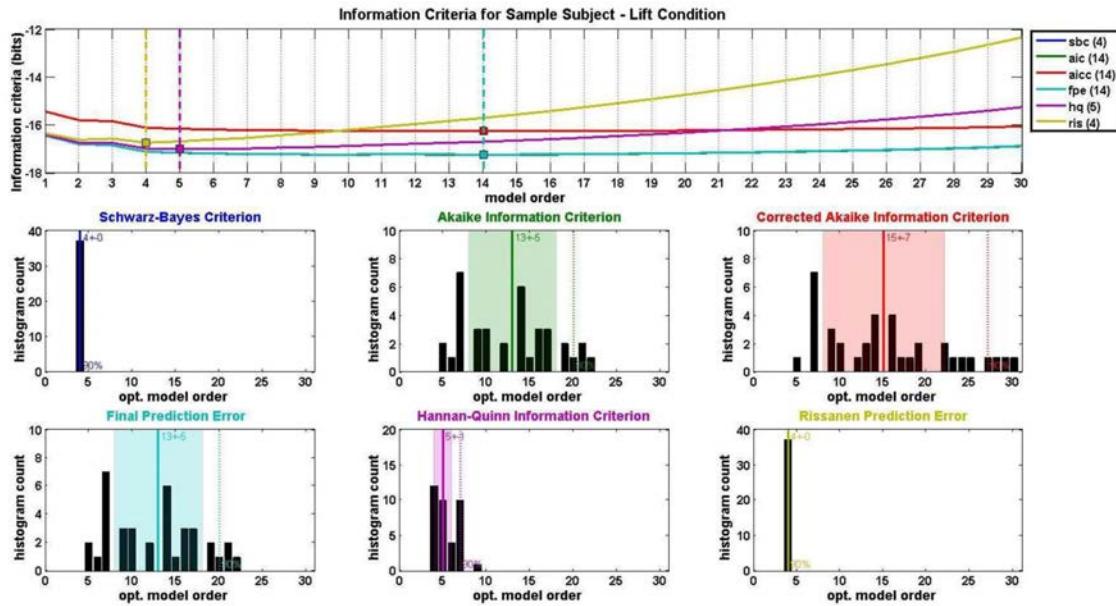
Model Validation

- If a model is poorly fit to data, then few, if any, inferences can be validly drawn from the model. There are a number of criteria which we can use to determine whether we have appropriately fit our VAR model. Here are three commonly used categories of tests:
- **Whiteness Tests:** checking the residuals of the model for serial and cross-correlation
- **Consistency Test:** testing whether the model generates data with same correlation structure as the real data
- **Stability Test:** checking the stability/stationarity of the model.

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Order selection *in reality*

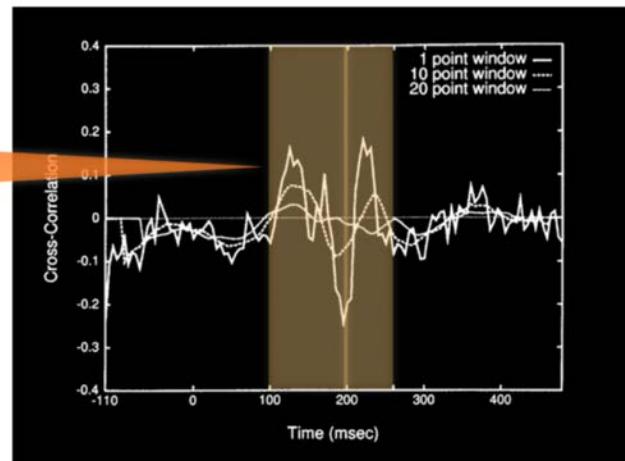


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Consideration: Local Stationarity

Too-large windows may not be locally-stationary



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Consideration: Sufficient data

M = number of variables

p = model order

N_{tr} = number of trials

W = length of each window (sample points)

We have M^2p model coefficients to estimate. This requires a minimum of M^2p independent samples.

So we have the constraint $M^2p \leq N_{tr} W$.

In practice, however, a better heuristic is $M^2p \leq (1/10)N_{tr} W$.

Or: $W \geq 10(M^2p/N_{tr})$

10x more data points than parameters to estimate

SIFT will let you know if your window length is not optimal

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Challenges of group-level Connectivity

- Not every participant has the same set of ICs!
 - What is the best way to aggregate across participants?
 - Methods that handle 'missing' ICs as 'missing data'
 - Methods that avoid the problem by doing ROI-based analyses, either based on ICs, or foregoing ICA entirely

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History of group-level SIFT

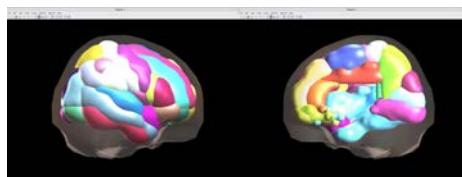
- Missing-data Approaches
 - Tim Mullen & Wes Thompson (since 2010...) 'Hierarchical Bayesian Modeling' that interpolate missing values (i.e. inconsistency in dipole locations across subjects).
- ROI-based approaches
 - Iversen, et al, 2014 ; Courellis, et al, 2017: project IC activation onto cortical surface and define activity in anatomically defined cortical ROIs.
 - Nima Bigdely-Shamlo (in his PhD dissertation in 2014) 'Network Projection' that uses dipole density and anatomical ROI.
 - **groupSIFT** created by Makoto Miyakoshi and Sandra Loo (UCLA) builds on network projection
 - **ROIconnect** plugin Arno Delorme & Stefan Haufe

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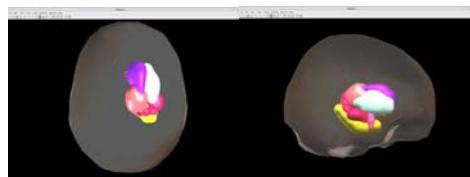
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groupSIFT
Makoto Miyakoshi
<https://github.com/sccn/groupSIFT>

- Based on Network Projection, but ROI-based
 - Divide Gaussian-smoothed dipole density into anatomical regions of interests (ROIs--used AAL atlas)



Defines 72 brain regions to include
 (36 for each hemisphere)

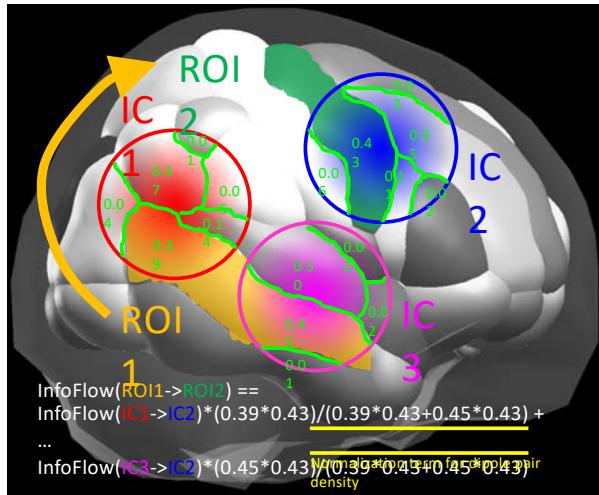


Defines 16 brain regions to exclude
 (8 for each hemisphere)

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How to normalize IC-network to anatomical ROIs

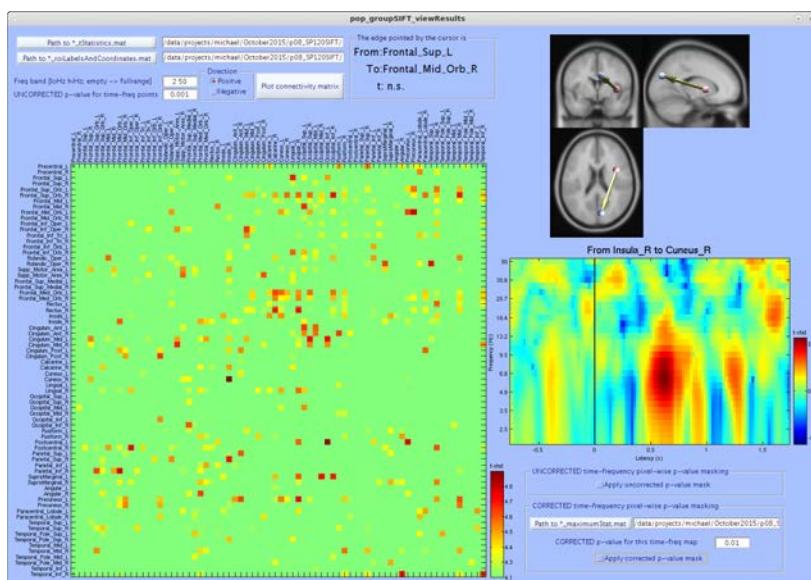


1. Run ICA, DIPFIT, and SIFT.
2. Smooth dipole positions into **dipole density** that is 3-D Gaussian sphere with FWHM == 14.2 mm (for the case of 9.6 mm error in average across all cortex; Akalin Acar et al., 2013.)
3. Compute the dipole density according to Automated Anatomical Labeling Atlas (Tzourio-Mazoyer et al., 2002)
4. Repeat above process for all ICs.
5. For each pair of regions (there are $72 \times 72 = 5184$ combinations), compute **pairwise dipole density** which is a product of two dipole densities.
6. Multiply info flow measure (rPDC, dDTF, etc) by normalized pairwise dipole density to calculate ROI-to-ROI info flow.

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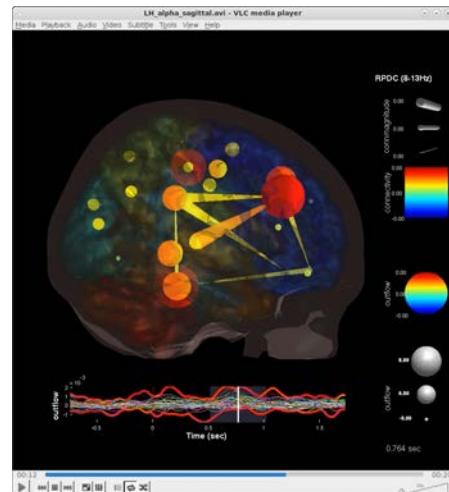
Connectivity Matrix Results



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Causal Flow Movie

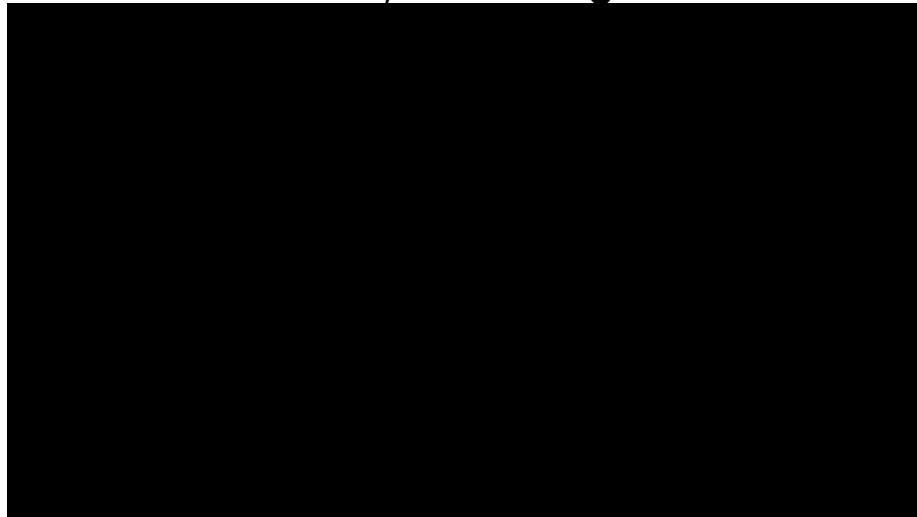


- It uses SIFT's GUI to make the movie by feeding single subject dataset replaced with group-mean data.

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Hyperconnectivity in chronic tic disorder Loo, et al 2019



Cued eye blink. Children with chronic tic disorder have abnormally high information flow from occipital to frontal. May be neural underpinning of CTD hyper-sensitivity.

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groupSIFT

Makoto Miyakoshi

Published works (updated 05/04/2021)

The dedicated technical paper is not prepared yet. But a couple of clinical researches using groupSIFT are already published. Loo et al. (2019) has relatively detailed description of the method in Supplement (which needs some update).

Loo et al. (2019) Neural activation and connectivity during cued eye blinks in Chronic Tic Disorders. *NeuroImage: Clinical* 24:101956

Koshiyama et al. (2020) Abnormal effective connectivity underlying auditory mismatch negativity impairments in schizophrenia. *Biological Psychiatry CNNI* 5:1028-1039.

Koshiyama et al. (2020) Neurophysiologic Characterization of Resting State Connectivity Abnormalities in Schizophrenia Patients. *Front Psychiatry* 11:608154.

Koshiyama et al. (2020) Auditory-Based Cognitive Training Drives Short- and Long-Term Plasticity in Cortical Networks in Schizophrenia. *Schizophrenia Bulletin Open* 1:sgaa065.

Miyakoshi et al. (2021) The AudioMaze: An EEG and motion capture study of human spatial navigation in sparse augmented reality. *European Journal of Neuroscience* Online ahead of print.

Jurgiel et al. (2021) Inhibitory control in children with tic disorder: aberrant fronto-parietal network activity and connectivity. *Brain Communications* 3:fcab067.

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Application of group SIFT

Received: 22 July 2020 | Revised: 21 December 2020 | Accepted: 19 January 2021
DOI: 10.1111/ejnn.15131

SPECIAL ISSUE ARTICLE

EJN European Journal of Neuroscience FENS WILEY

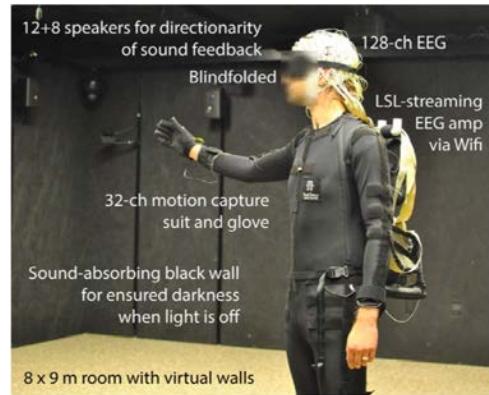
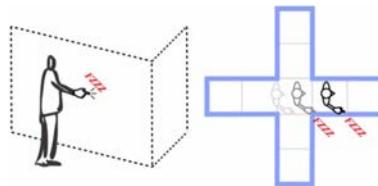
The *AudioMaze*: An EEG and motion capture study of human spatial navigation in sparse augmented reality

Makoto Miyakoshi¹  | Lukas Gehrke²  | Klaus Gramann^{2,3}  | Scott Makeig¹ | John Iversen¹

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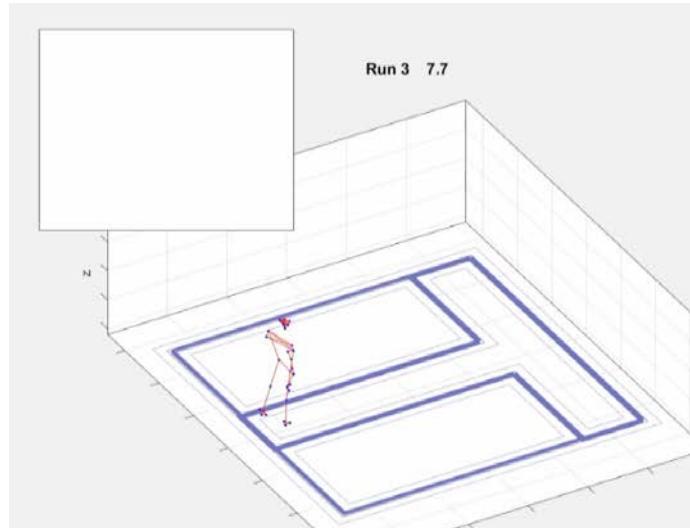
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Audiomaze



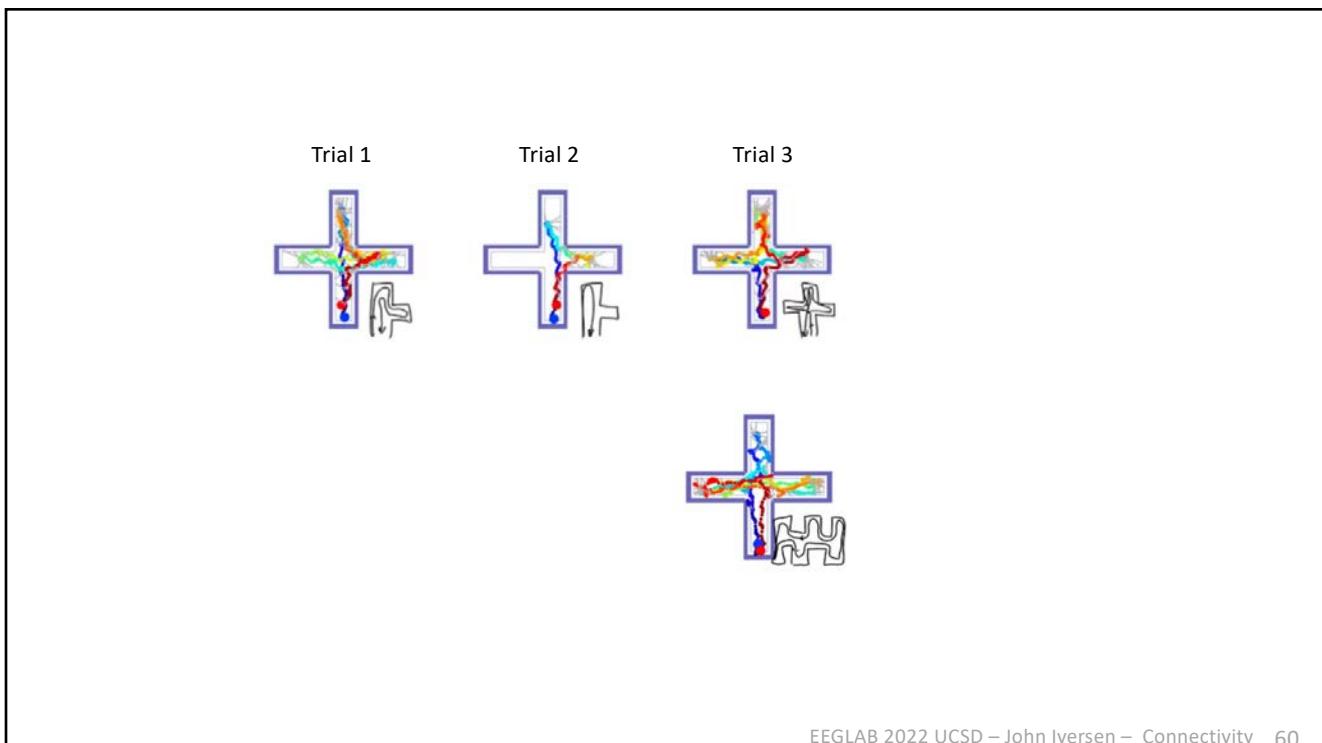
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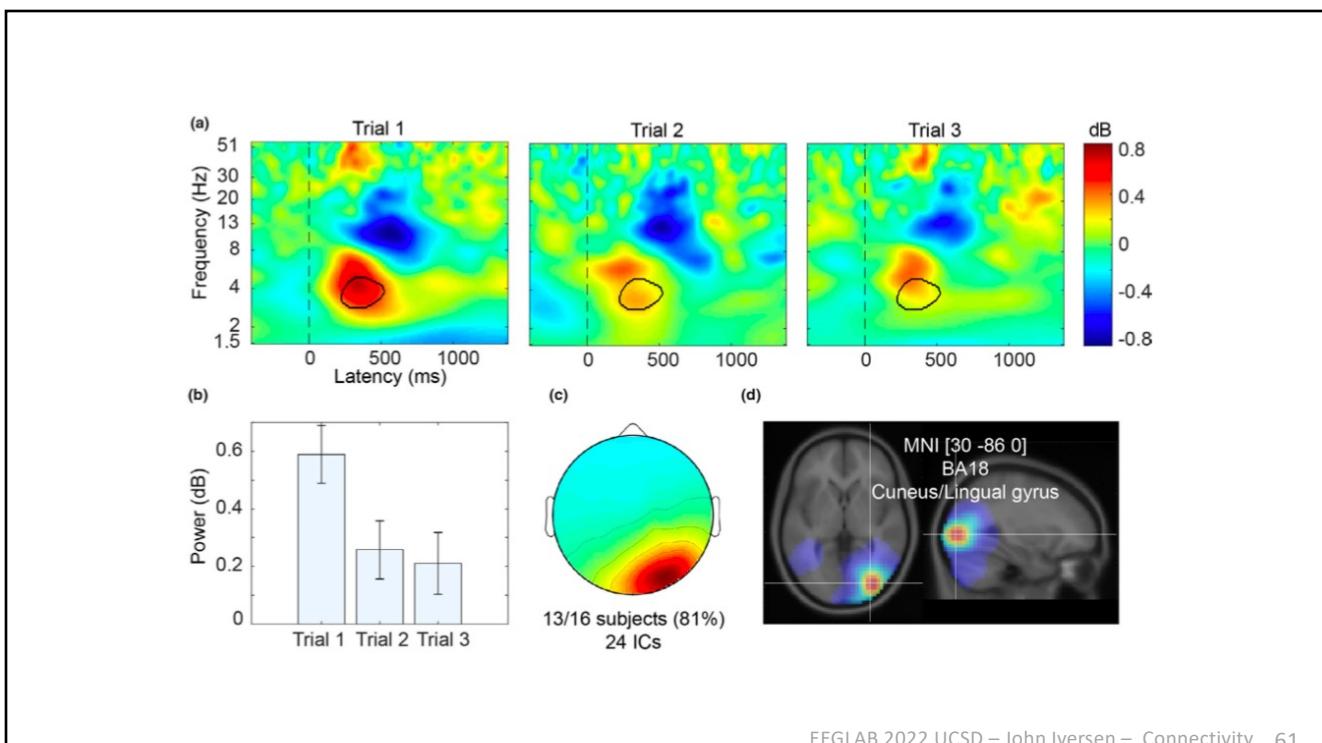


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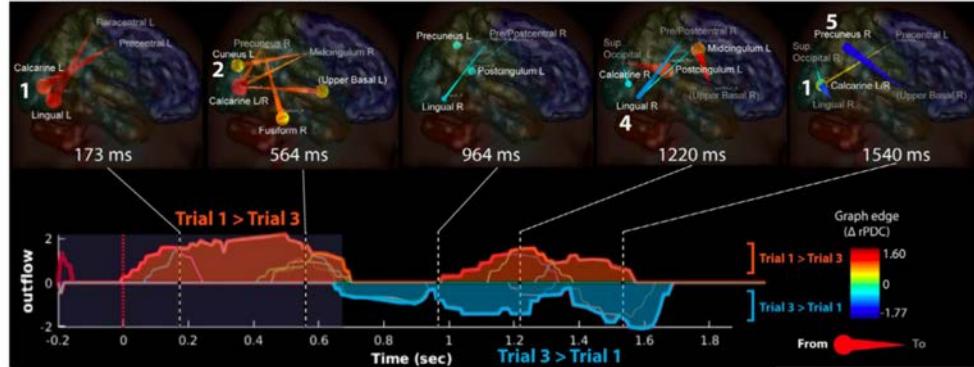


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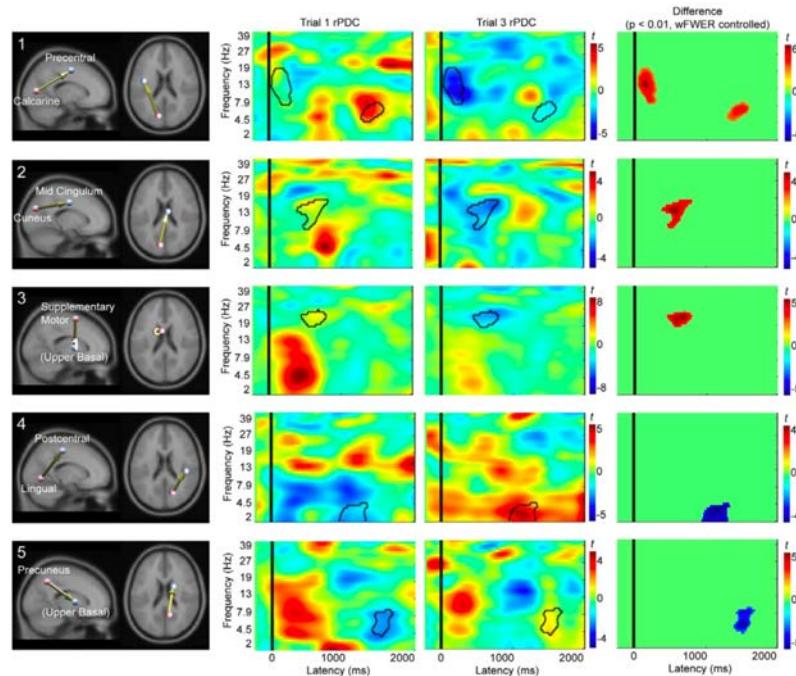
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groupSIFT result

(a) Low-frequency (2-13 Hz) Δ rPDC

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Discussion

- **Decreases** in short-latency occipital to central causal flow during maze learning
- **Increases** in causal flow from precuneus and lingual gyrus to motor regions at longer latencies

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Example Application

EEG-Based Quantification of Cortical Current Density and Dynamic Causal Connectivity Generalized across Subjects Performing BCI-Monitored Cognitive Tasks

Hristos Courellis^{1,2}, Tim Mullen¹, Howard Poizner³, Gert Cauwenberghs^{2,3} and John R. Iversen¹*

Frontiers in Neuroscience | www.frontiersin.org

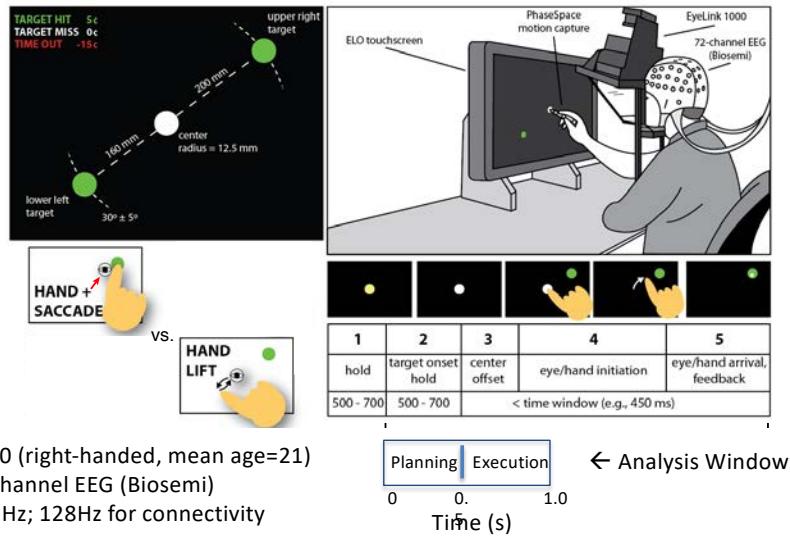
May 2017 | Volume 11 | Article 180

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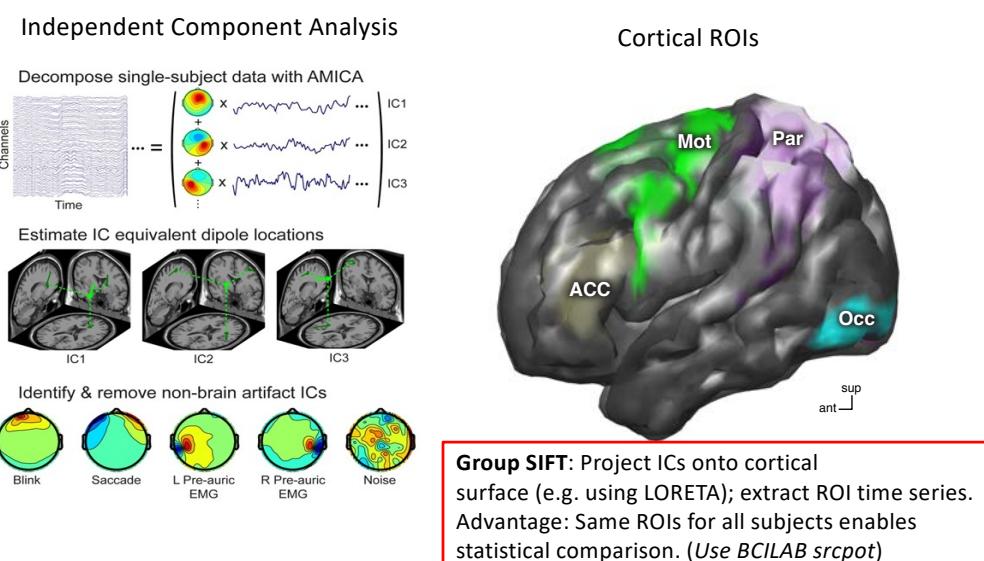
How does brain plan visually guided movements?

- Pointing Task (Park, et al. 2014, *IEEE Trans Neural Syst Rehabil Eng*)



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ICA source space analysis



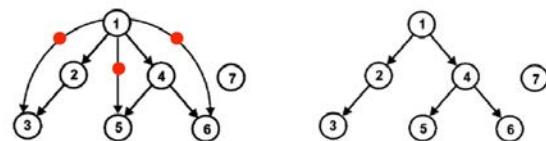
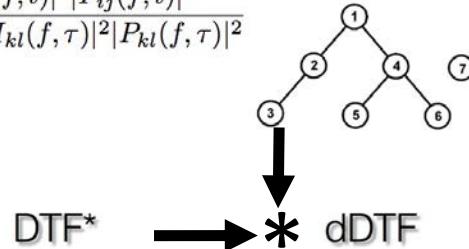
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dDTF

Partial Coherence

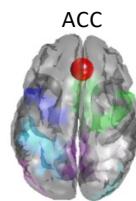
$$\eta_{ij}^2(f, t) = \frac{|H_{ij}(f, t)|^2 |P_{ij}(f, t)|^2}{\sum_{klf\tau} |H_{kl}(f, \tau)|^2 |P_{kl}(f, \tau)|^2}$$



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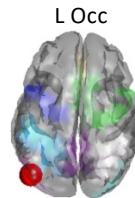
SIFT Analysis



- Time-varying SdDTF

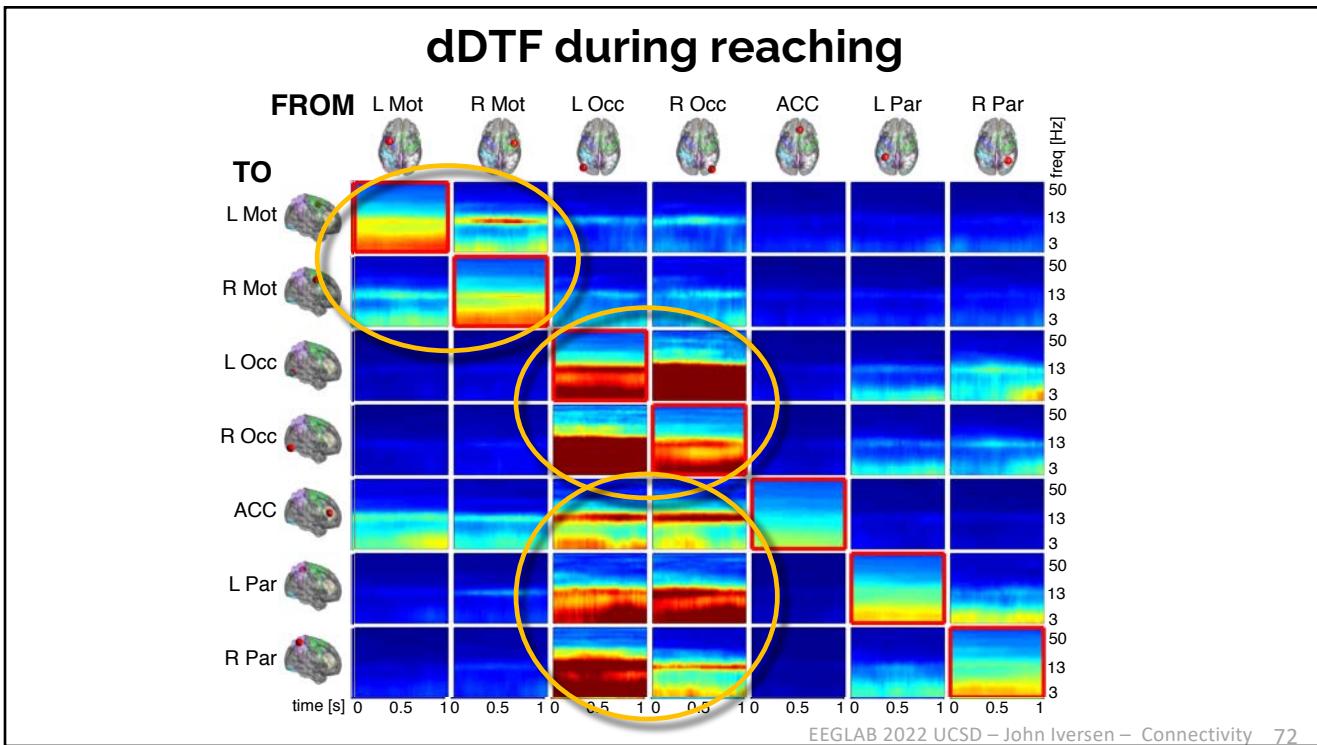
Directed measure of direct causal flow between ROIs

Averaged across subjects

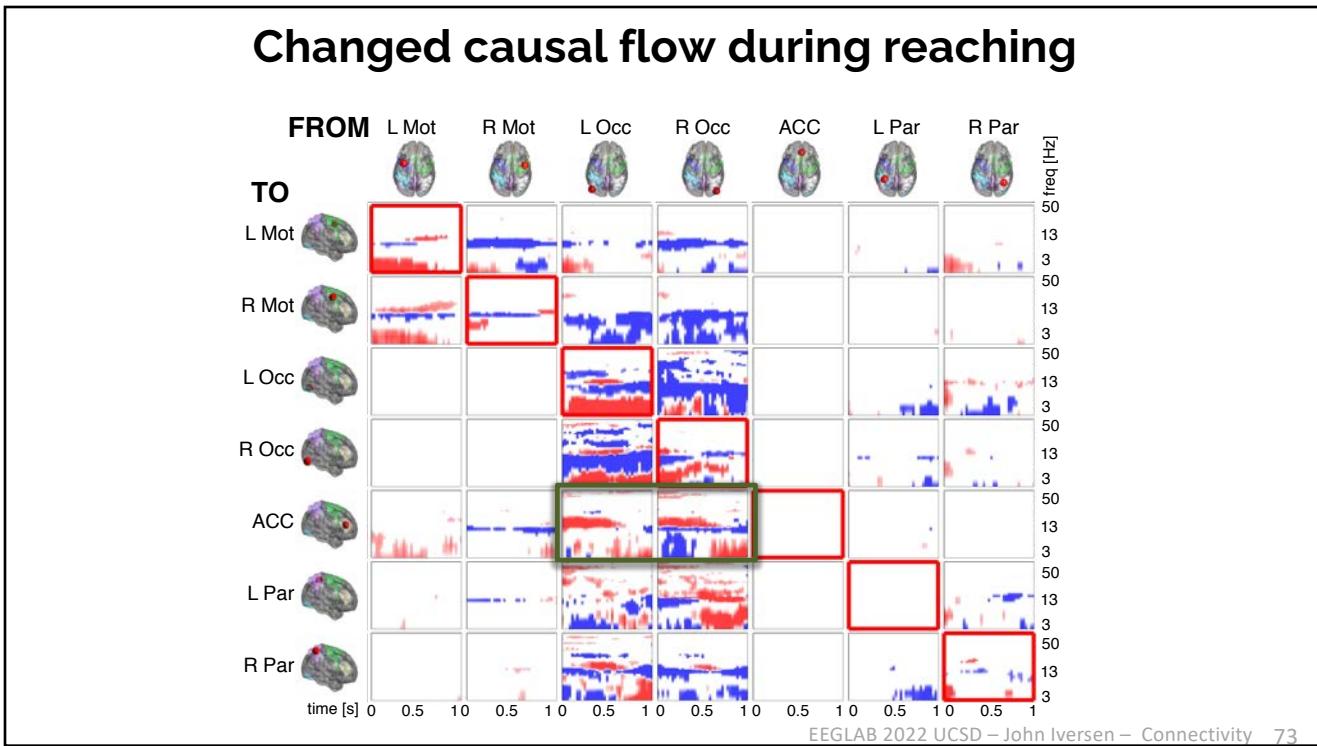


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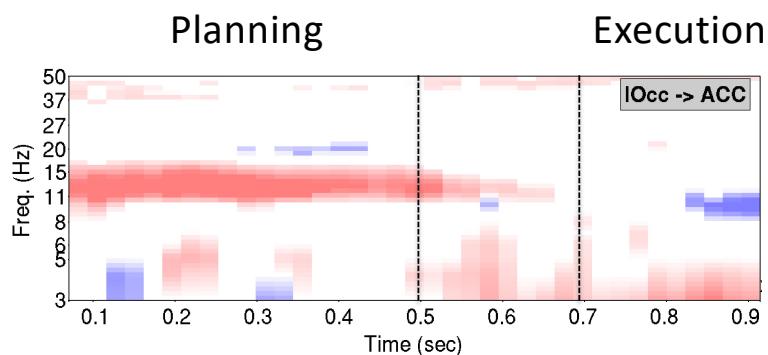


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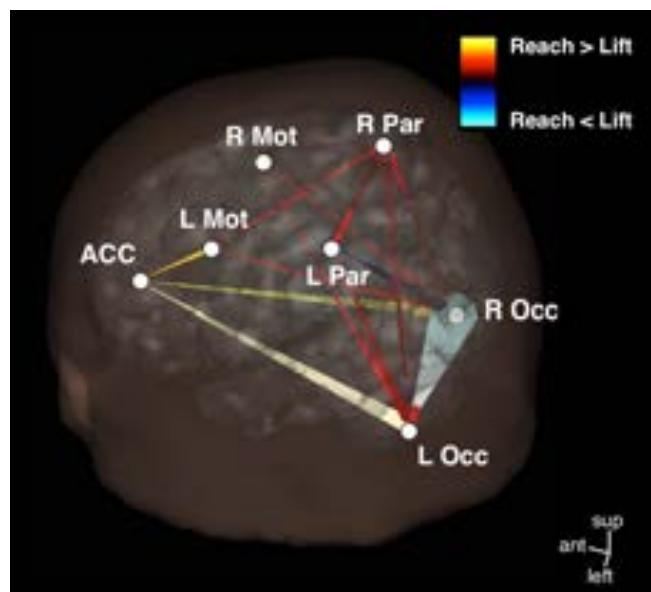
Occipital → ACC



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Greater causal flow during movement planning

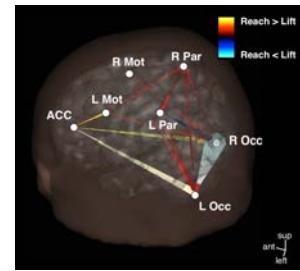


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Discussion

- SIFT is a capable toolkit for causal dynamical analysis at source level
- **Parietal** network expected for visually guided action (e.g. Heider, et al., 2010)
- **ACC** more strongly driven by Occipital & Motor. Locus for translation of intention into action (Paus, 2001; Srinivasan, et al. 2013). ACC drives SMA (not shown).
- Causal network results depend on the number of nodes
 - E.g. Occipital → ACC could be mediated by region not included in model
 - There will always be a tradeoff between network size and amount of data needed to fit the model.



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