Cognition and Brain Sciences Unit

Effective Connectivity (for fMRI and M/EEG)

Rik Henson

MRC CBU, Cambridge

Structural, functional & effective connectivity

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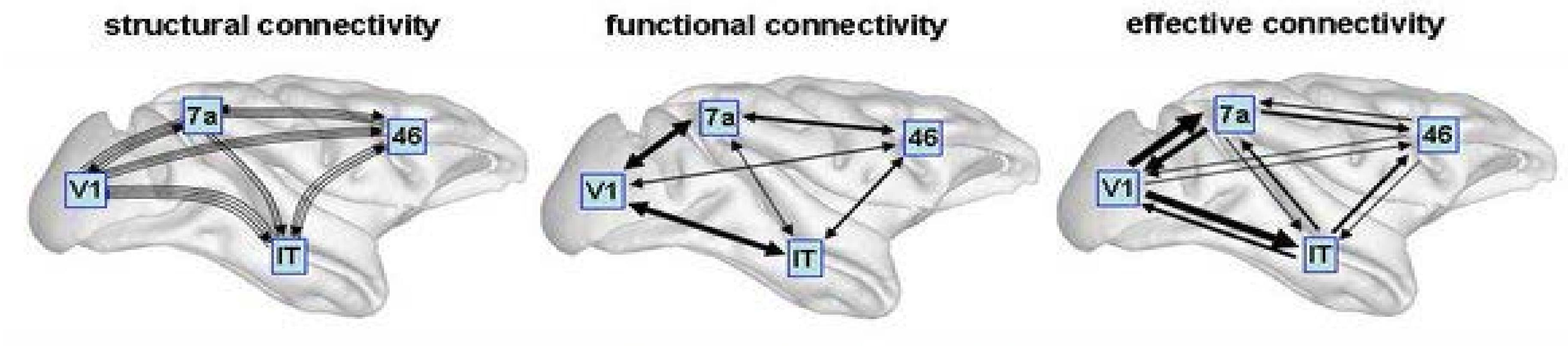

- **Structural/anatomical connectivity**
  = presence of axonal connections / white matter tracks (eg, DWI)

- **Functional connectivity**
  = statistical dependencies between regional time series (eg, ICA)

- **Effective connectivity**
  = causal (directed) influences between neuronal populations (eg, DCM)
  (based on explicit network models)

Structural vs Functional connectivity

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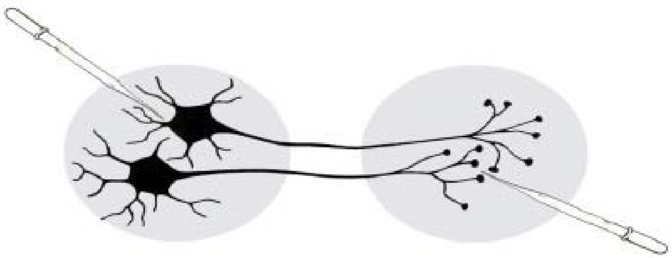
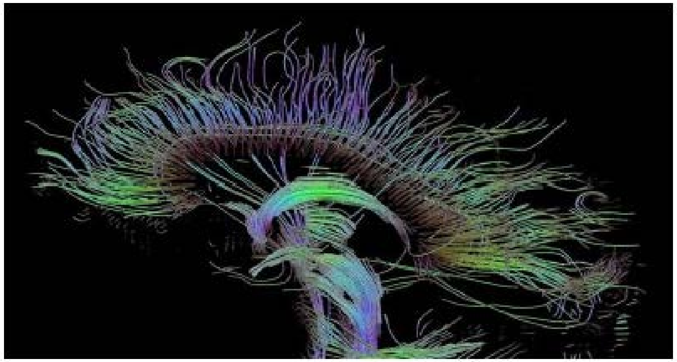
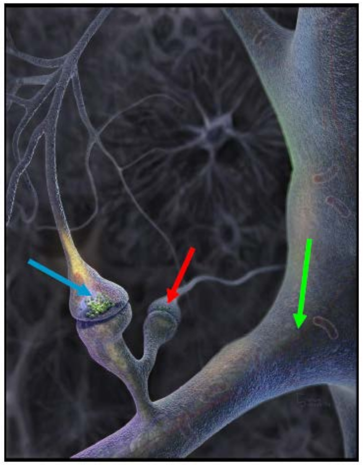


- Tracing studies



- Tractography from DWI



But functionally, effect of one neuron on another can depend on:

– Activity of a third (gating)

– Rapid changes in plasticity

Functional vs Effective connectivity

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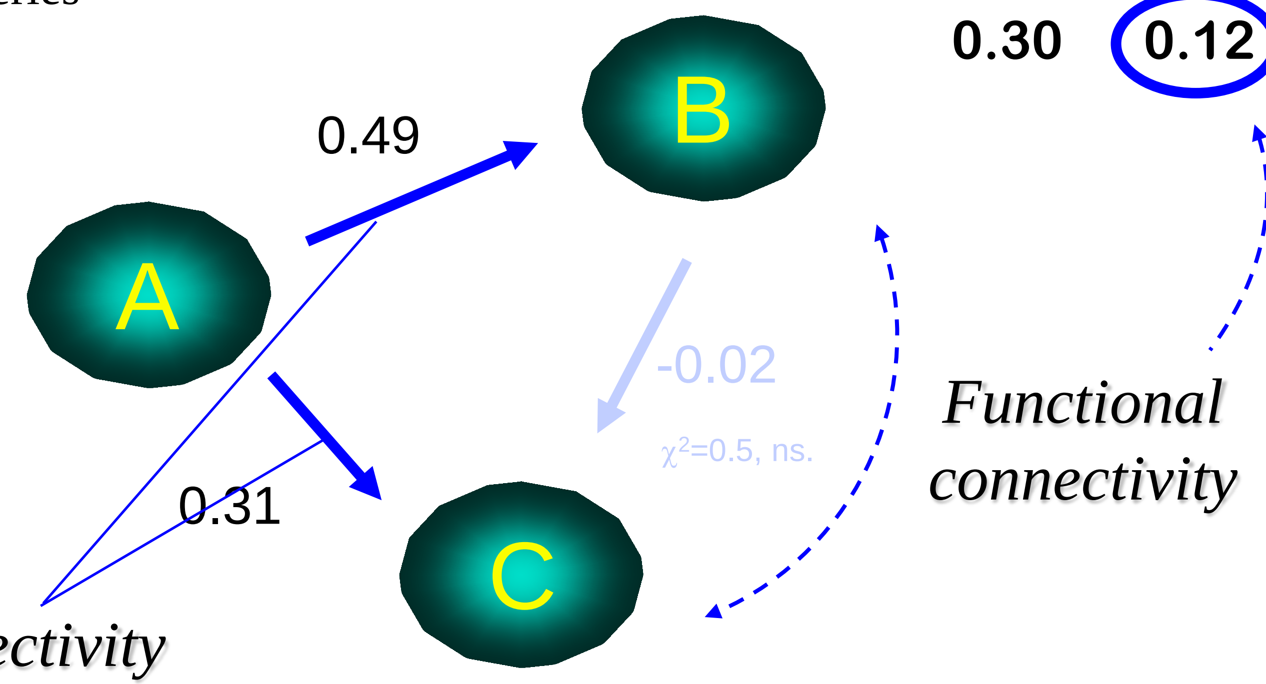
No connection between B and C, yet B and C correlated because of common input from A, eg:

A = V1 fMRI time-series
B = 0.5 * A + e1
C = 0.3 * A + e2

Correlations:

A	B	C
1		
0.49	1	
0.30	0.12	1



Effective connectivity

Functional/Effective Connectivity for fMRI

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Functional connectivity

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- Useful when no model, no experimental perturbation (eg resting state)
- Popular examples: seed-voxel correlations, PCA, ICA, etc
- Graph-theory summaries of functional networks

- Correlations in fMRI timeseries could be spurious haemodynamics (e.g, effects of heart-rate/breathing; movement confounds…)
- Condition-dependent changes in functional connectivity (e.g, PPIs…)

Effective-connectivity: Definitions of Causality?

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1. Direct experimental interventions (e.g, lesion, drugs)
2. Indirect experimental manipulations (e.g, PPI, DCM)
3. Network model inference (e.g, SEM, DCM)
4. Temporal precedence (e.g, Granger Causality, DCM)
5. …

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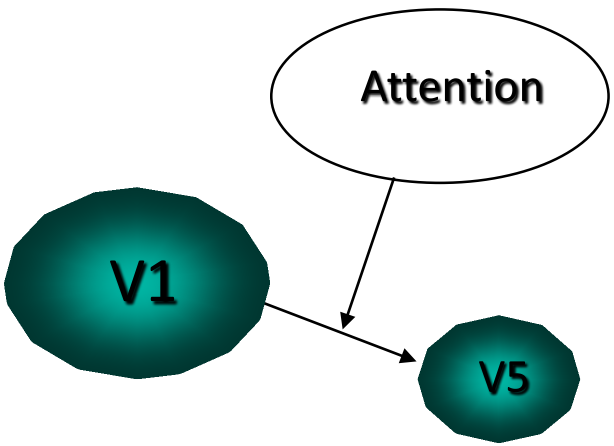
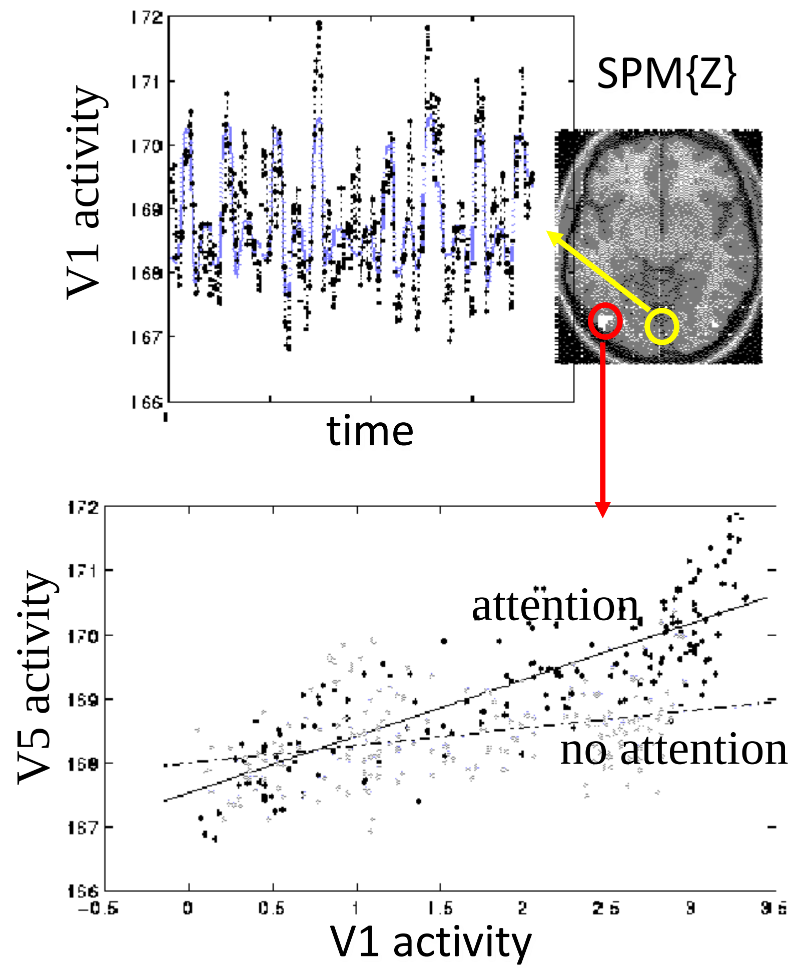
2. Condition-dependent changes: eg PPI

Parametric, factorial design, in which one factor is psychological (eg attention)

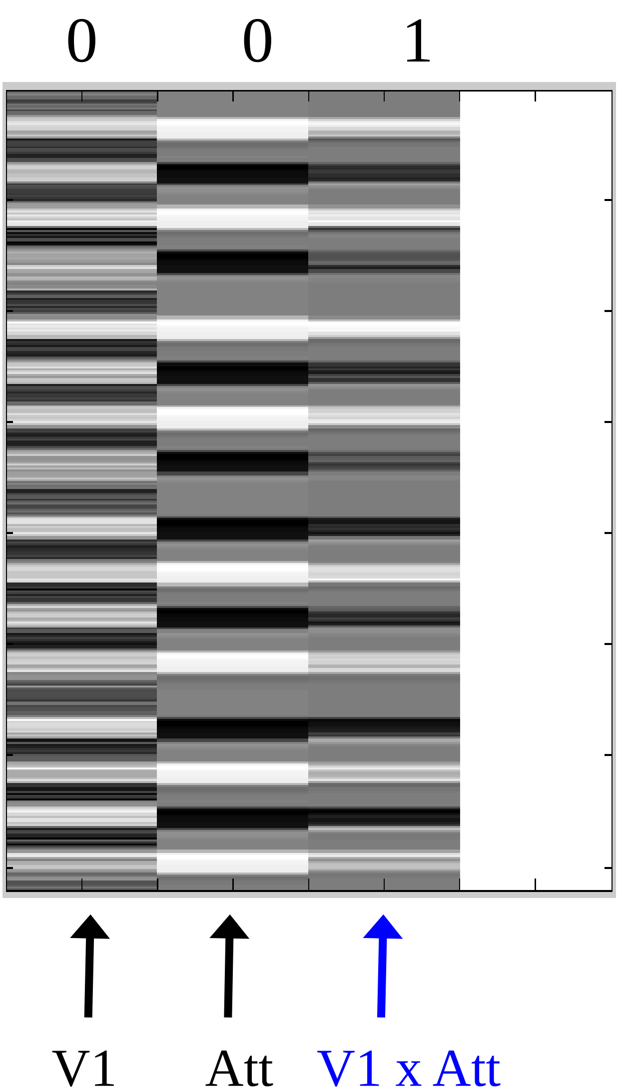
...and other is physiological *(viz. activity extracted from a brain region of interest)*

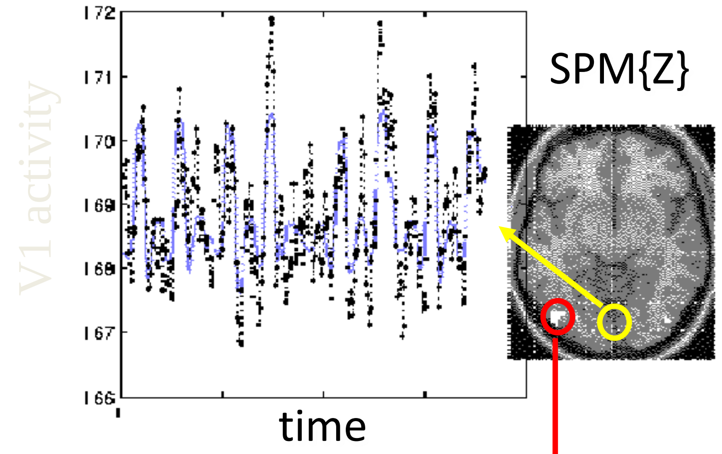


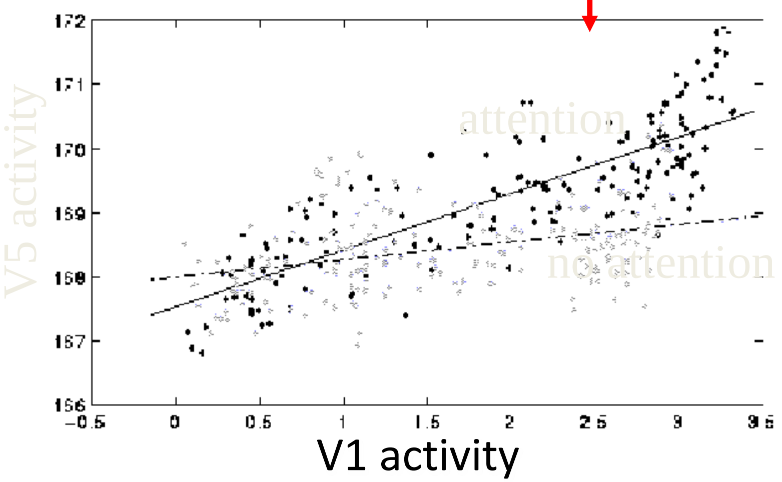
Attentional modulation of V1 - V5 connectivity



2. Condition-dependent changes: eg PPI







Effective-connectivity: Definitions of Causality?

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5. …

3. Explicit Network Models of Causality

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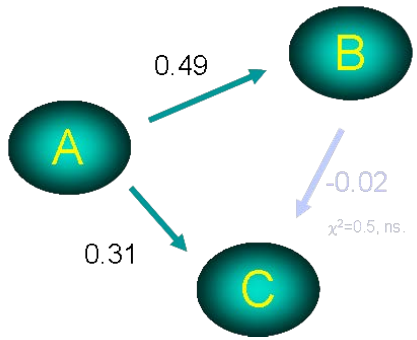
- (Bivariate) correlations do not use an explicit network (graph) model



- Structural Equation Modelling (SEM) can test different network models, by simply comparing *predicted* with *observed* covariance matrices, but…
  - has no dynamical model (stationary covariances)
  - has no neural–BOLD model
  - cannot test some graphs, eg loops (no temporal definition of direction)
  - restricted to classical inference comparing nested models

Effective-connectivity: Definitions of Causality?

1. Direct experimental interventions (e.g, lesion, drugs)
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5. …

4. Temporal definition of Causality

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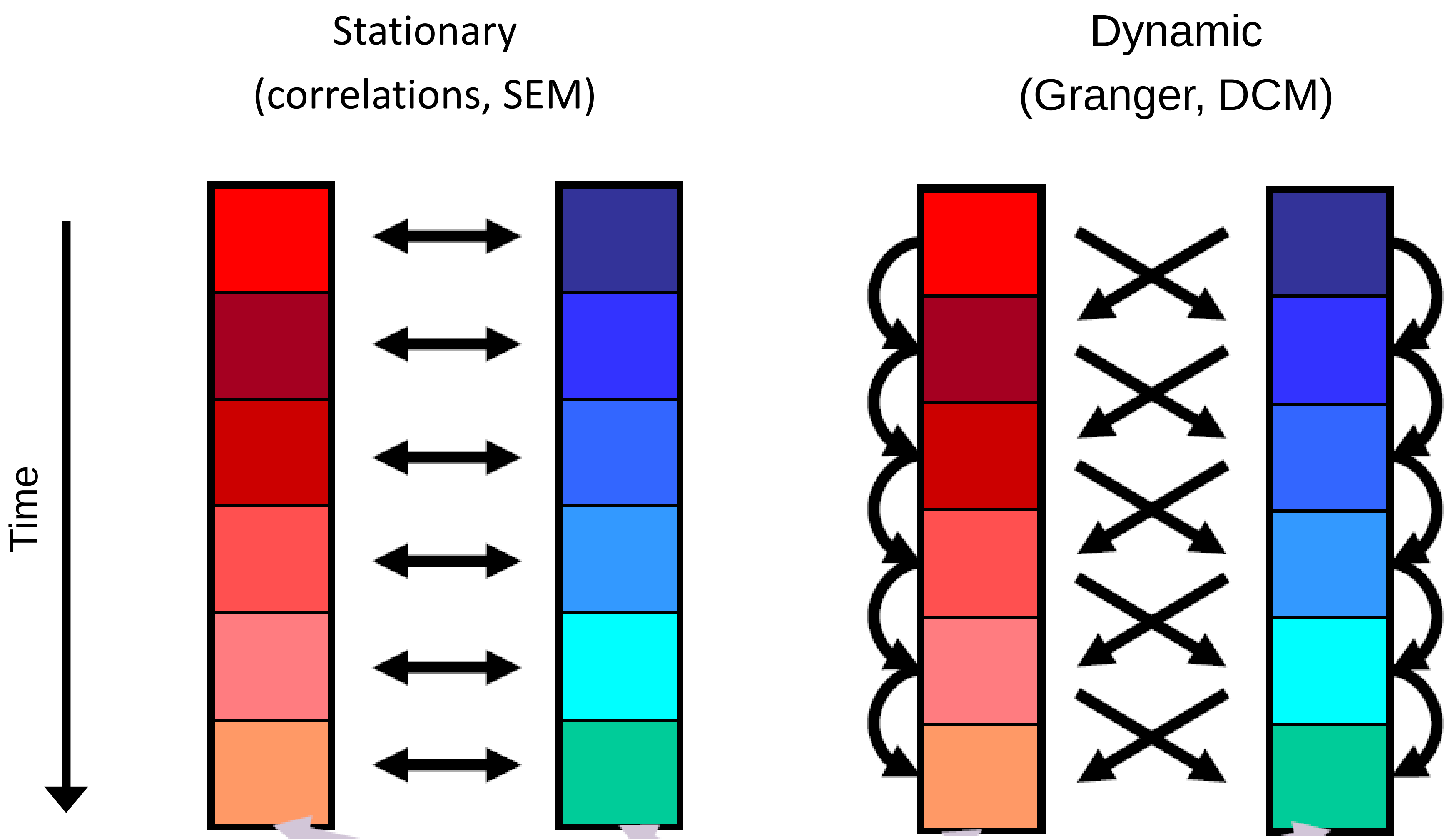

("unfolding" in time is one way to infer direction of connectivity)

4. Note on temporal causality and fMRI

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- Problem with time-based measures of connectivity arises with fMRI: BOLD timeseries is not direct reflection of Neural timeseries
  - (e.g, peak BOLD response in motor cortex can precede that in visual cortex in a visually-cued motor task, owing to different neural-BOLD mappings)

- This compromises methods like Granger Causality and Multivariate Auto-Regressive models (MAR) that operate directly on fMRI data

  (Friston, 2010; Smith et al, 2011)

- Note that this does not preclude these methods (eg MAR) for MEG/EEG timeseries, assuming these are more direct measures of neural activity

=> Development of DCM

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1. *Dynamic:* based on first-order differential equations
   - at level of neural activity, with separate haemodynamic model for fMRI

2. *Causal:* based on explicit directed graph models

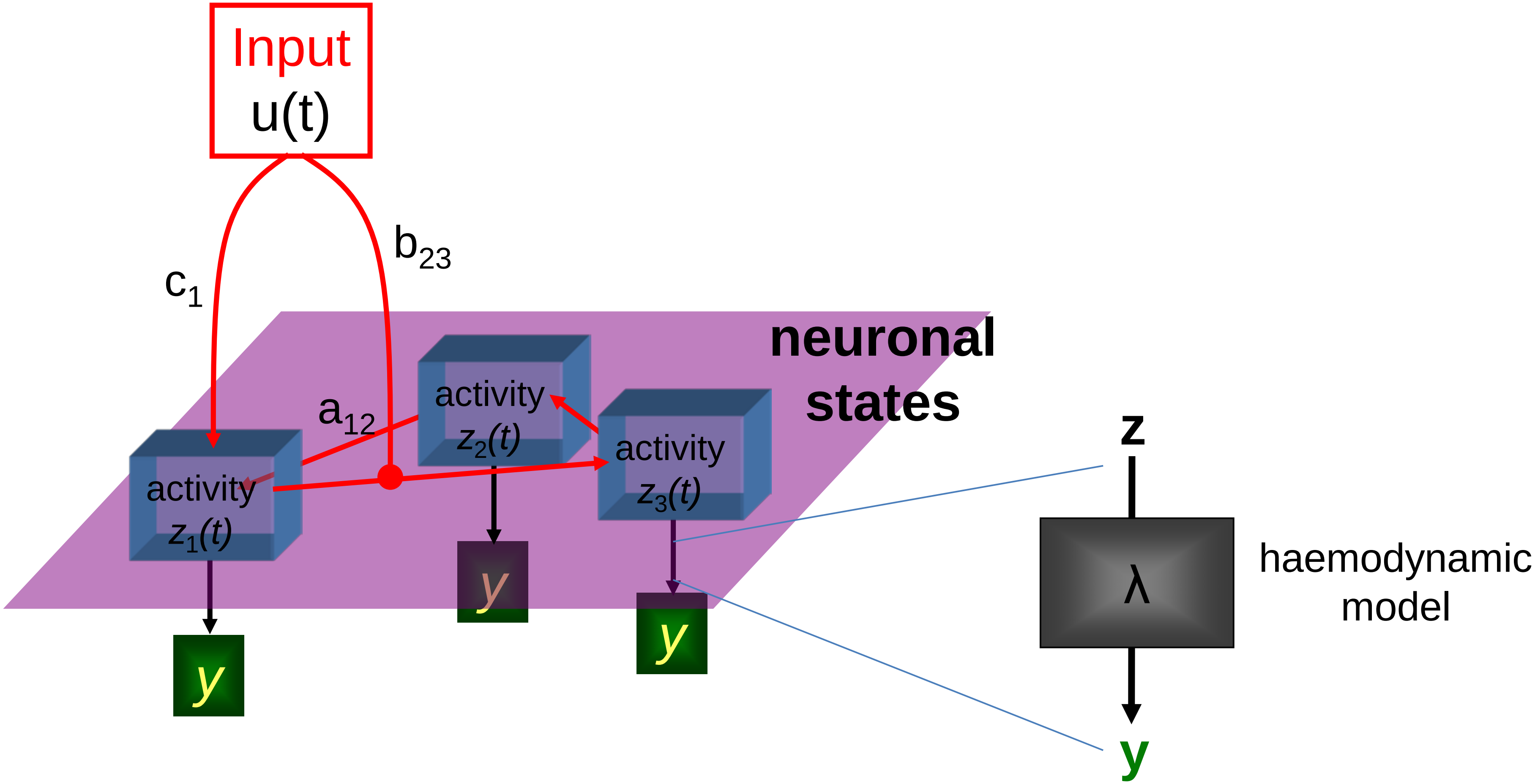
3. *Modelling:* designed to test experimental manipulations
   - "bilinear" approximation to interactive dynamics

4. (Estimated in a Bayesian context, allowing formal comparison of any number/type of models…)

Rough comparison of popular methods?

	Experimental modulation	Temporal/ Dynamical	Network model	Haemodynamic Model (for fMRI)
Correlation / ICA / PCA				
PPI	Y			
Granger		Y		
SEM			Y	
DCM	Y	Y	Y	Y

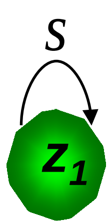
DCM overview



Friston et al. 2003, *NeuroImage*

DCM Neural Level

Oridinary Differential Equations:



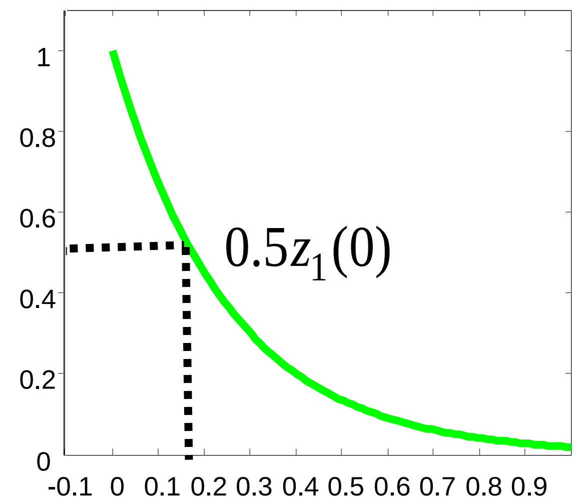
$$\frac{dz_1}{dt} = -sz_1 \quad \Rightarrow \quad z_1(t) = z_1(0)\exp(-st), \qquad z_1(0) = 1$$

Half-life τ

$$z_1(\tau) = 0.5 z_1(0)$$
$$= z_1(0)\exp(-s\tau)$$

$$\Rightarrow \quad s = \ln 2 / \tau$$

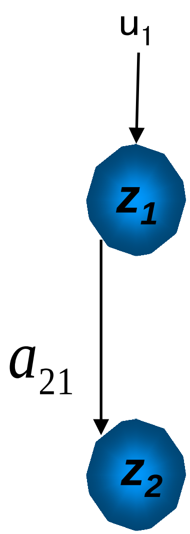
Decay function

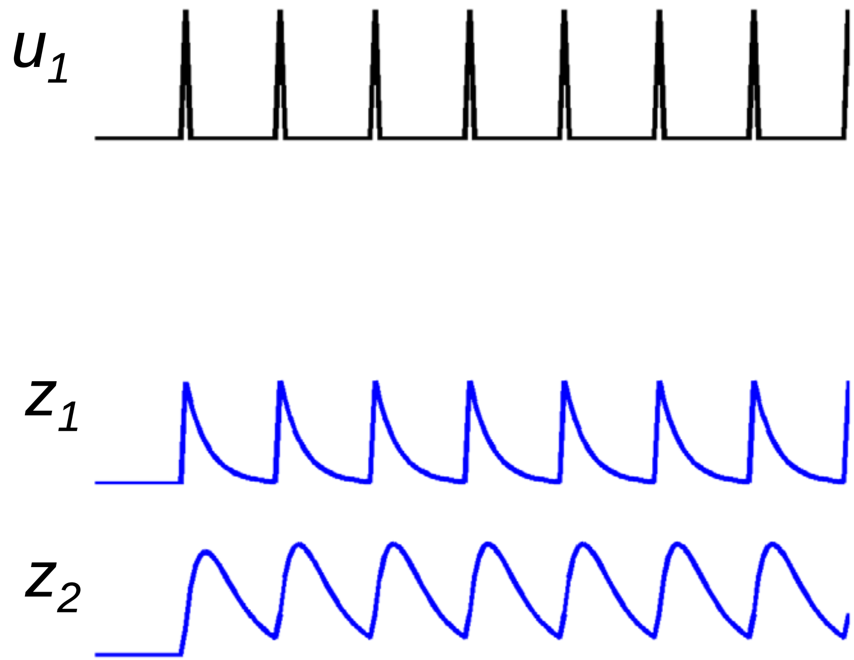


$$\tau = \ln 2 / s$$

DCM parameters = rate constants

Neurodynamics: 2 nodes, 1 driving input





$$\begin{bmatrix}\dot{z}_1\\ \dot{z}_2\end{bmatrix} = s\begin{bmatrix}-1 & 0\\ a_{21} & -1\end{bmatrix}\begin{bmatrix}z_1\\ z_2\end{bmatrix}+\begin{bmatrix}c\\ 0\end{bmatrix}u_1$$

$$a_{21} > 0$$

Neurodynamics: …+1 modulatory input

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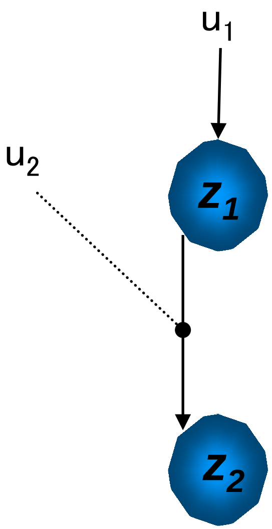
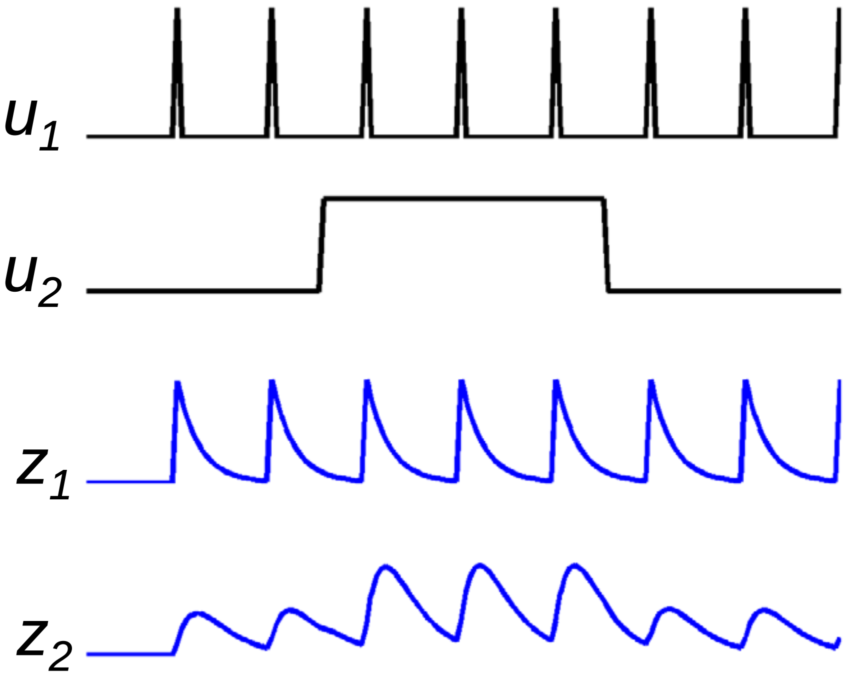
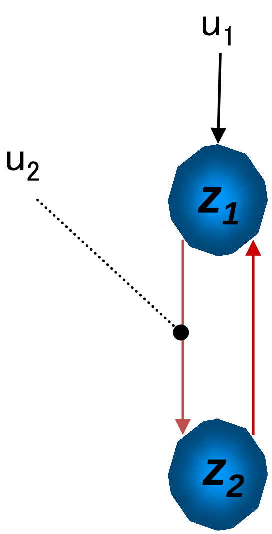
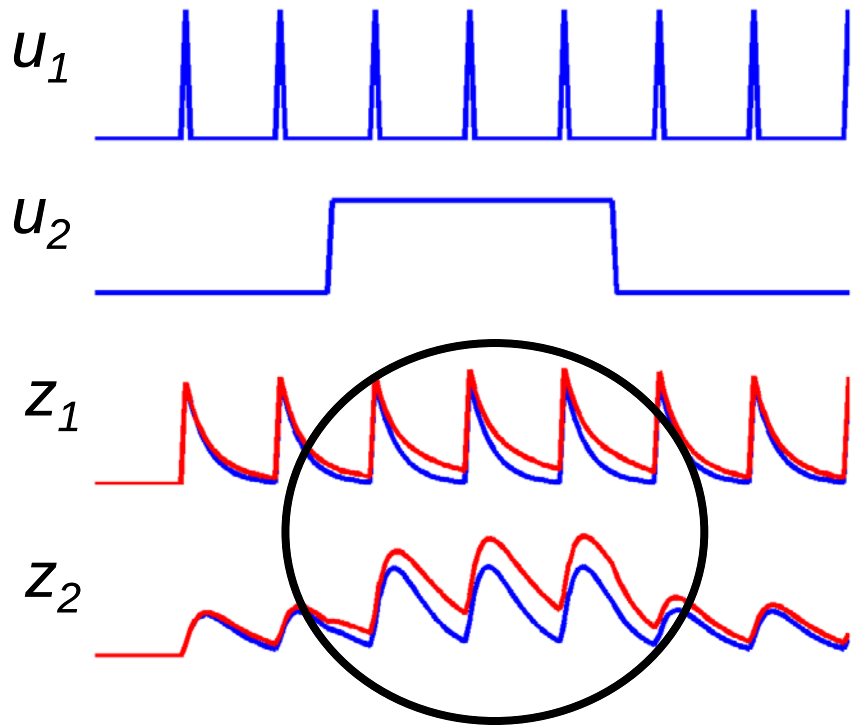




$$\begin{bmatrix}\dot{z}_1\\ \dot{z}_2\end{bmatrix} = s\begin{bmatrix}-1 & 0\\ a_{21} & -1\end{bmatrix}\begin{bmatrix}z_1\\ z_2\end{bmatrix} + u_2\begin{bmatrix}0 & 0\\ b_{21} & 0\end{bmatrix}\begin{bmatrix}z_1\\ z_2\end{bmatrix} + \begin{bmatrix}c\\ 0\end{bmatrix}u_1 \qquad a_{21}, b_{21} > 0$$

Neurodynamics:
...+ reciprocal connections





reciprocal connection disclosed by u_2

$$\begin{bmatrix}\dot{z}_1\\ \dot{z}_2\end{bmatrix} = s\begin{bmatrix}-1 & a_{12}\\ a_{21} & -1\end{bmatrix}\begin{bmatrix}z_1\\ z_2\end{bmatrix} + u_2\begin{bmatrix}0 & 0\\ b_{21} & 0\end{bmatrix}\begin{bmatrix}z_1\\ z_2\end{bmatrix} + \begin{bmatrix}c\\ 0\end{bmatrix}u_1$$

$$a_{12}, a_{21}, b_{21} > 0$$

Bilinear state equation

modulatory inputs

intrinsic connectivity — **modulatory connectivity** — **direct inputs** — **driving inputs**

$$\begin{bmatrix} \dot{z}_1 \\ \vdots \\ \dot{z}_n \end{bmatrix} = \left\{ \begin{bmatrix} a_{11} & \cdots & a_{1n} \\ \vdots & \ddots & \vdots \\ a_{n1} & \cdots & a_{nn} \end{bmatrix} + \sum_{j=1}^{m} u_j \begin{bmatrix} b_{11}^{(j)} & \cdots & b_{1n}^{(j)} \\ \vdots & \ddots & \vdots \\ b_{n1}^{(j)} & \cdots & b_{nn}^{(j)} \end{bmatrix} \right\} \begin{bmatrix} z_1 \\ \vdots \\ z_n \end{bmatrix} + \begin{bmatrix} c_{11} & \cdots & c_{1d} \\ \vdots & \ddots & \vdots \\ c_{n1} & \cdots & c_{nd} \end{bmatrix} \begin{bmatrix} u_1 \\ \vdots \\ u_d \end{bmatrix}$$

n regions — *m mod* inputs — *d drv* inputs

$$\dot{z} = (A + \sum_{j=1}^{m} u_j B^{(j)}) z + Cu$$

The haemodynamic “Balloon” model

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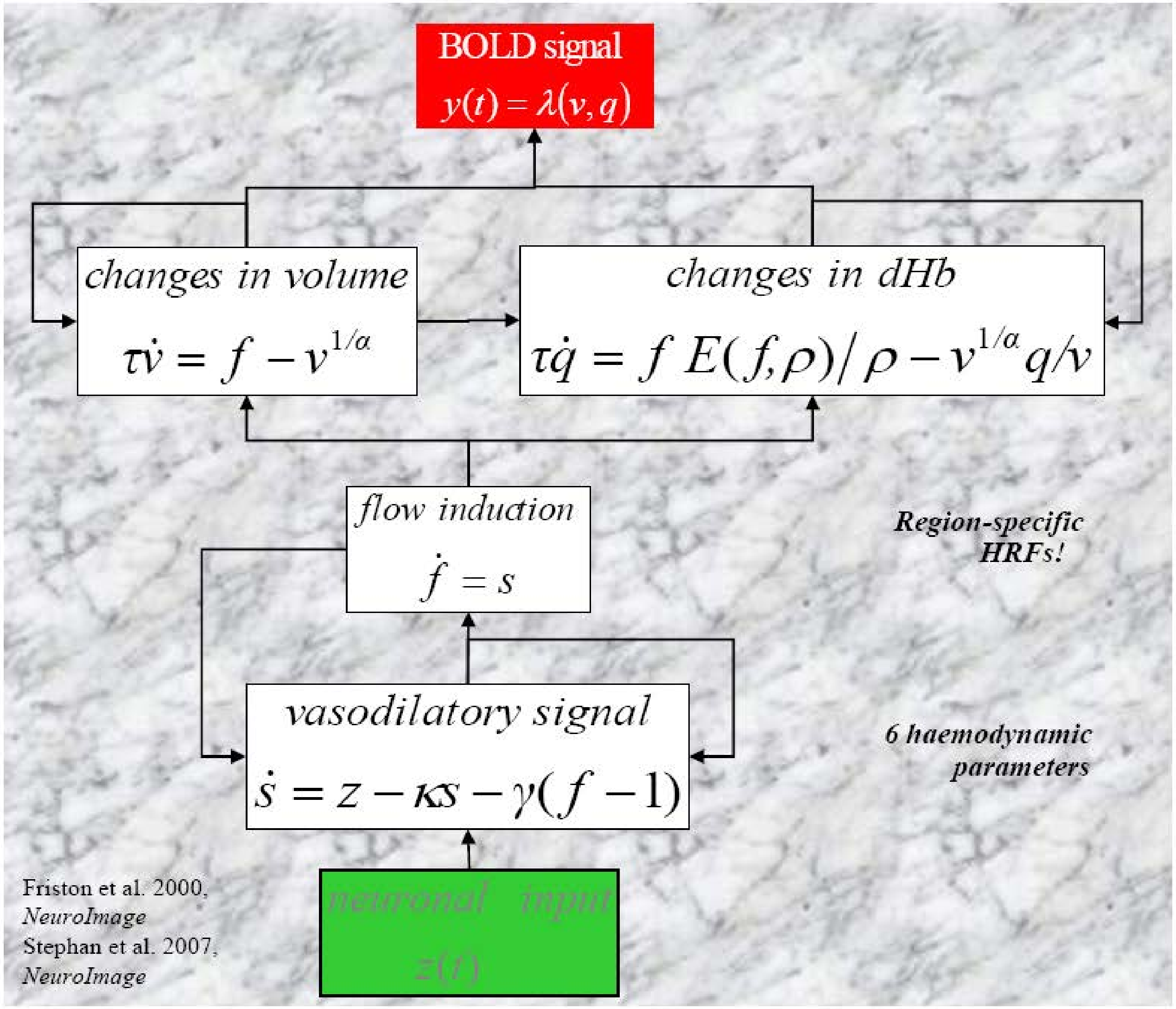
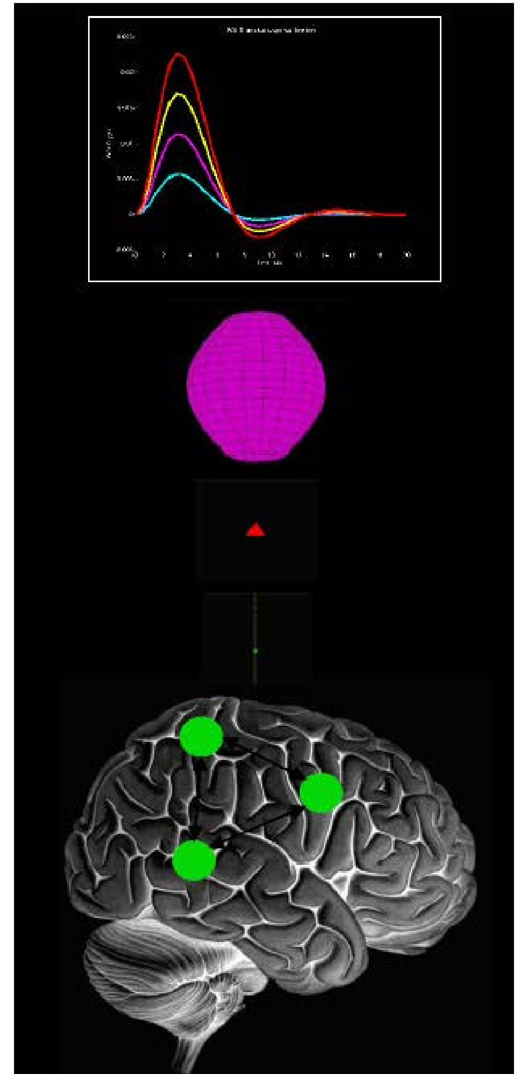
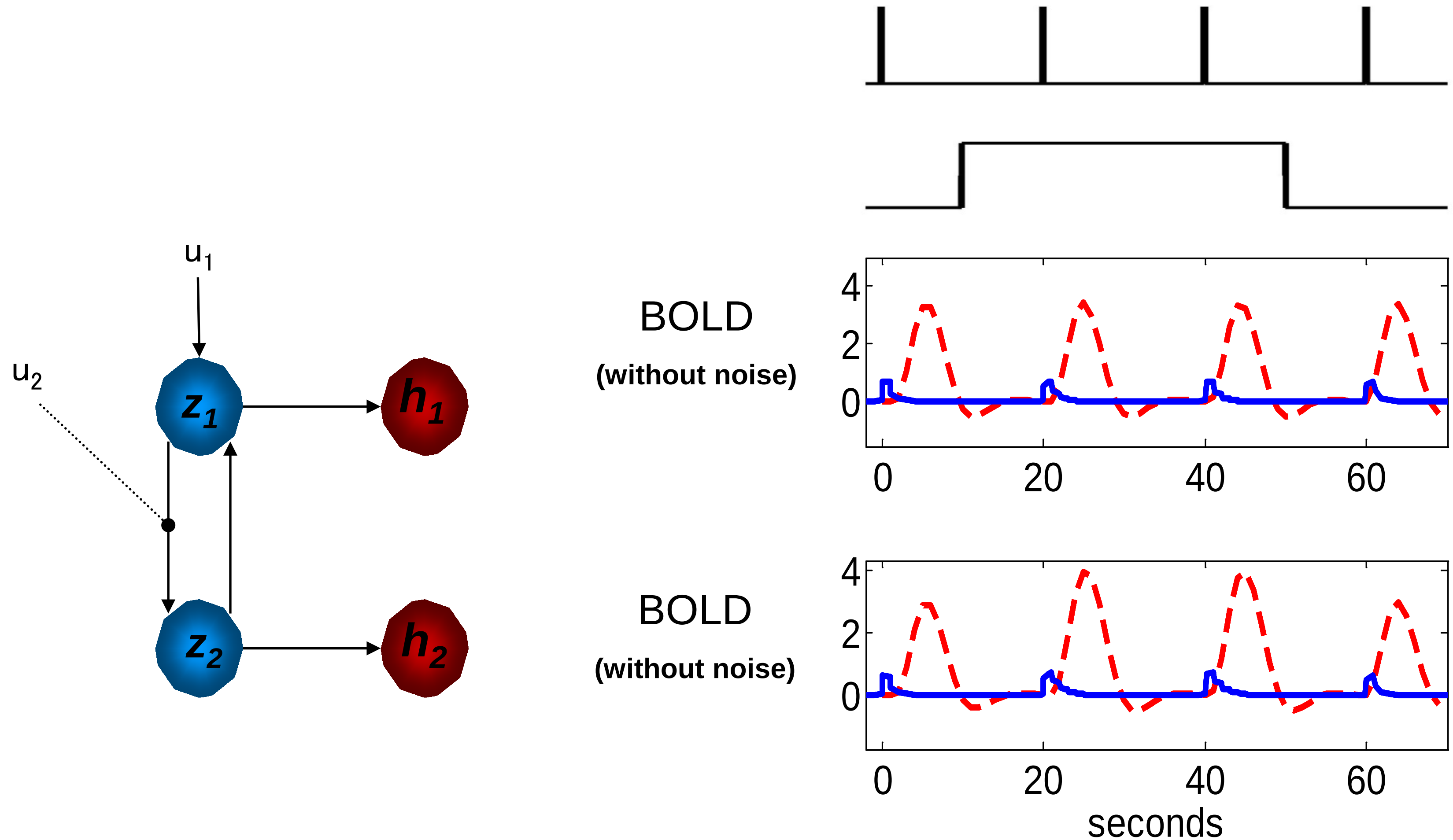




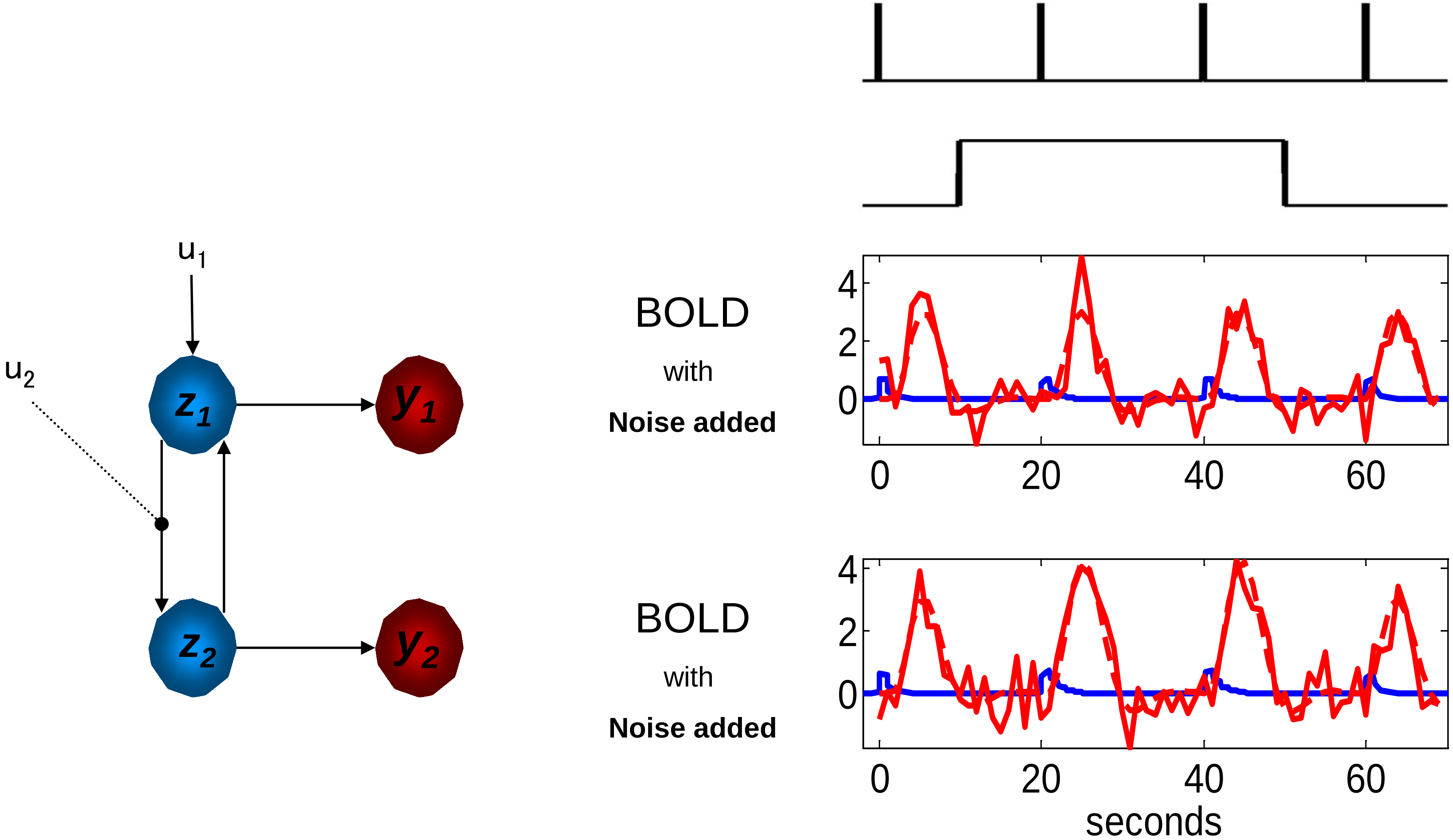
Haemodynamics: reciprocal connections



h(u,θ) represents the BOLD response (balloon model) to input

blue: neuronal activity
red: BOLD response

Haemodynamics: reciprocal connections



y represents simulated observation of BOLD response, i.e. includes noise

$$y = h(u, \theta) + e$$

Conceptual overview

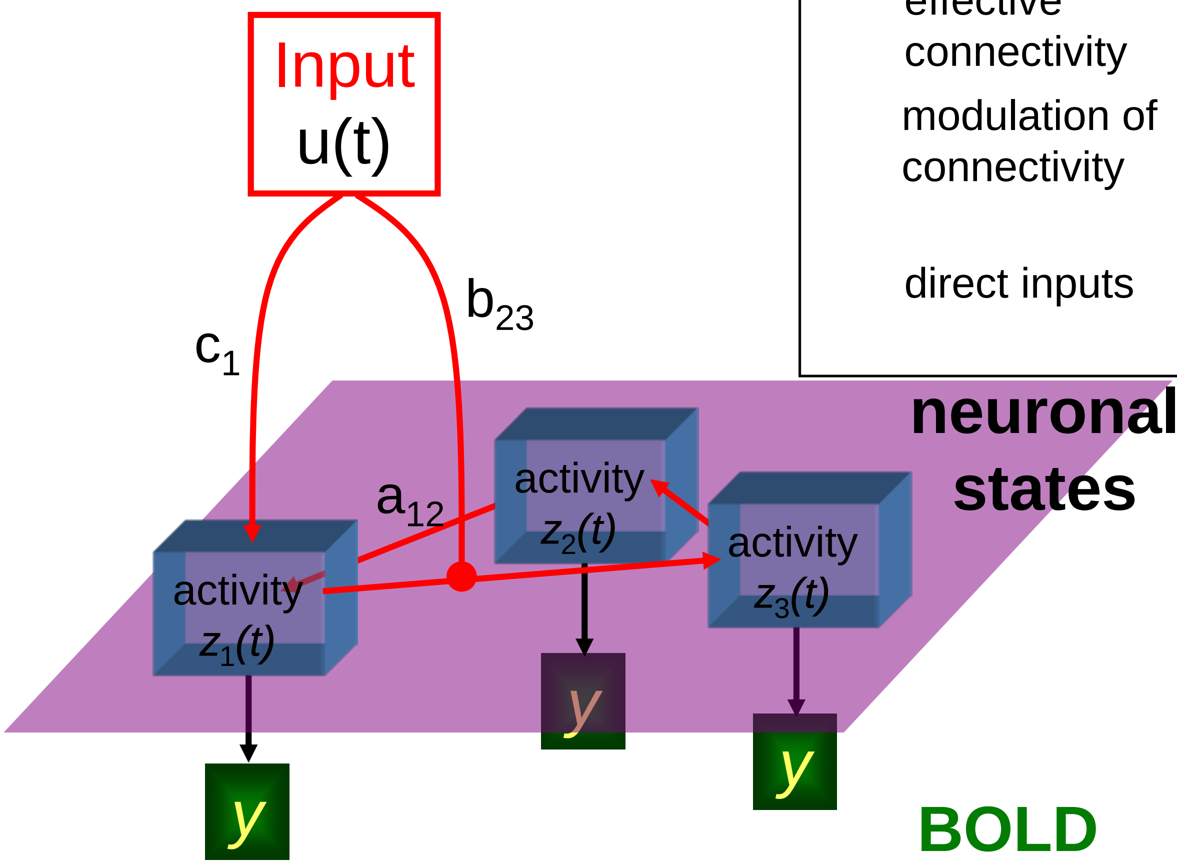
Neuronal state equation $\dot{z} = F(z, u, \theta^n)$

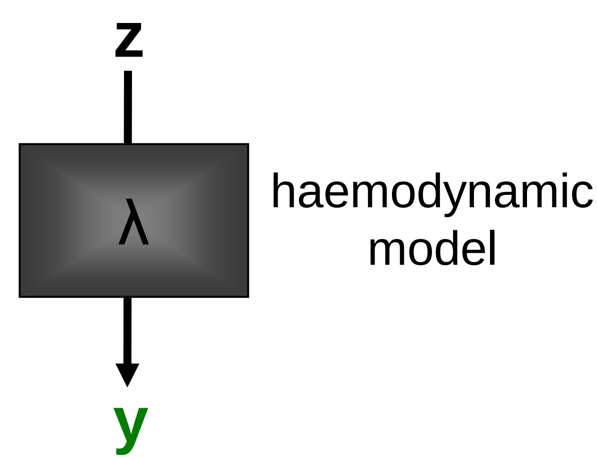
The bilinear model $\dot{z} = (A + \sum u_j B^j) z + Cu$

effective connectivity $$A = \frac{\partial F}{\partial z} = \frac{\partial \dot{z}}{\partial z}$$

modulation of connectivity $$B^j = \frac{\partial^2 F}{\partial z \partial u_j} = \frac{\partial}{\partial u_j} \frac{\partial \dot{z}}{\partial z}$$

direct inputs $$C = \frac{\partial F}{\partial u} = \frac{\partial \dot{z}}{\partial u}$$

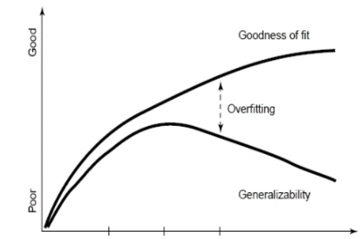




Friston et al. 2003, *NeuroImage*

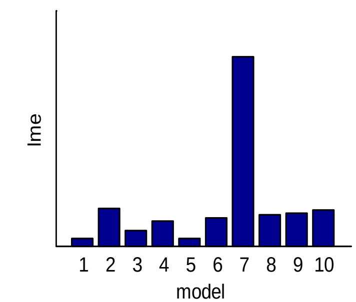
Inference on model space

Model evidence: The optimal balance of fit and complexity



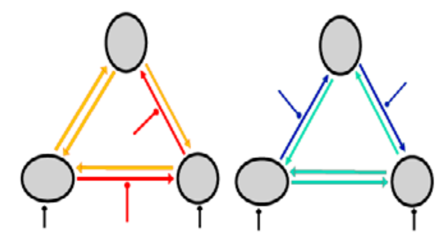
Comparing models

- Which is the best model?

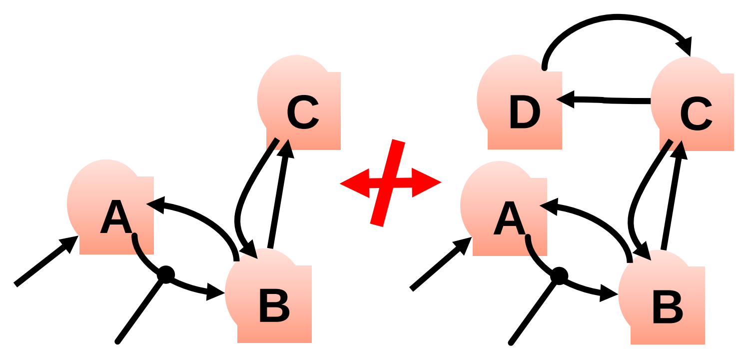


Comparing families of models

- What type of model is best?
  - Feedforward vs feedback
  - Parallel vs sequential processing
  - With or without modulation



Only compare models with the same data



Example DCM:
Attention to motion

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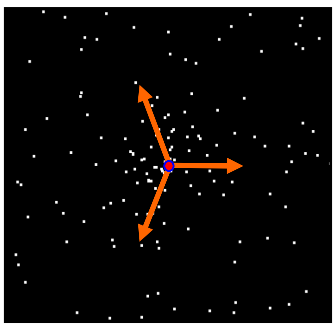
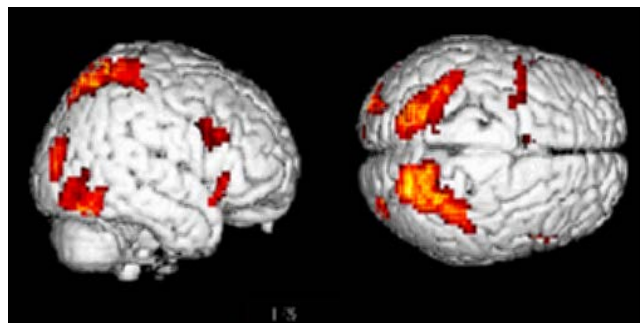
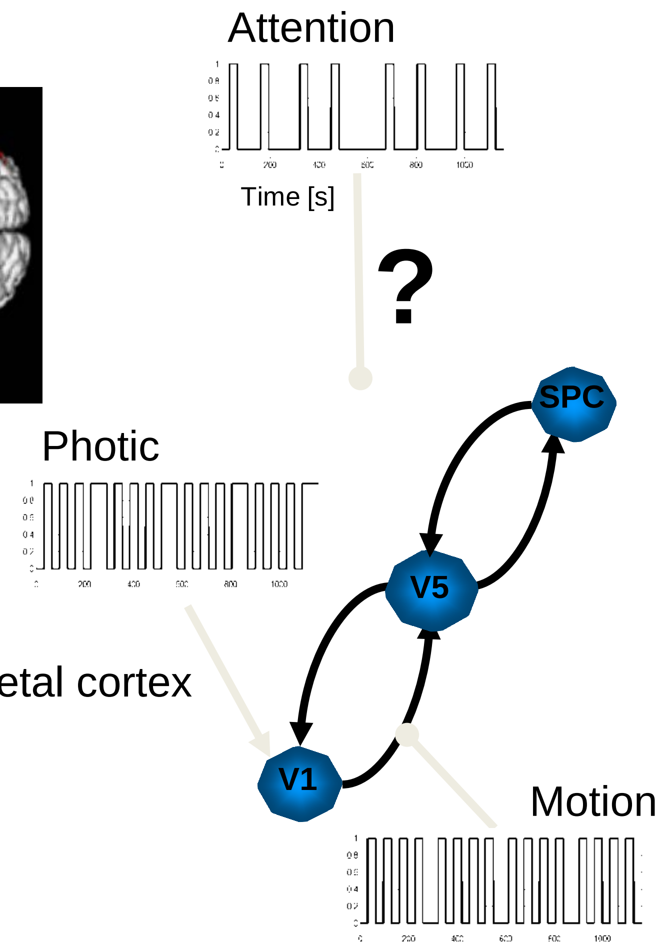
What is site of ***attention modulation*** during *visual motion processing*





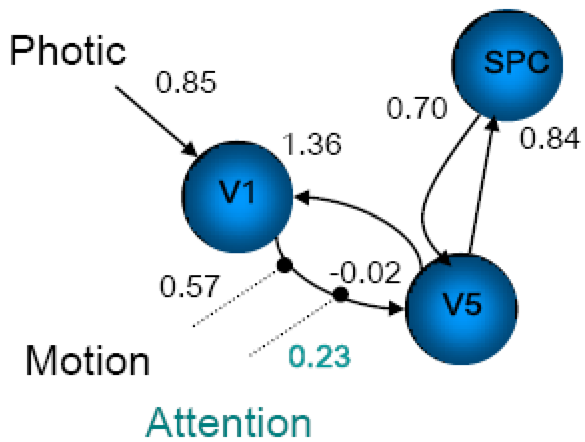
- fixation only		
- observe static dots	+ photic	→ V1
- observe moving dots	+ motion	→ V5
- task on moving dots	+ attention	→ V5 + parietal cortex



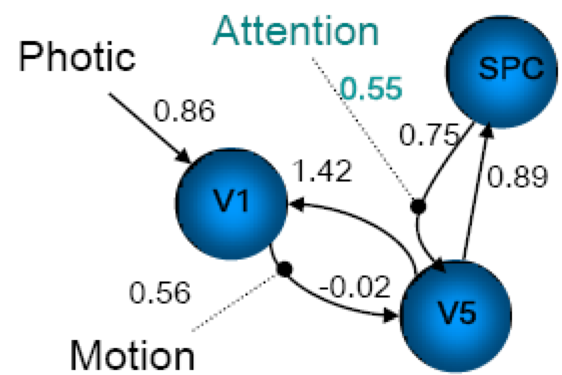
Friston et al. 2003, *NeuroImage*

Example DCM: Attention to motion

Model 1:
attentional modulation of V1→V5



Model 2:
attentional modulation of SPC→V5



Bayesian model selection: Model 1 better than model 2

$$\log p(y \mid m_1) >> \log p(y \mid m_2)$$

→ attention primarily modulates V1→V5 (in these data)

So, DCM….

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- enables one to **infer hidden neuronal processes**
- allows one to **test mechanistic hypotheses** about observed effects
  - uses a deterministic differential equation to model neuro-dynamics (represented by matrices A, B and C)
- is informed by anatomical and physiological principles
- uses a **Bayesian framework** to estimate model parameters
- is a generic approach to modelling experimentally perturbed dynamic systems
  - provides an observation model for neuroimaging data, e.g. fMRI, M/EEG
  - DCM is **not model or modality specific** (models will change and the method extended to other modalities e.g. LFPs)

Variants of DCM

- DCM for fMRI
  - "non-linear" DCM: modulatory input (B) equal to activity in another region
  - "two-state" DCM: inhibitory and excitatory neuronal subpopulations
  - "stochastic" DCM: random element to activity (e.g, for resting state)

- DCM for E/MEG
  - "evoked" responses (complex neuronal model based on physiology)
  - "induced" responses (within/across frequency power coupling; no physiological model (more like DCM for fMRI))
  - "steady-state" responses
  - with (e.g, EEG/MEG) or without (e.g, LFP, iEEG) a forward (head) model

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Functional/Effective Connectivity for M/EEG

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Functional Connectivity Background

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- Much interest in functional connectivity in fMRI
- And yet many neural interactions (e.g, coupled oscillations) occur at a timescale faster than visible by fMRI
- So, real promise of MEG/EEG is functional connectivity?

Talk Overview

1. Problem of Field Spread (Volume Conduction)
2. Linear vs Nonlinear measures
3. Directed vs Undirected measures
4. Direct vs Indirect measures
5. Generative Models

Field Spread Problem

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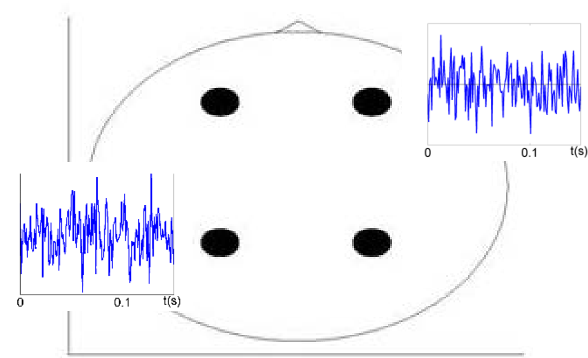
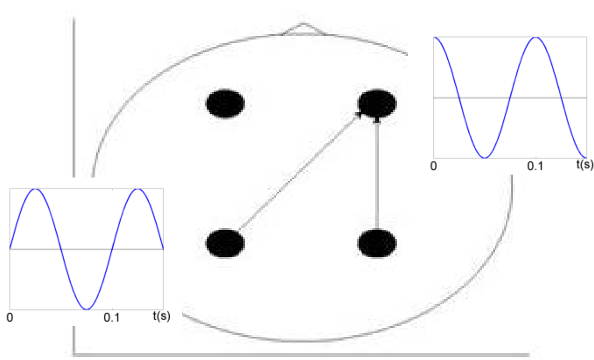
Many (zero-lag) measures of functional connectivity between sensors can be spurious, i.e, reflect activity from single source

No true source connectivity



True source connectivity



Field Spread Problem

Source reconstruction reduces field spread problem…

…and allows easier comparison with fMRI connectivity

BUT spurious connections between sources can remain ("point-spread")

Hillebrand et al (2012) Neuroimage

One approach is to orthogonalise raw data, then correlate (0-lag) power envelopes…

Colclough et al (2015) Neuroimage

…another uses fact that field-spread is instantaneous, so time- or phase-lagged measures are immune to field spread (though assume no true zero-lag connectivity)

Different Types of Connection

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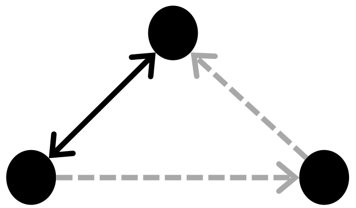
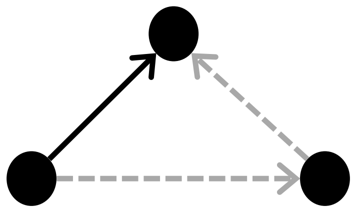
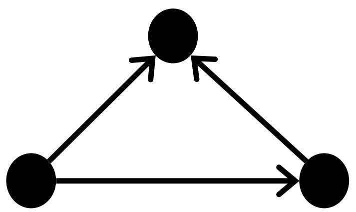
Undirected, Indirect (bivariate)



Directed, Indirect (bivariate)



Directed, Direct (multivariate)
(“effective connectivity”)



Cross-Correlation

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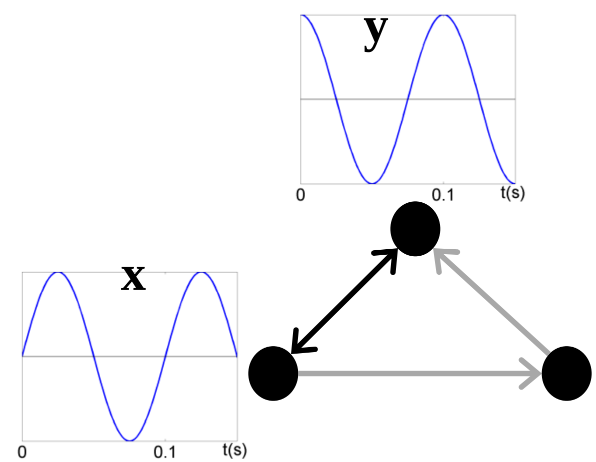
Undirected, Indirect, Linear (sensitive to Field-spread when $l=0$)

$$c_{xy}(l) = \left\langle \left(x_t - \overline{x}\right)\left(y_{t+l} - \overline{y}\right) \right\rangle_t$$

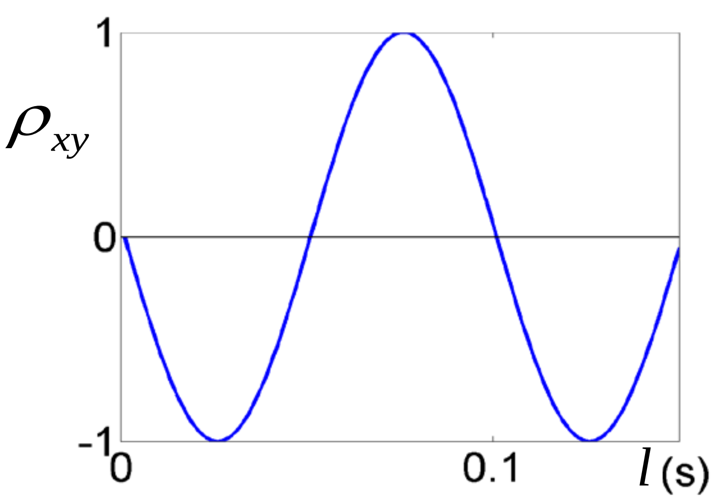
Cross-covariance

l = *"lag"*



$$\rho_{xy}(l) = \frac{c_{xy}(l)}{\sigma_x \sigma_y}$$

Cross-correlation

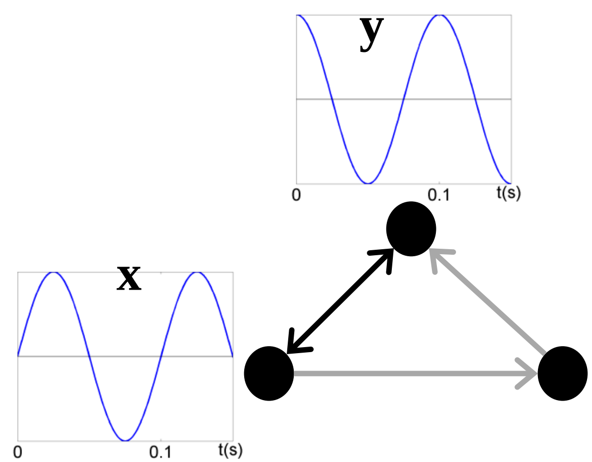


Coherency
(Fourier transform of cross-covariance)

Undirected, Indirect, Linear, sensitive to Field-spread

$$c_{xy}(l) = \left\langle \left(x_t - \overline{x}\right)\left(y_{t+l} - \overline{y}\right) \right\rangle_t$$

Cross-covariance

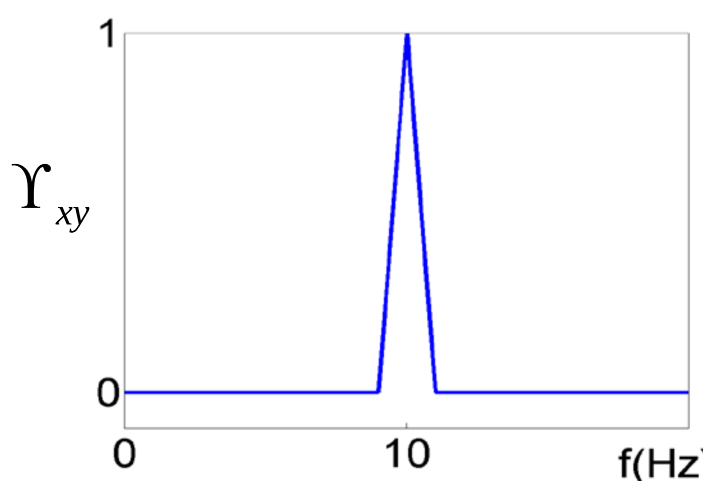


$$C_{xy}(f) = \sum_l c_{xy}(l) e^{-2\pi i.l.f}$$

Coherency

$$\Upsilon_{xy}(f) = \frac{\left|C_{xy}(f)\right|^2}{\left|C_{xx}(f)\right|\left|C_{yy}(f)\right|}$$

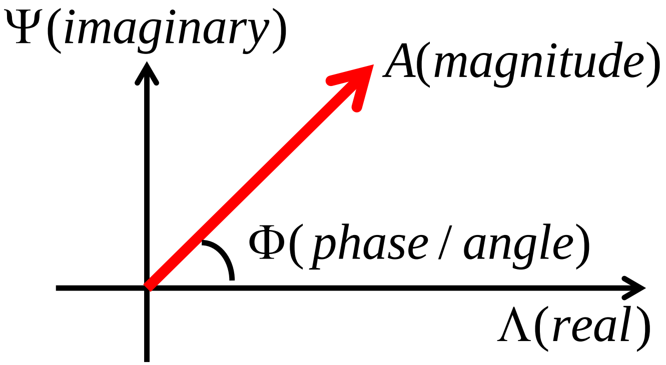
(Magnitude-squared) Coherence



Digression on Complex Numbers

An oscillation of frequency *f* can be represented in terms of amplitude and phase (polar coordinates), which can also be represented by a complex number

$$C(f) = A(f)e^{i\Phi(f)}$$
$$= \Lambda(f) + i\Psi(f)$$



$$A(f) = |C(f)| = \sqrt{\Lambda^2(f) + \Psi^2(f)}$$

$$\Phi(f) = \arctan(\Psi(f)/\Lambda(f))$$

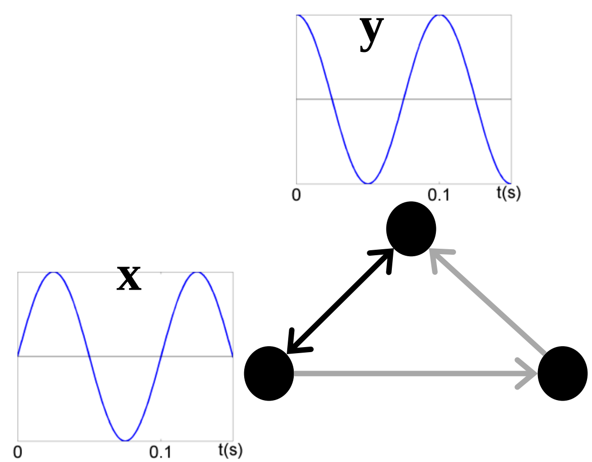
Coherence

MRC Cognition and Brain Sciences Unit

Undirected, Indirect, Linear, sensitive to Field-spread

$$c_{xy}(l) = \left\langle \left(x_t - \overline{x} \right) \left(y_{t+l} - \overline{y} \right) \right\rangle_t$$

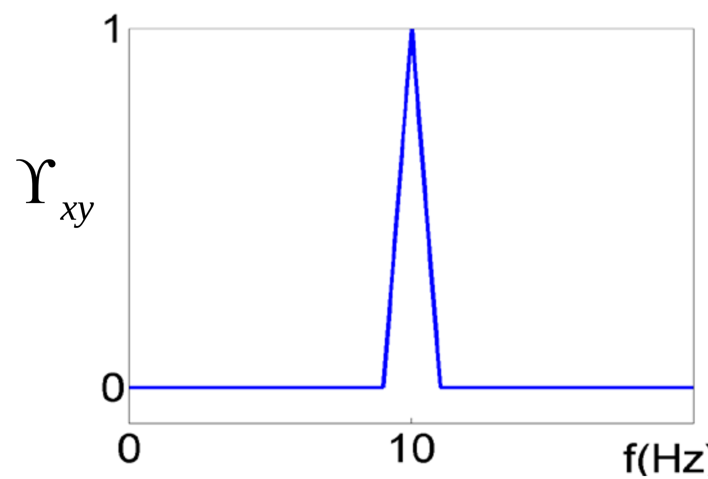
Cross-covariance



$$C_{xy}(f) = \sum_l c_{xy}(l) e^{-2\pi i.l.f}$$

Coherency

$$\Upsilon_{xy}(f) = \frac{\left| C_{xy}(f) \right|^2}{\left| C_{xx}(f) \right| \left| C_{yy}(f) \right|}$$



(Magnitude-squared) Coherence

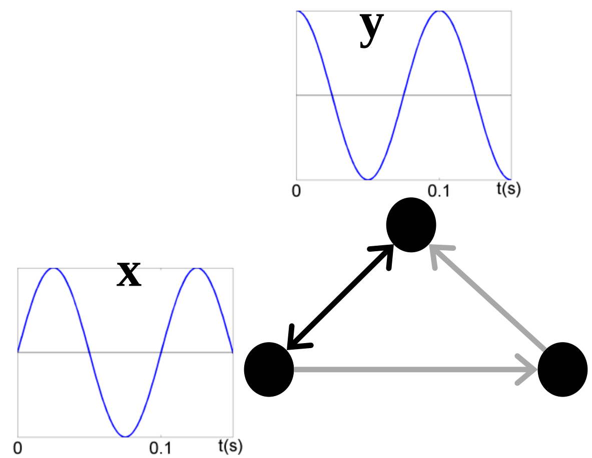
Imaginary Coherency

Undirected, Indirect, Linear, immune to Field-spread

$$c_{xy}(l) = \left\langle \left(x_t - \overline{x}\right)\left(y_{t+l} - \overline{y}\right) \right\rangle_t$$

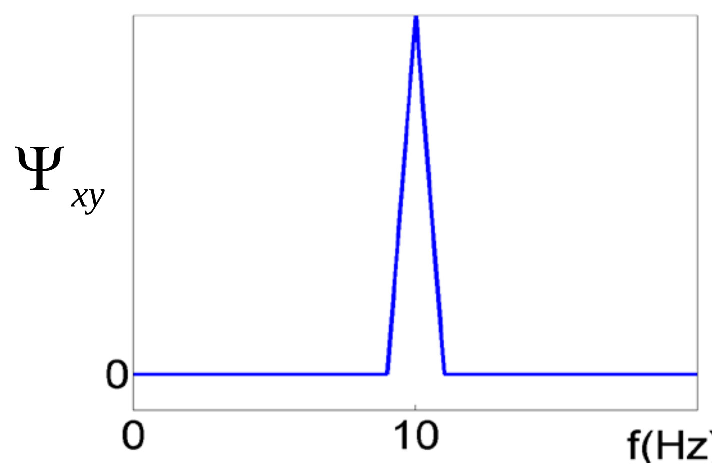
$$C_{xy}(f) = \sum_l c_{xy}(l) e^{-2\pi i.l.f}$$

Coherency



$$\Psi_{xy}(f) = imag(C_{xy}(f))$$

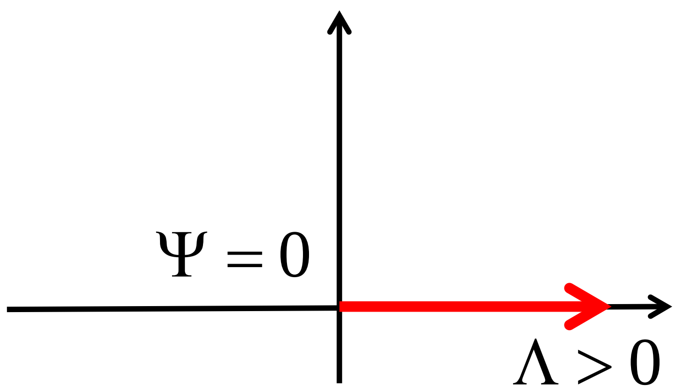
Imaginary Coherency

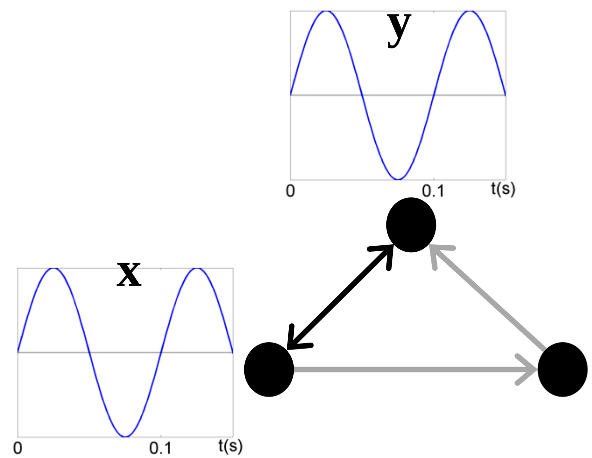


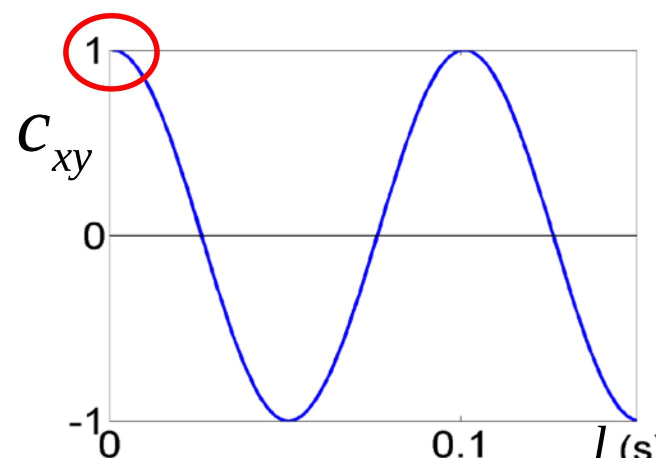
Nolte et al (2004) Clin Neurophys

Imaginary Coherency

A zero imaginary component implies a phase of the coherency of either 0º or 180º, which could be caused by field-spread…







$$\Psi_{xy}(f) = imag(C_{xy}(f))$$

Nolte et al (2004) Clin Neurophys

Imaginary Coherency

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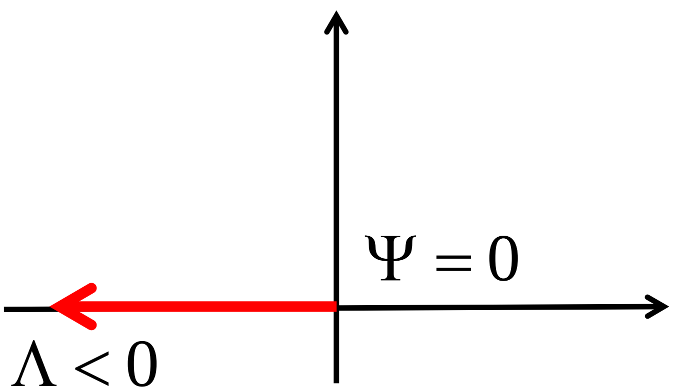
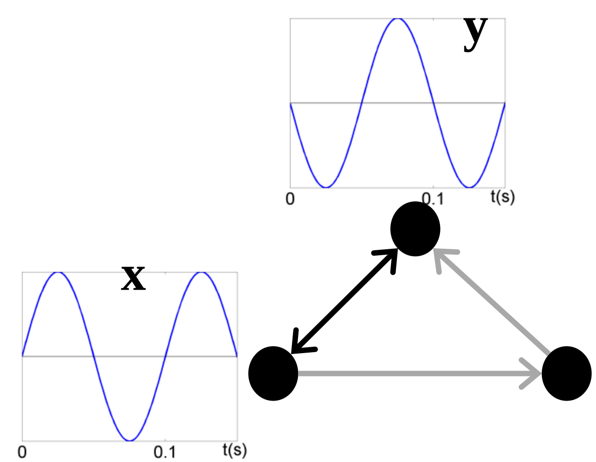
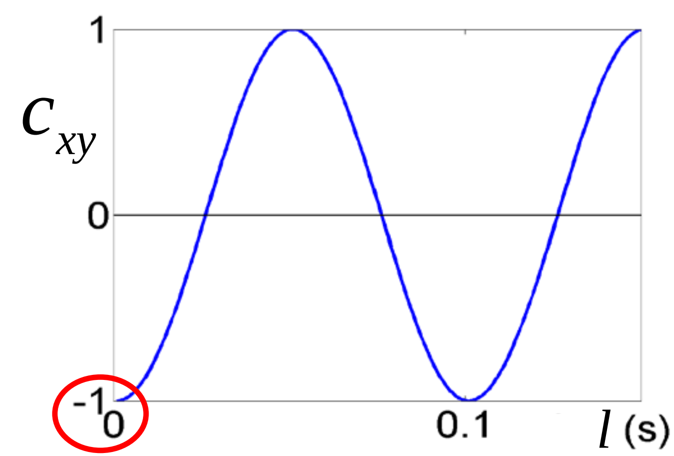
A zero imaginary component implies a phase of the coherency of either 0º or 180º, which could be caused by field-spread…





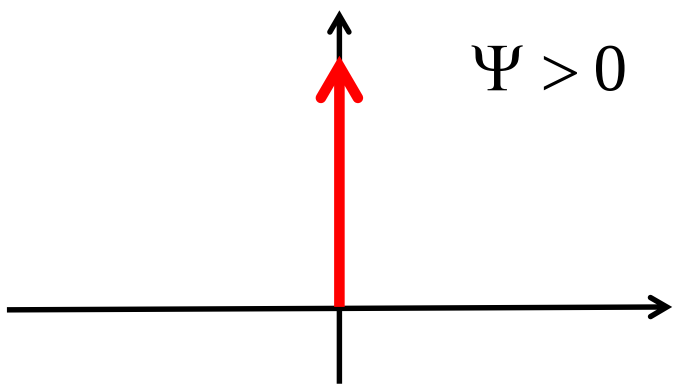


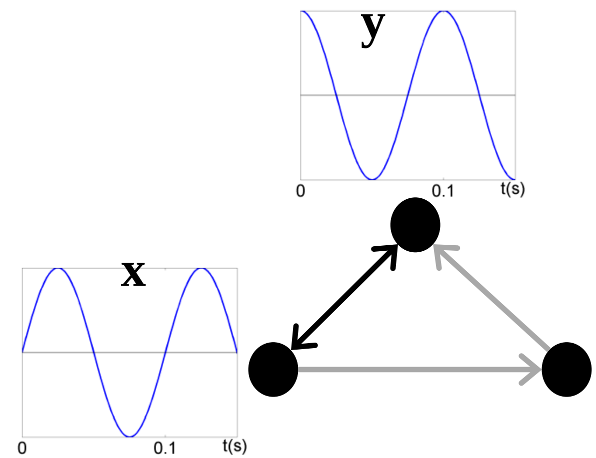
$$\Psi_{xy}(f) = imag(C_{xy}(f))$$

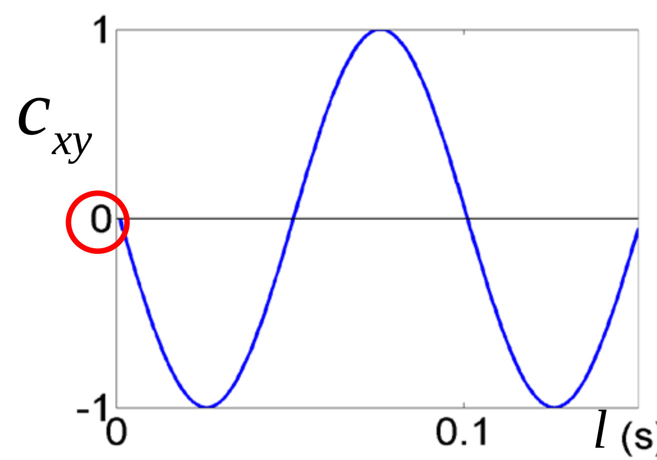
Nolte et al (2004) Clin Neurophys

Imaginary Coherency

…whereas a NON-zero imaginary component implies a phase of the coherency other than 0º or 180º, which can NOT be caused by field-spread







$$\Psi_{xy}(f) = imag(C_{xy}(f))$$

Nolte et al (2004) Clin Neurophys

Digression on Analytic Signals

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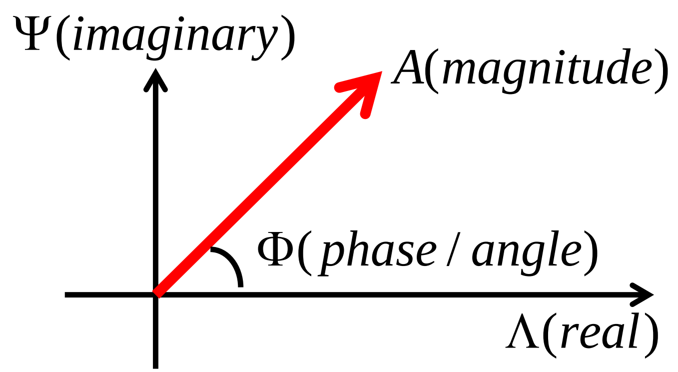
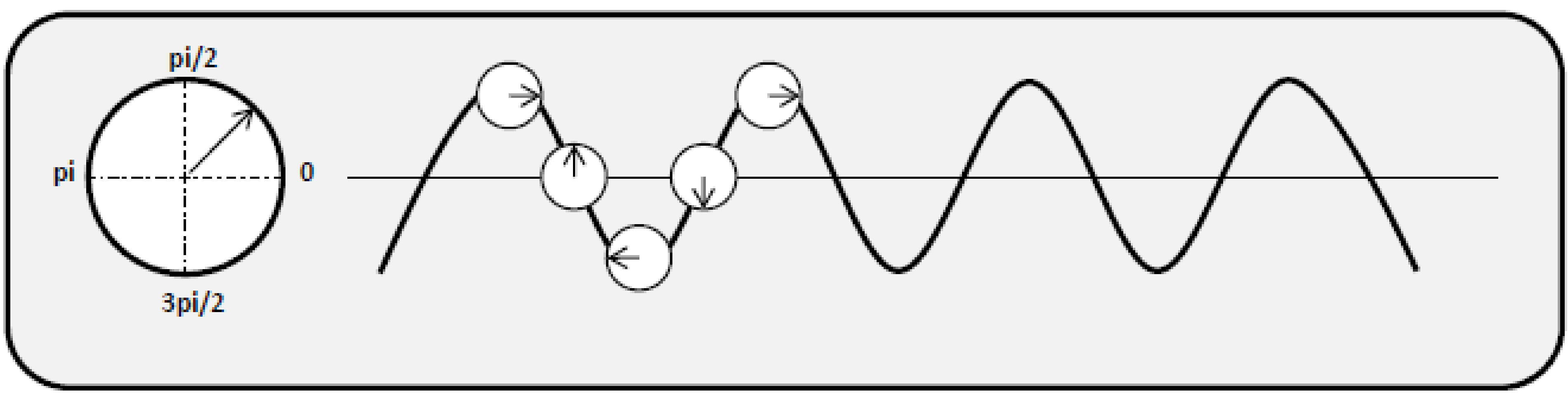
A signal can be represented analytically in terms of its amplitude and phase over time (within a narrow frequency band) (e.g, using Hilbert transform)

$$x(t,f) = A(t,f)e^{i\Phi(t,f)}$$





Phase-related Measures

MRC Cognition and Brain Sciences Unit

Undirected, Indirect, Linear, immune to Field-spread (when $\Delta\Phi \neq 0$)

$$x(t) = A_x(t)e^{i\Phi_x(t)}$$

$$y(t) = A_y(t)e^{i\Phi_y(t)}$$

$$\Delta\Phi(t) = \Phi_x(t) - \Phi_y(t)$$

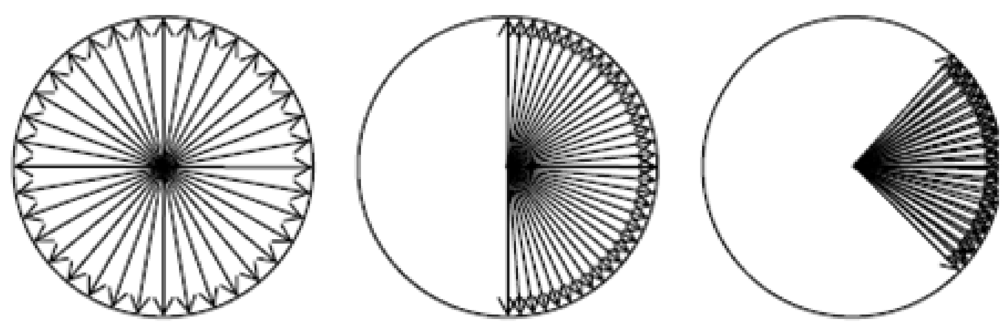
$$PLV = \left\langle e^{i\Delta\Phi(t)} \right\rangle_t$$

Phase-Locking Value

$$PLI = \left\langle sign(\Delta\Phi(t)) \right\rangle_t$$

Phase-Lag Index

PLV=0 *PLV=0.5* *PLV=0.75*



Stam et al (2007) Human Brain Mapp

Cross-frequency coupling

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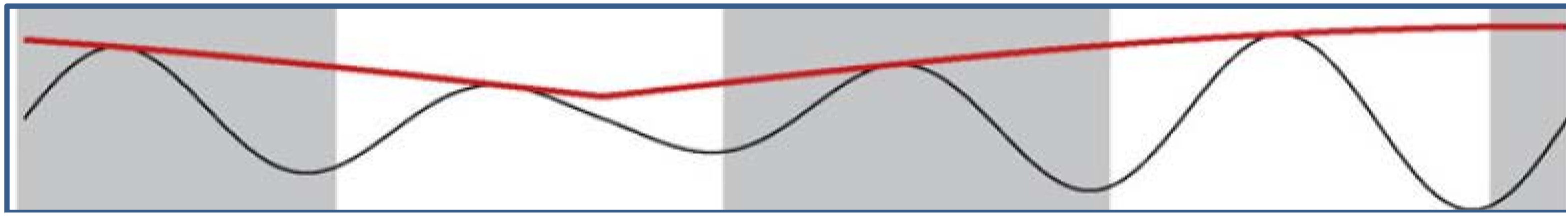
$x(t)$



$y(t)$

Power-Power
$A_x(t):A_y(t)$

Phase-Phase
$\Phi_x(t):\Phi_y(t)$

Phase-Freq
$\Phi_x(t):F_y(t)$

Phase-Power
$\Phi_x(t):A_y(t)$

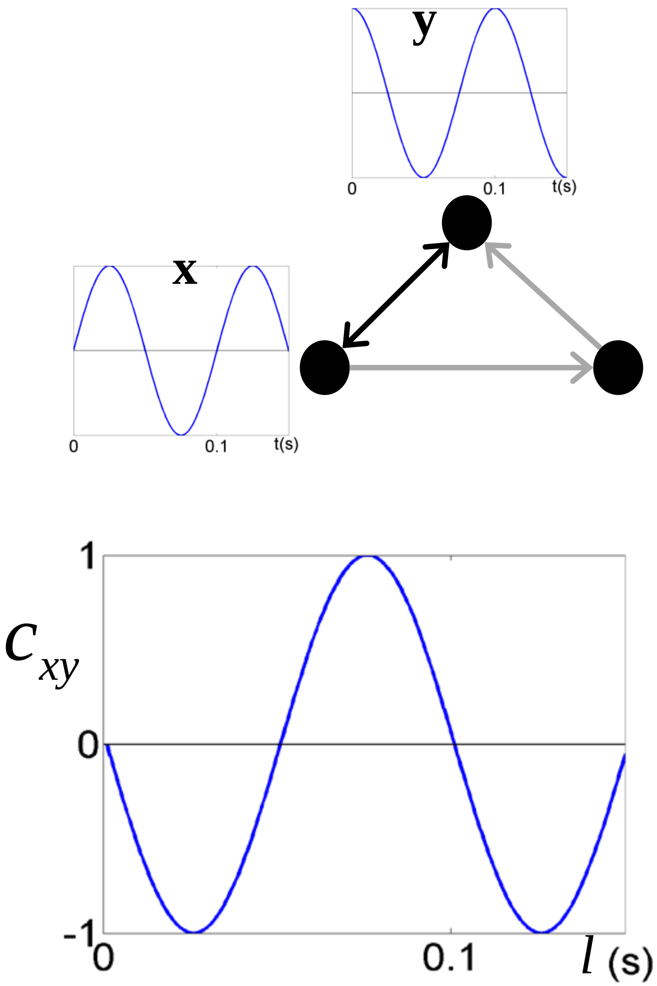
Jenson & Colgin (2007) TICS

Talk Overview

MRC | Cognition and Brain Sciences Unit

1. Problem of Field Spread (Volume Conduction)
2. Linear vs Nonlinear measures
3. Directed vs Undirected measures
4. Direct vs Indirect measures
5. Generative Models

Nonlinear Measures



Nonlinear Measures

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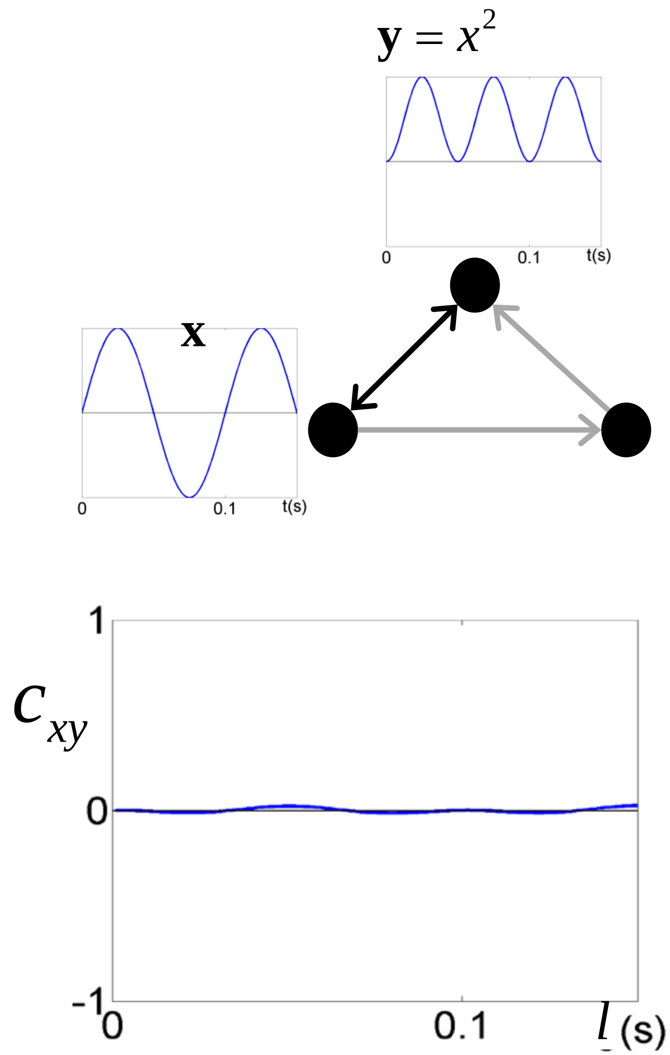
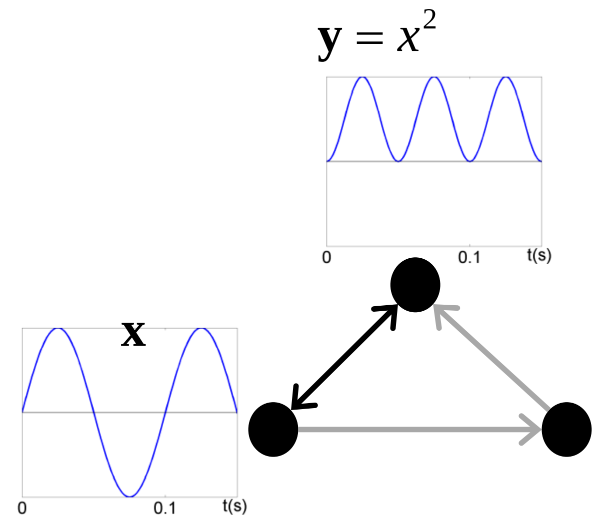
Cross-correlation/coherence insensitive to nonlinear dependencies

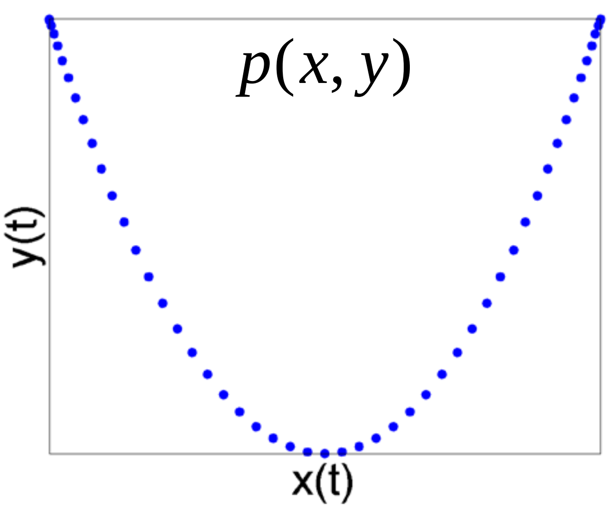


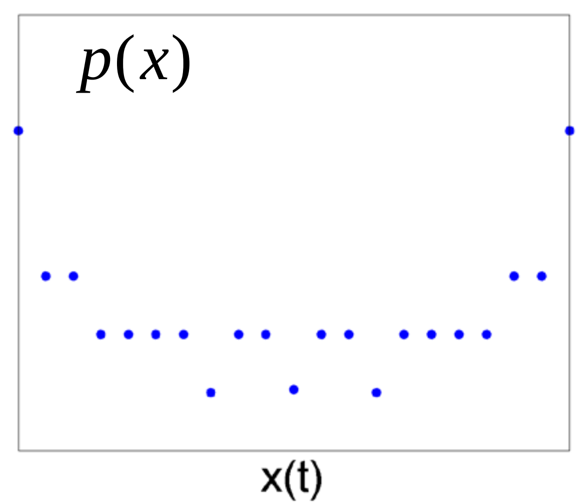
Mutual Information

Cognition and Brain Sciences Unit

$$MI(x,y) = \sum_{x,y} p(x,y) \log\left(\frac{p(x,y)}{p(x)\,p(y)}\right)$$



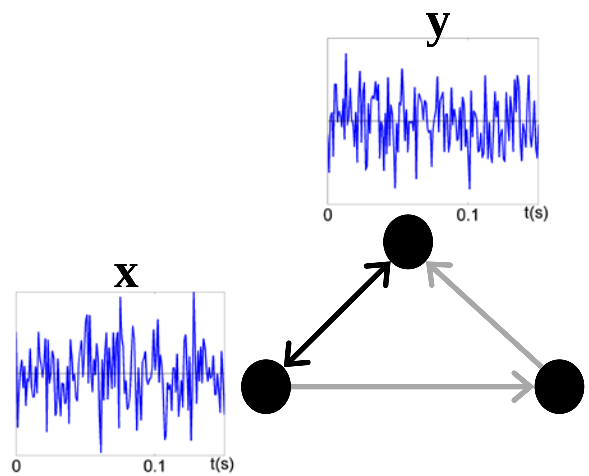


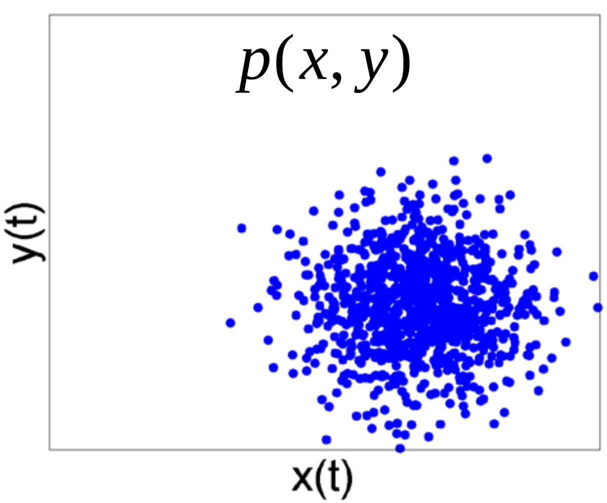


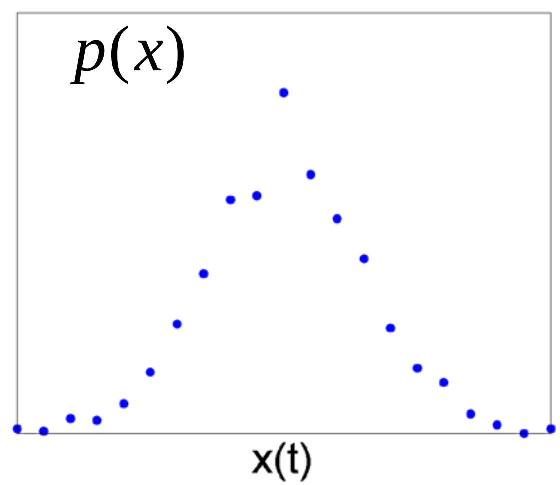
Mutual Information

Sensitive to Field-spread, Undirected, Indirect, Nonlinear

$$MI(x,y) = \sum_{x,y} p(x,y) \log\left(\frac{p(x,y)}{p(x)\,p(y)}\right)$$







Talk Overview

MRC | Cognition and Brain Sciences Unit

1. Problem of Field Spread (Volume Conduction)

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5. Generative Models

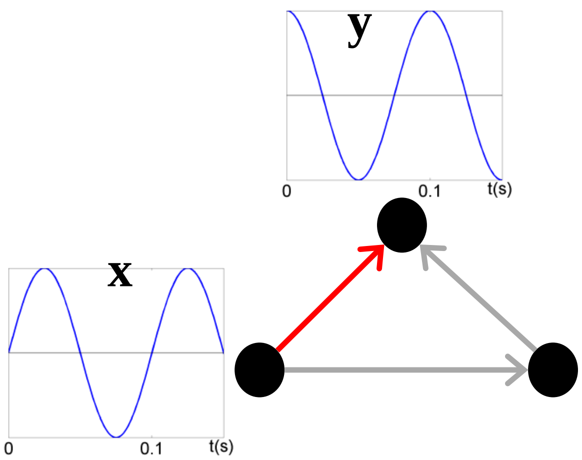
(bivariate) Granger Causality

Immune to Field-spread, Directed, Indirect, Linear

Auto-regressive model to order *p*
(assuming mean-corrected, with residuals *e*)

$$y_y(t) = a_1 y(t-1) + \ldots + a_p y(t-p) + e(t)$$

$$= \sum_{l=1}^{p} a_l y(t-l) + e(t)$$



Augmented model including past values of *x* (to order *q*)

$$y_{y \leftarrow x}(t) = \sum_{l=1}^{p} a_l y(t-l) + \sum_{l=1}^{q} b_l x(t-l) + e(t)$$

If classical F-test shows *b* parameters are non-zero, then *x* “Granger-causes” *y*
(special case of MVAR; see later)

Directed, Nonlinear Measures

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Transfer Entropy (lagged generalisation of mutual information)

Immune to Field-spread, Directed, Indirect, Nonlinear

$$TE_{y \to x}(l) = \sum_{x_{n+l}, x_n, y_n} p(x_{n+l}, x_n, y_n) \log\left(\frac{p(x_{n+l} \mid x_n, y_n)}{p(x_{n+l} \mid x_n)}\right)$$

$$TE_{x \to y}(l) = \sum_{y_{n+l}, y_n, x_n} p(y_{n+l}, x_n, y_n) \log\left(\frac{p(y_{n+l} \mid x_n, y_n)}{p(y_{n+l} \mid y_n)}\right)$$

Schreiber (2000) Phys Rev Letters

Generalised Synchronisation

Sensitive to Field-spread, Directed, Indirect, Nonlinear

$$\mathbf{x}_t = [x_t, x_{t+l}, \ldots, x_{t+(m-1)l}]$$

$$\mathbf{y}_t = [y_t, y_{t+l}, \ldots, y_{t+(m-1)l}]$$

$$S(x \mid y) = \frac{1}{N} \sum_{t=1}^{N} \frac{D_t(x)}{D_t(x \mid y)}$$

m is the embedding dimension and *l* lag

D is the Euclidean distance between x_t and embedded neighbours

Quian Quiroga et al (2000) Phys Rev E

Talk Overview

MRC | Cognition and Brain Sciences Unit

1. Problem of Field Spread (Volume Conduction)
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Direct Measures

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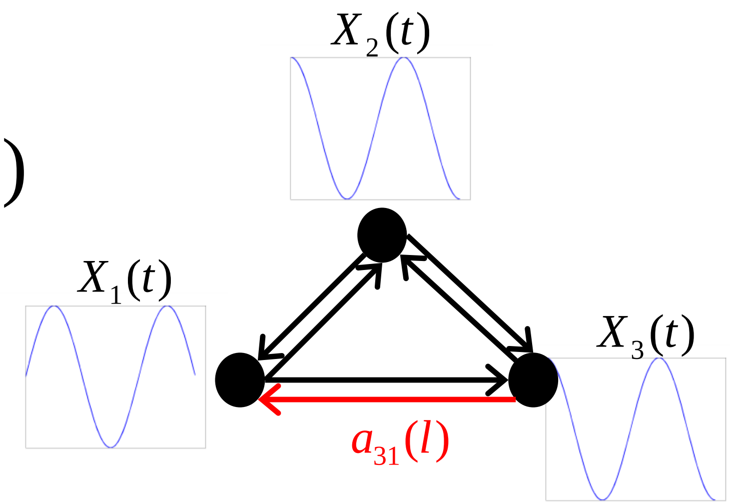
Multivariate Autoregressive Modelling (MVAR)

Immune to Field-spread, Directed, Direct, Linear

$$X_i(t) = \sum_{j=1}^{N} \sum_{l=1}^{p} a_{ij}(l) X_j(t-l) + u_i(t)$$



Various summary measures, eg,
Partial Directed Coherence (PDC):

$$PDC_{ij}(f) = \frac{A_{ij}(f)}{\sqrt{\sum_{k=1}^{M} \left| A_{kj}(f) \right|^2}}$$

$$A_{ij}(f) = F(a_{ij}(l))$$

Generalised form of Granger Causality

Though insensitive to true zero-lag dependencies (occur in reality?)

Baccala & Sameshima (2001) Biol Cybernet

Talk Overview

1. Problem of Field Spread (Volume Conduction)

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Generative Models

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Immune to Field-spread, Directed, Direct, Nonlinear, model-driven

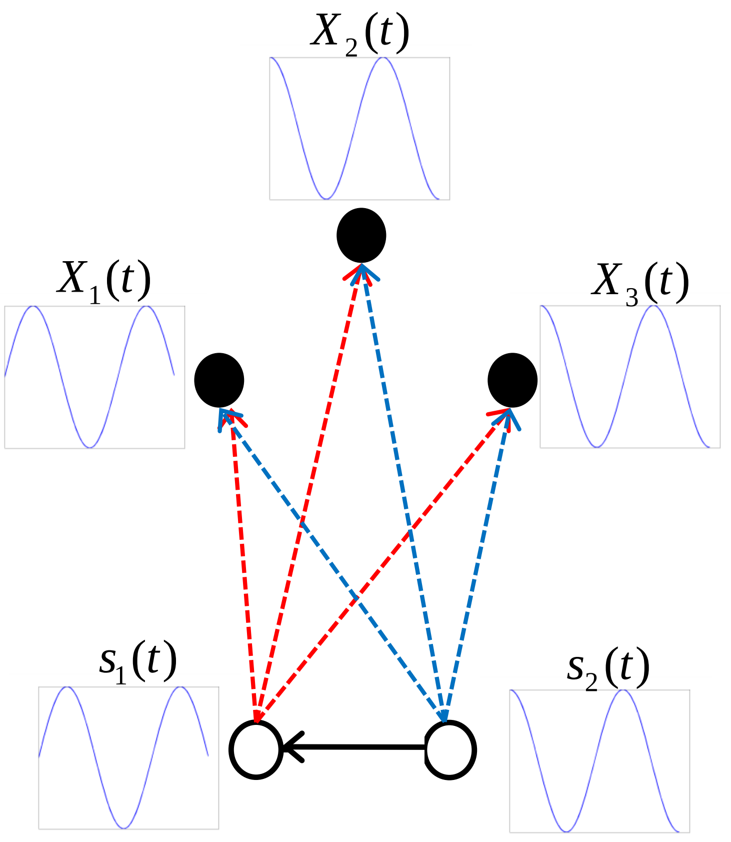
Connectivity modelled between sources

Projected to sensors via headmodel

Typically a handful of sources, and a range of networks fit to data

Bayesian methods for comparing which network model is best

Dynamic Causal Modelling (DCM) is one approach



Chen et al, 2009, Neuroimage

Measure	Immume to Field Spread	Directed	Nonlinear	Direct
Cross-Correlation	Y (l>0)	N	N	N
Coherence	Y (imaginary)	N	N	N
PLV/PLI	Y	N	N	N
Granger (bivariate)	Y	Y	N	N
Mutual Information	N	N	Y	N
Generalised Synchrony	N	Y	Y	N
Transfer Entropy	Y	Y	Y	N
MVAR (eg, PDC)	Y	Y	N	Y
Generative (eg, DCM)	Y	Y	Y	Y

The End

DCM Neural Level

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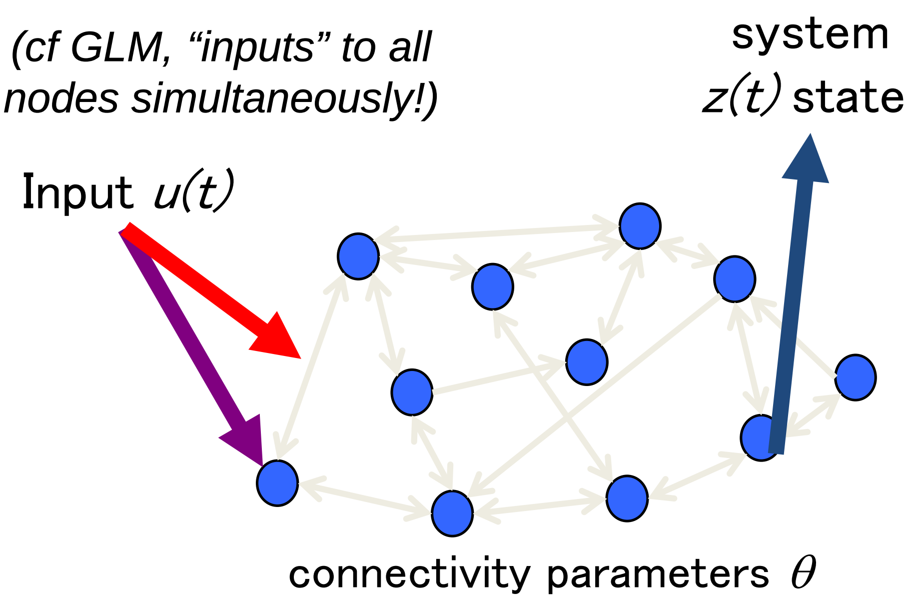
System changes depend on:

- the current state z
- the connectivity θ
- external inputs u
  - driving (to nodes)
  - modulatory (on links)
- time constants & delays

(cf GLM, "inputs" to all nodes simultaneously!)



$$\frac{dz}{dt} = F(z, u, \theta)$$

DCM Estimation: Bayesian framework

Models of
•Haemodynamics in a single region
•Neuronal interactions

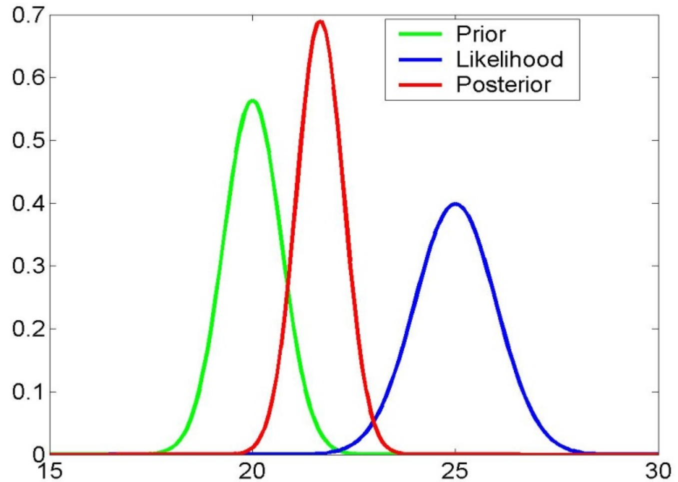
Constraints on
•Haemodynamic parameters
•Connections

likelihood $p(y \mid \theta)$

$p(\theta)$ priors

posterior $p(\theta \mid y) \propto p(y \mid \theta)\, p(\theta)$

Bayesian estimation



Inferences on:

1. *Parameters*
2. *Models*

Parameter estimation: an example

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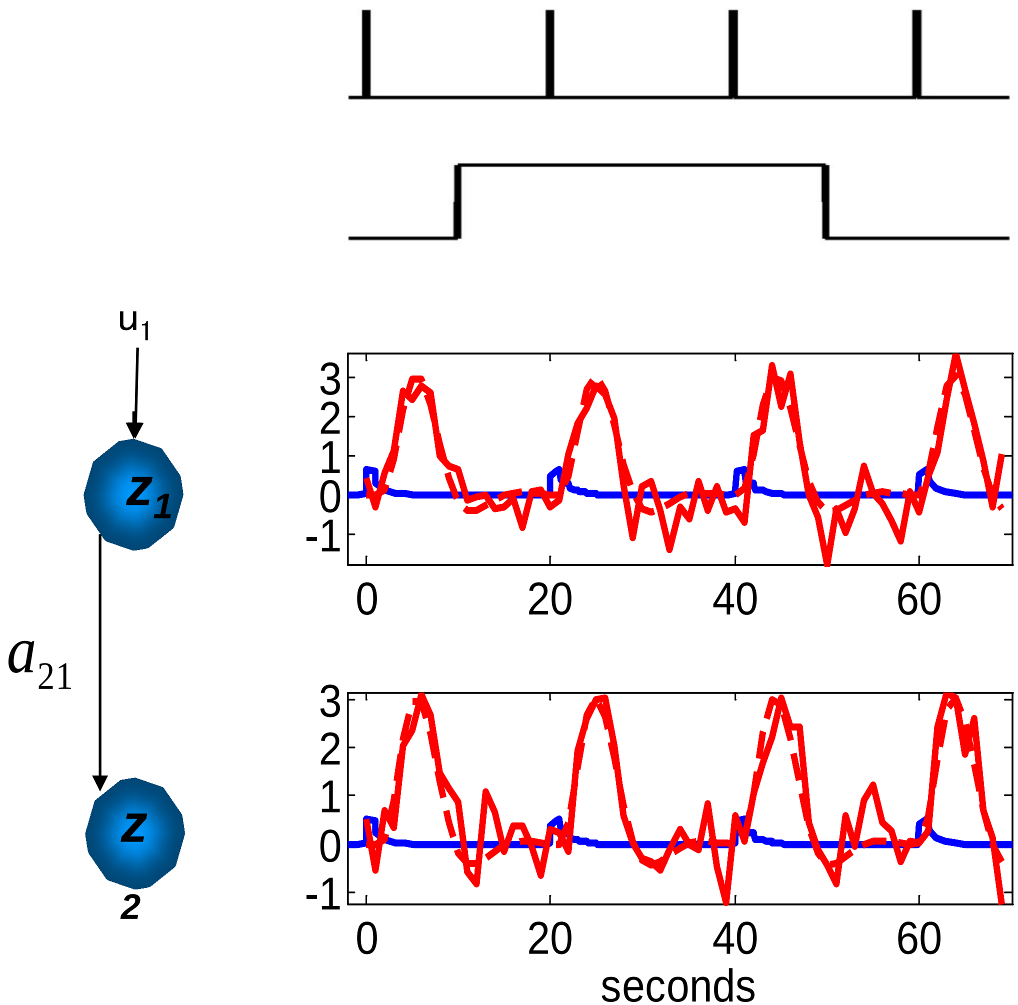
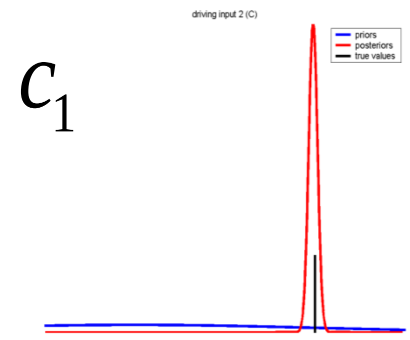
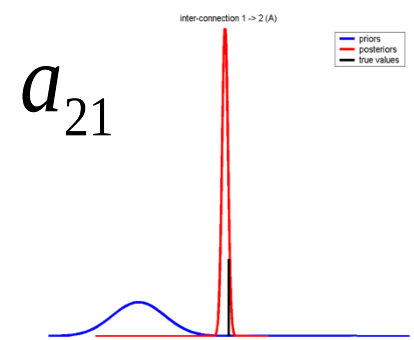


Input coupling, c_1



Forward coupling, a_{21}



Prior density — Posterior density — true values —

Inference about DCM parameters:

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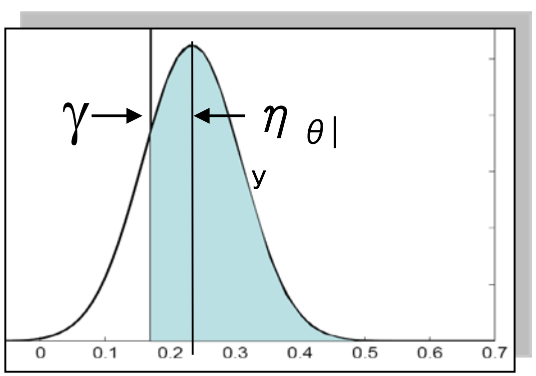
Bayesian single subject analysis

- The model parameters are distributions that have a mean $\eta_{\theta|y}$ and covariance $C_{\theta|y}$
  - Use of the cumulative normal distribution to test the probability that a certain parameter is above a chosen threshold γ:



Classical frequentist test across Ss

- Test summary statistic: mean $\eta_{\theta|y}$
  - One-sample t-test: Parameter>0?
  - Paired t-test: parameter 1 > parameter 2?
  - rmANOVA: e.g. in case of multiple sessions per subject

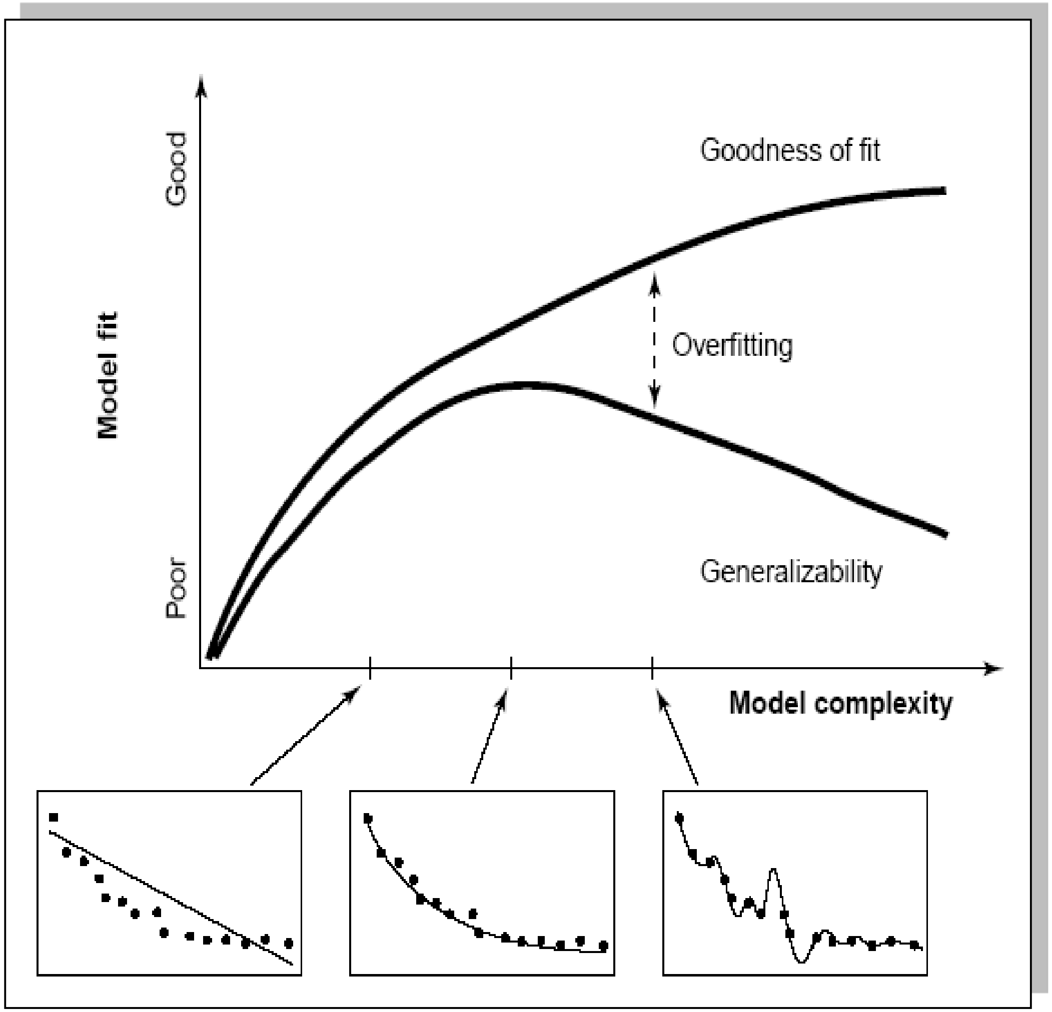
Model comparison and selection

Given competing hypotheses, which model is the best?

$$\log p(y \mid m) = accuracy(m) - complexity(m)$$

$$B_{ij} = \frac{p(y \mid m = i)}{p(y \mid m = j)}$$

Bayes Factor



Pitt & Miyung (2002) *TICS*