Dynamic causal modelling for fMRI

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Statistical Parametric Mapping for fMRI 2012 course

Overview

Brain connectivity: types & definitions

Anatomical connectivity
Functional connectivity
Effective connectivity

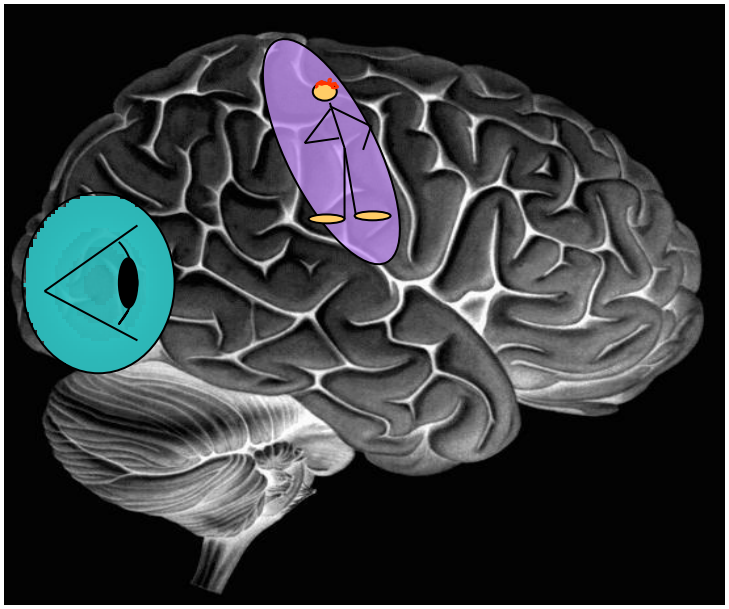
Dynamic causal models (DCMs)

Neuronal model
Hemodynamic model
Estimation: Bayesian framework

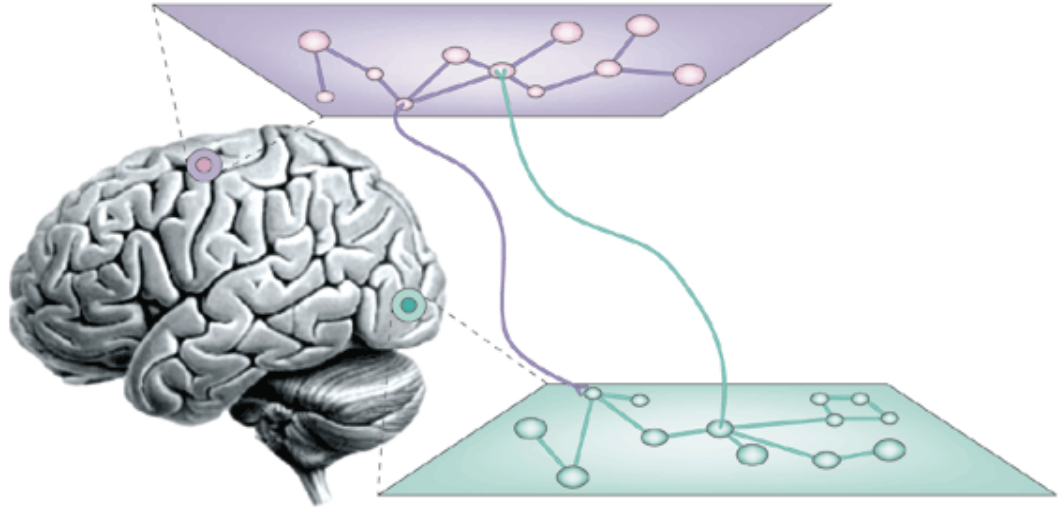
Applications & extensions of DCM to fMRI data

Principles of Organisation

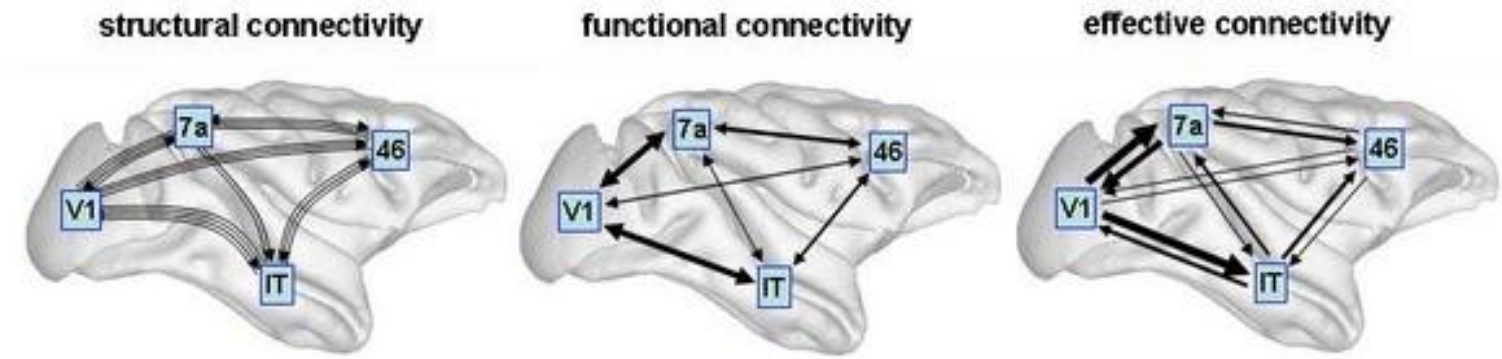
Functional specialization



Functional integration



Structural, functional & effective connectivity



Sporns 2007, *Scholarpedia*

- **anatomical/structural connectivity**
  = presence of axonal connections

- **functional connectivity**
  = statistical dependencies between regional time series

- **effective connectivity**
  = causal (directed) influences between neurons or neuronal populations

MECHANISM-FREE

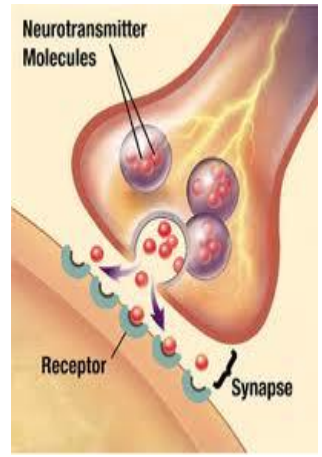
MECHANISTIC MODEL

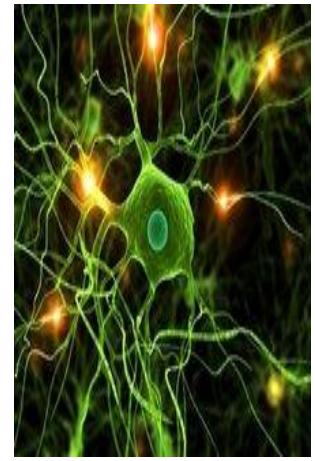
Anatomical connectivity

Definition:

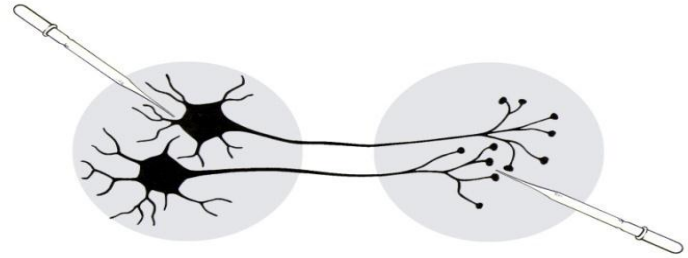
presence of axonal connections

- neuronal communication via synaptic contacts

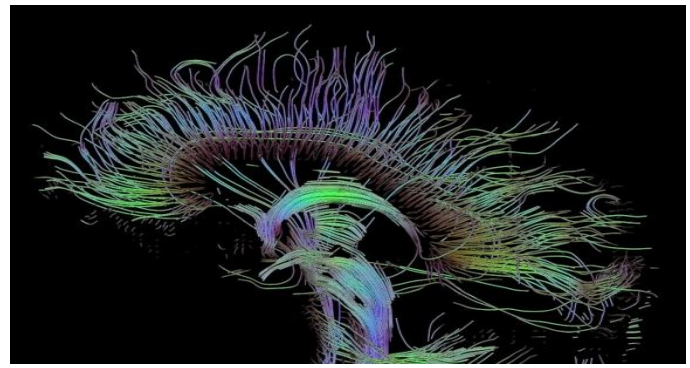




- Measured with
  - tracing techniques



  - diffusion tensor imaging (DTI)

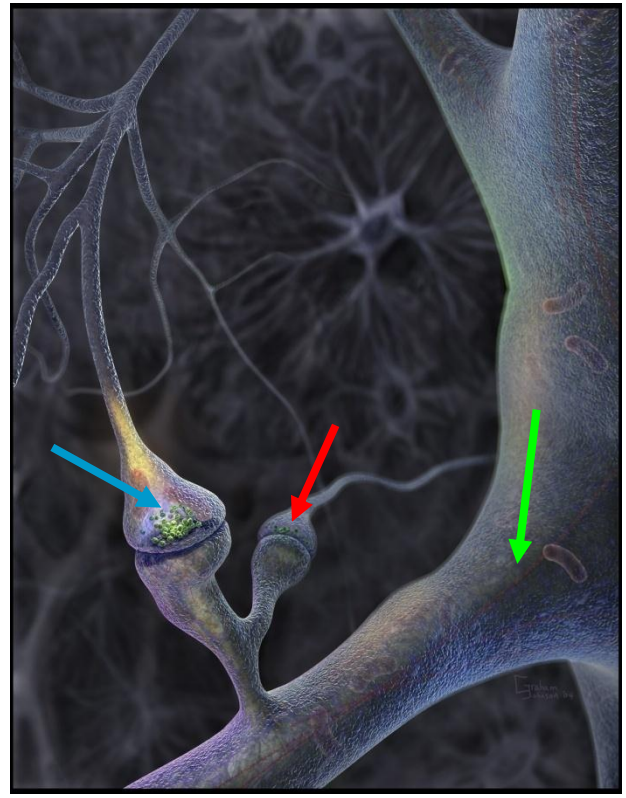


Knowing anatomical connectivity is not enough...

- Context-dependent recruiting of connections :
  - Local functions depend on network activity

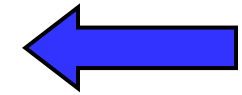
- Connections show synaptic plasticity
  - change in the structure and transmission properties of a synapse
  - even at short timescales

→ Look at functional and effective connectivity



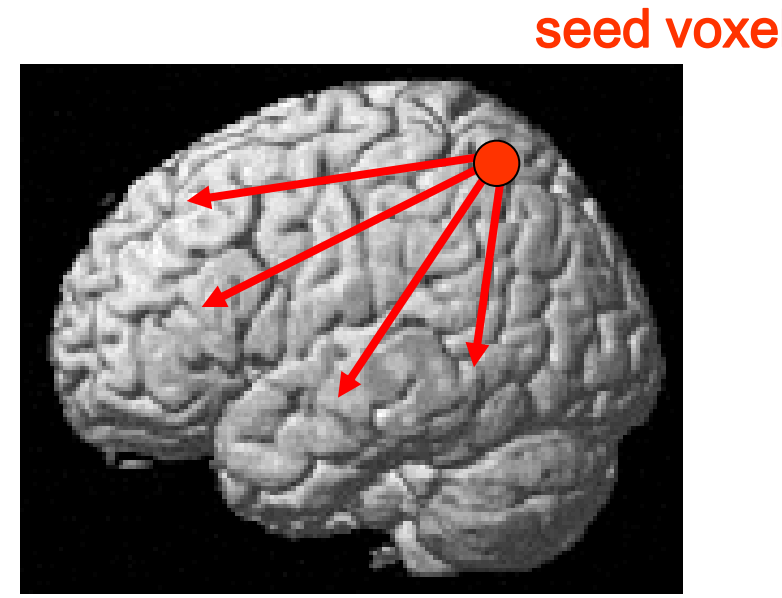
Functional connectivity

Definition: statistical dependencies between regional time series

- Seed voxel correlation analysis 
- Coherence analysis
- Eigen-decomposition (PCA, SVD)
- Independent component analysis (ICA)
- any technique describing statistical dependencies amongst regional time series

Seed-voxel correlation analyses

- hypothesis-driven choice of a seed voxel
- extract reference time series
- voxel-wise correlation with time series from all other voxels in the brain

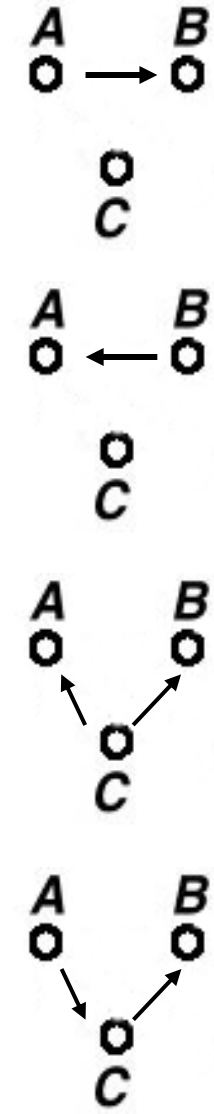


Pros & Cons of functional connectivity analysis

- Pros:
  - useful when we have no experimental control over the system of interest and no model of what caused the data (e.g. sleep, hallucinations, etc.)

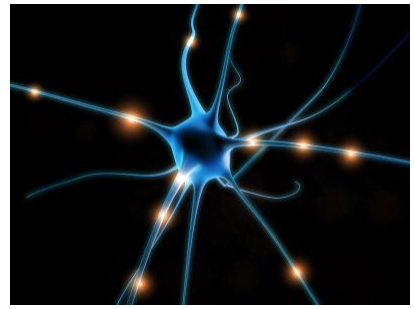
- Cons:
  - interpretation of resulting patterns is difficult / arbitrary
  - no mechanistic insight
  - usually suboptimal for situations where we have a priori knowledge / experimental control

→ Effective connectivity



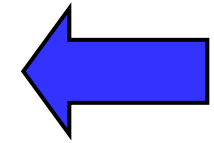
Effective connectivity

Definition: ***causal*** *(directed) influences between neurons or neuronal populations*

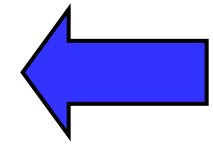
- *In vivo* and *in vitro* stimulation and recording
- 
- Models of causal interactions among neuronal populations
  - explain *regional* effects in terms of *interregional* connectivity

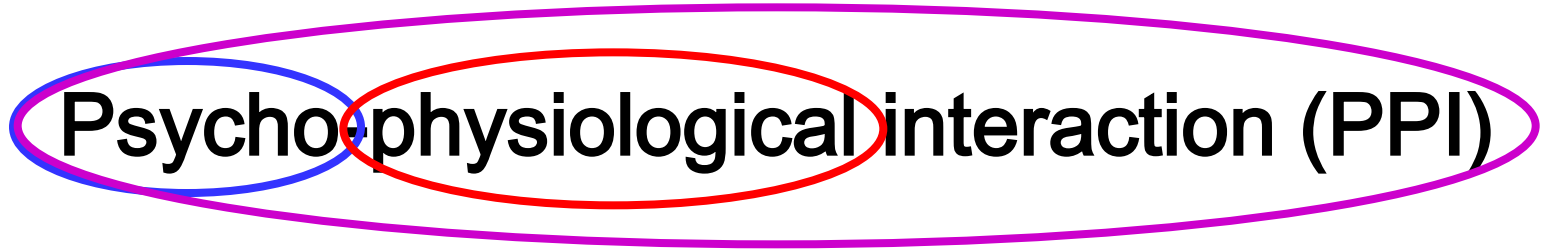
Some models for computing effective connectivity from fMRI data

- Structural Equation Modelling (SEM)
  McIntosh et al. 1991, 1994; Büchel & Friston 1997; Bullmore et al. 2000
- regression models
  (e.g. psycho-physiological interactions, PPIs)
  Friston et al. 1997



- Volterra kernels
  Friston & Büchel 2000
- Time series models (e.g. MAR, Granger causality)
  Harrison et al. 2003, Goebel et al. 2003
- Dynamic Causal Modelling (DCM)
  bilinear: Friston et al. 2003; *nonlinear:* Stephan et al. 2008



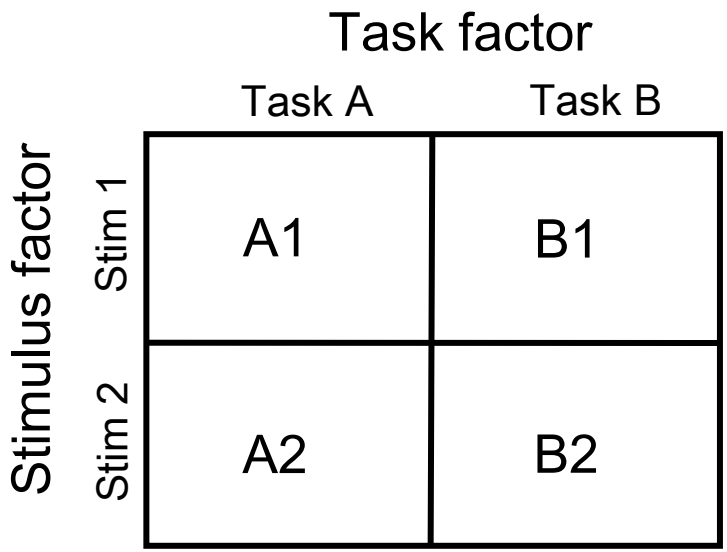


- bilinear model of how the psychological context A changes the influence of area B on area C :

$$B \times A \rightarrow C$$

- A PPI corresponds to differences in regression slopes for different contexts.

Psycho-physiological interaction (PPI)



GLM of a 2x2 factorial design:

$$y = (T_A - T_B)\,\beta_1 + (S_1 - S_2)\beta_2 + (T_A - T_B)(S_1 - S_2)\beta_3 + e$$

main effect of task

main effect of stim. type

interaction

We can replace one main effect in the GLM by the time series of an area that shows this main effect.

$$y = (T_A - T_B)\,\beta_1 + V1\beta_2 + (T_A - T_B)V1\beta_3 + e$$

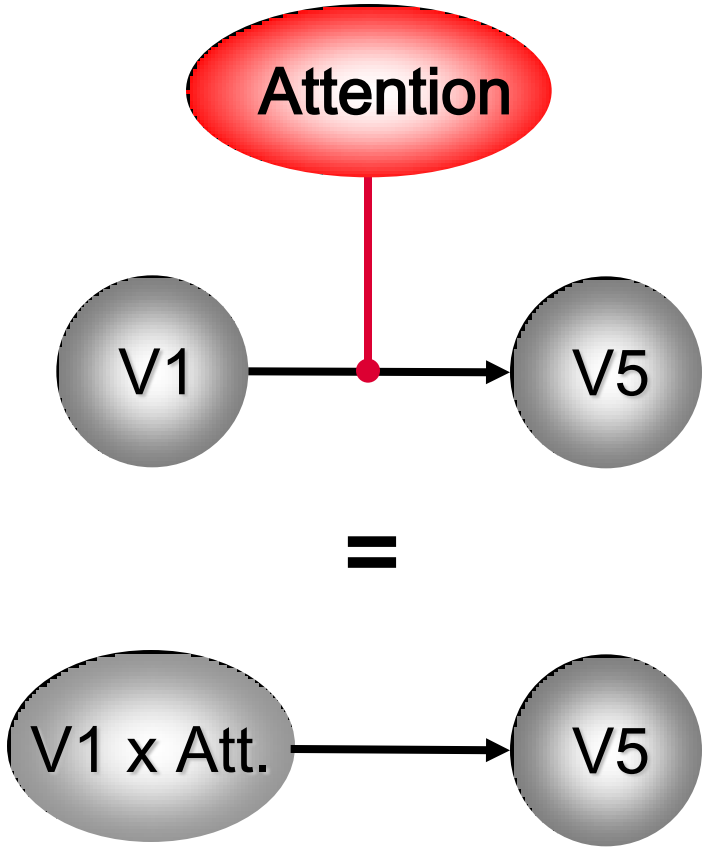
main effect of task

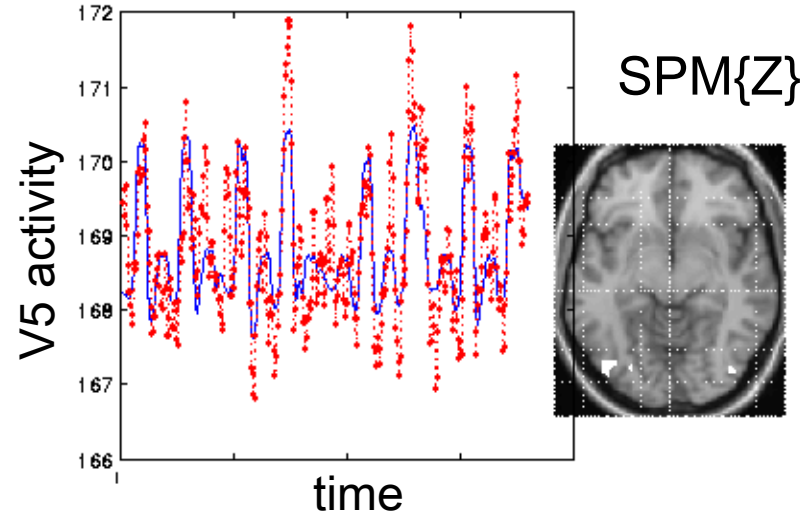
V1 time series ≈ main effect of stim. type

psycho-physiological interaction

Friston et al. 1997, *NeuroImage*

Example PPI: Attentional modulation of V1→V5CC



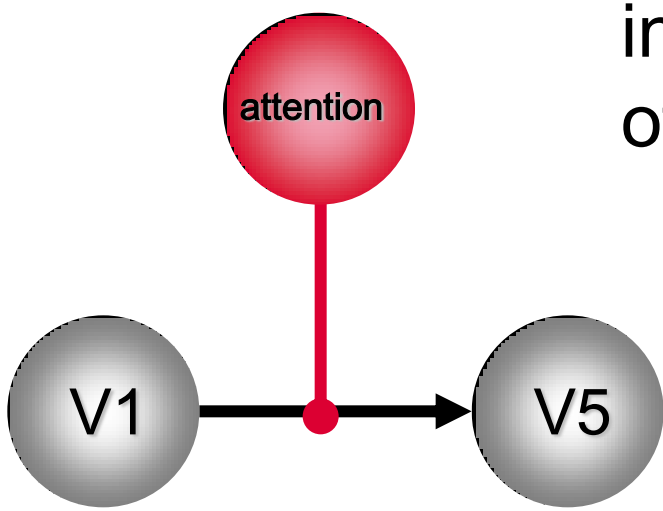


Friston et al. 1997, *NeuroImage*
Büchel & Friston 1997, *Cereb. Cortex*

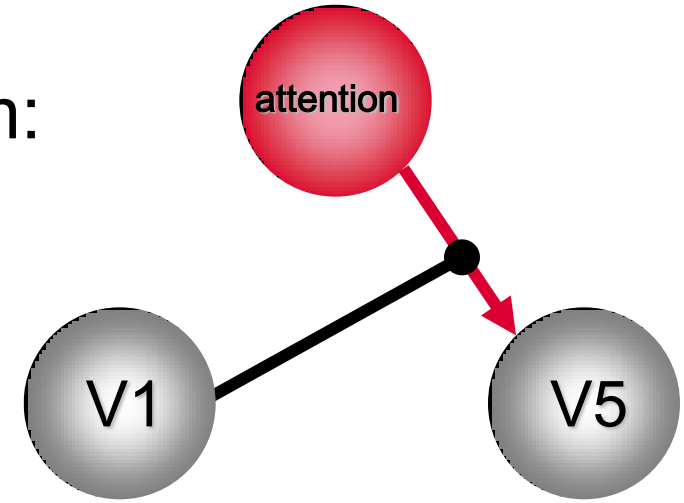
PPI: Interpretation

$$y = (T_A - T_B)\,\beta_1 + V1\beta_2 + (T_A - T_B)\,V1\beta_3 + e$$

Two possible interpretations of the PPI term:



Modulation of V1→V5 by attention

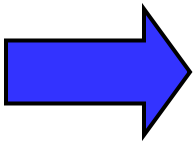


Modulation of the impact of attention on V5 by V1

Pros & Cons of PPIs

- Pros:
  - given a single source region, we can test for its context-dependent connectivity across the entire brain
  - easy to implement
- Cons:
  - only allows to model contributions from a single area
  - operates at the level of BOLD time series (SPM 99/2).

    SPM 5/8 deconvolves the BOLD signal to form the proper interaction term, and then reconvolves it.
  - ignores time-series properties of the data



Dynamic Causal Models

needed for more robust statements of effective connectivity.

Overview

Brain connectivity: types & definitions

Anatomical connectivity
Functional connectivity
Effective connectivity

Dynamic causal models (DCMs)

Basic idea
Neuronal model
Hemodynamic model
Parameter estimation, priors & inference

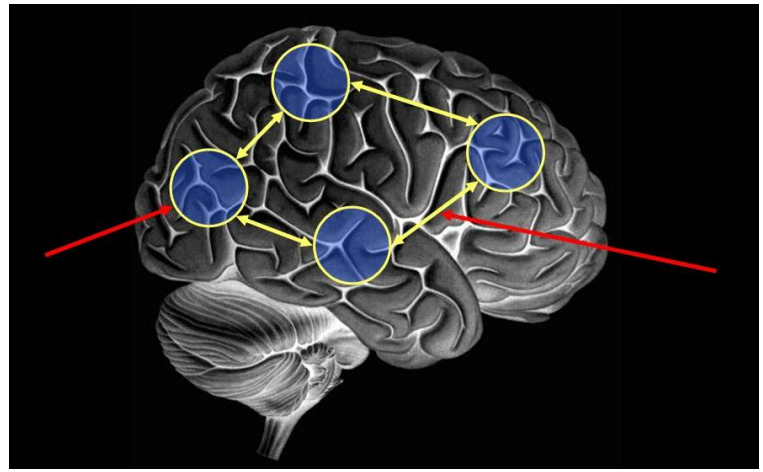
Applications & extensions of DCM to fMRI data

Basics of Dynamic Causal Modelling

DCM allows us to look at how areas within a network interact:

Investigate functional integration & modulation of specific cortical pathways

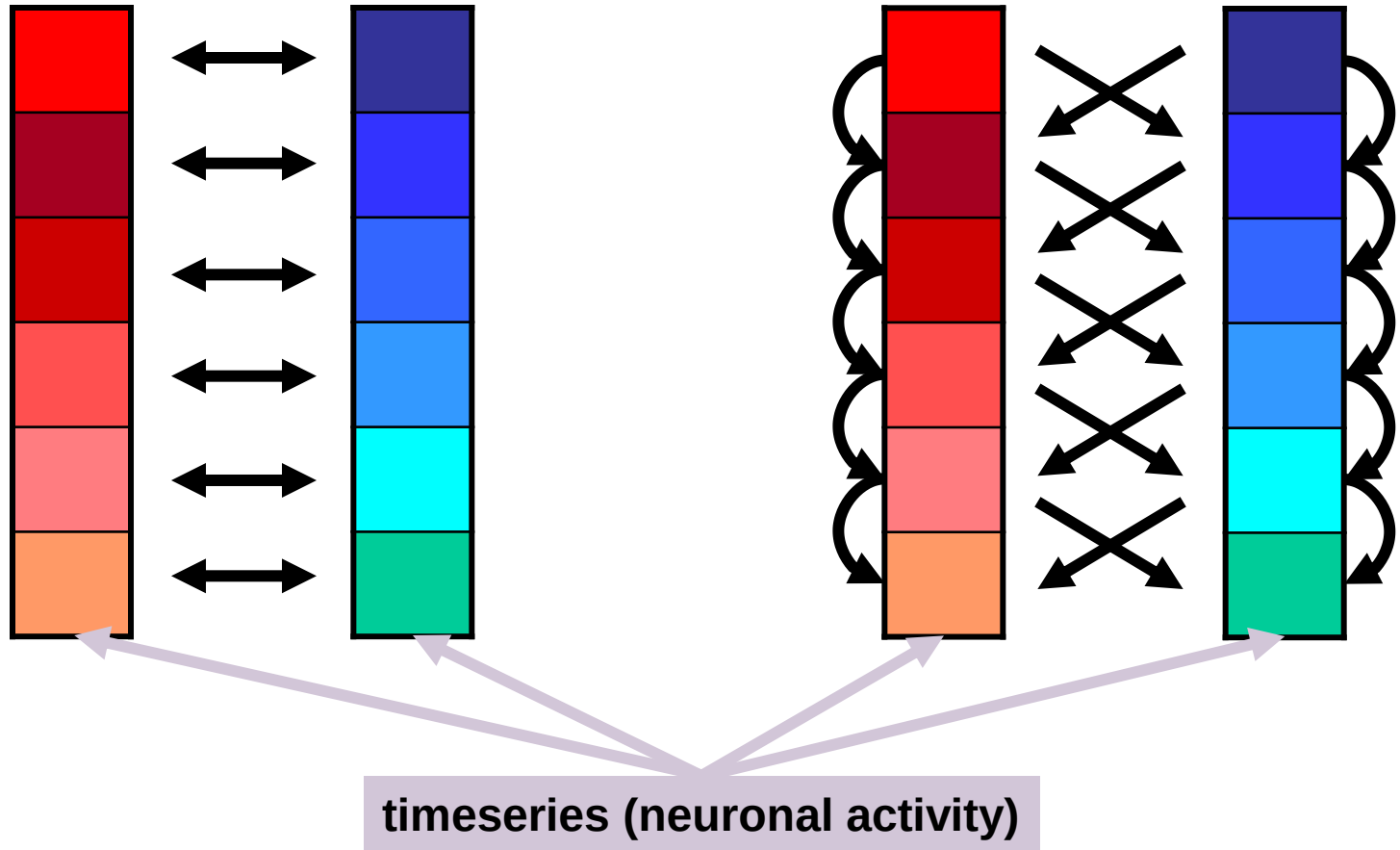
- Temporal dependency of activity within and between areas (causality)



Temporal dependence and causal relations

Seed voxel approach, PPI etc.

Dynamic *Causal* Models

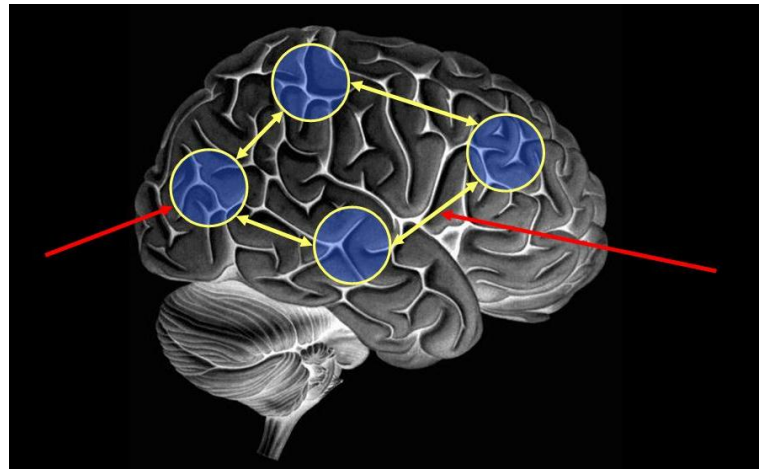


Basics of Dynamic Causal Modelling

DCM allows us to look at how areas within a network interact:

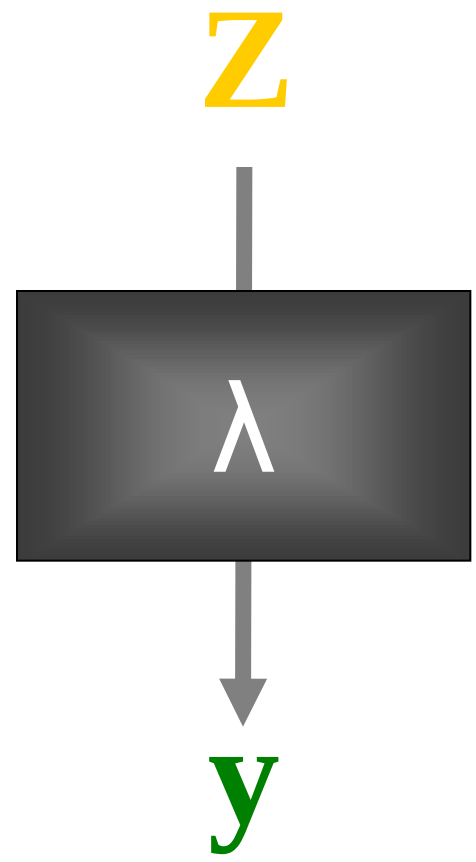
Investigate functional integration & modulation of specific cortical pathways

- Temporal dependency of activity within and between areas (causality)
- Separate neuronal activity from observed BOLD responses



Basics of DCM: Neuronal and BOLD level

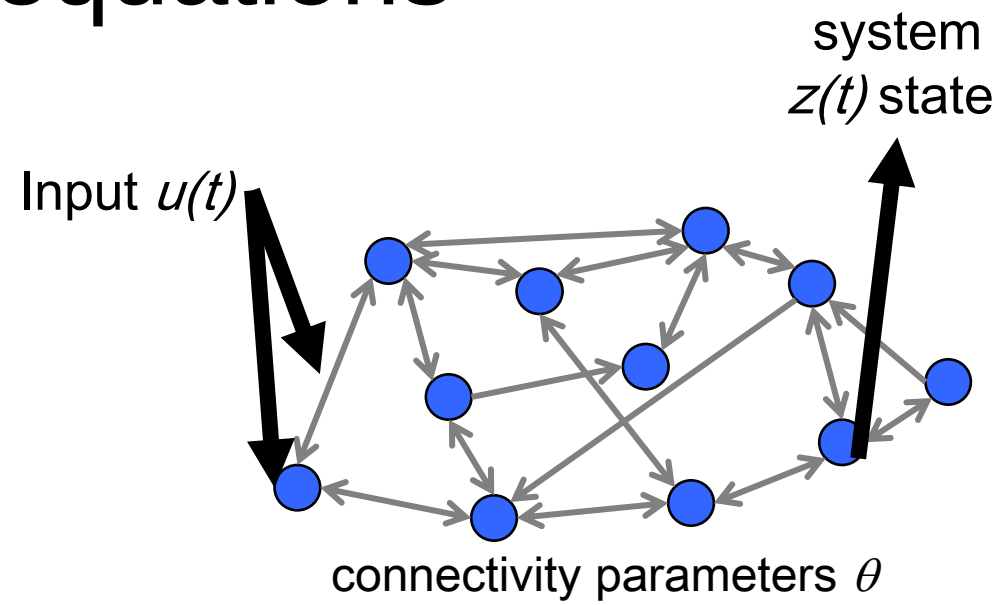
- Cognitive system is modelled at its underlying neuronal level (not directly accessible for fMRI).
- The modelled neuronal dynamics (**Z**) are transformed into area-specific BOLD signals (**y**) by a hemodynamic model (**λ**).



The aim of DCM is to estimate parameters at the neuronal level such that the modelled and measured BOLD signals are optimally similar.

Neuronal systems are represented by differential equations

A System is a set of elements $z_n(t)$ which interact in a spatially and temporally specific fashion



State changes of the system states are dependent on:

- the current state z
- external inputs u
- its connectivity θ
- time constants & delays

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

DCM parameters = rate constants

Generic solution to the ODEs in DCM:

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

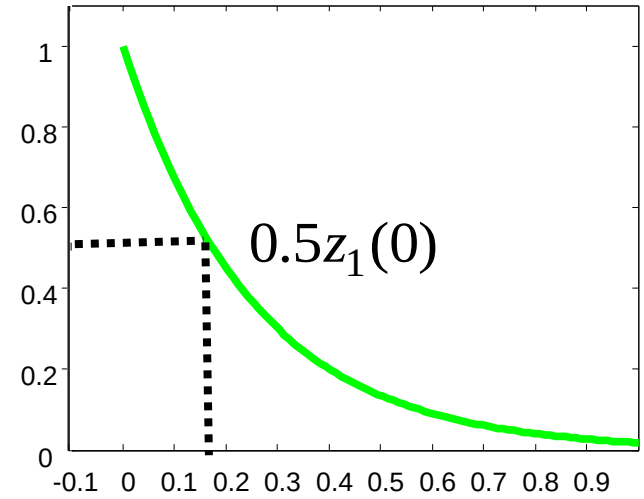
Half-life τ

$$z_1(\tau) = 0.5z_1(0)$$

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

$$\Rightarrow \boxed{s = \ln 2 / \tau}$$

Decay function



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

DCM parameters = rate constants

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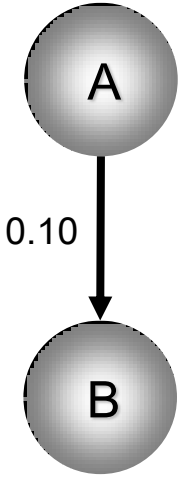
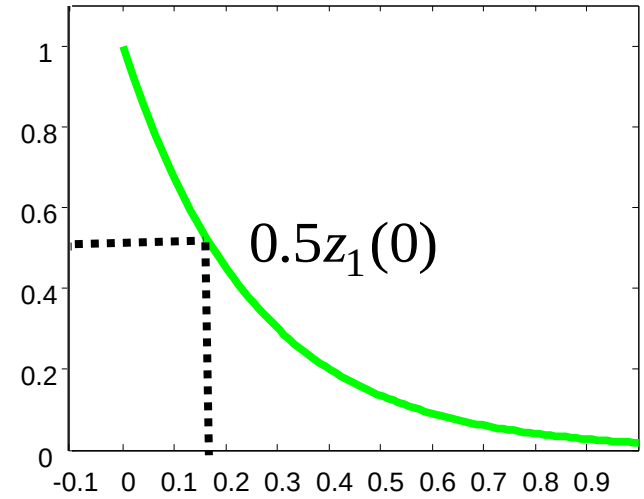


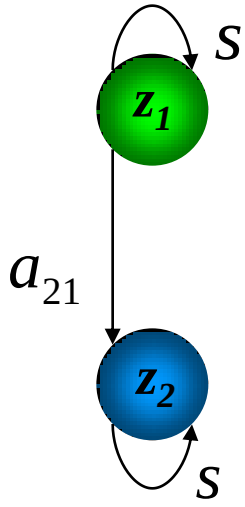
If A→B is 0.10 s^{-1} this means that, per unit time, the increase in activity in B corresponds to 10% of the activity in A

Decay function



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

Linear dynamics: 2 nodes

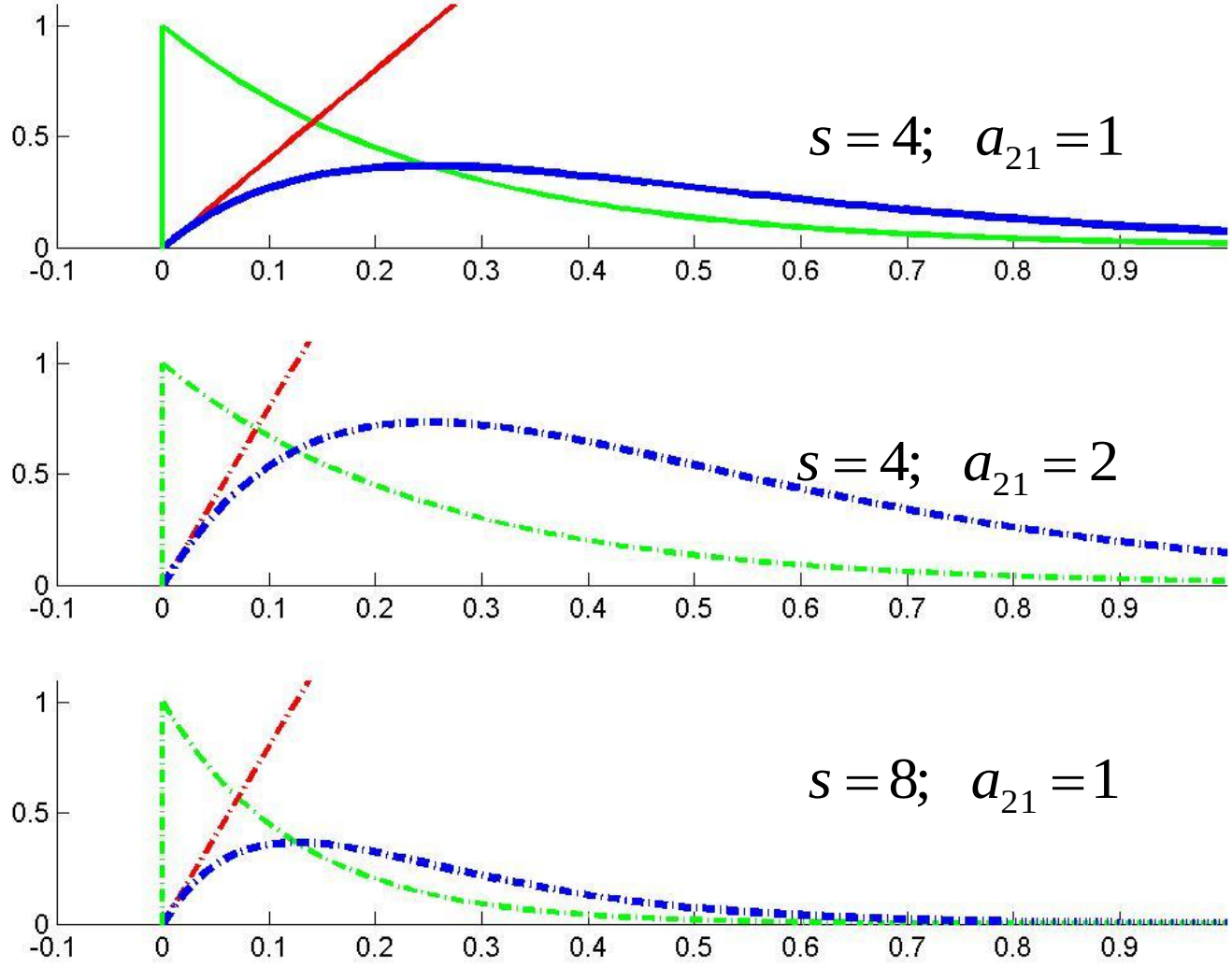


$$\dot{z}_1 = -sz_1$$
$$\dot{z}_2 = s\left(a_{21}z_1 - z_2\right)$$

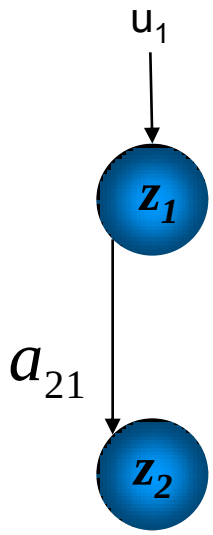
$$z_1(0) = 1$$
$$z_2(0) = 0$$

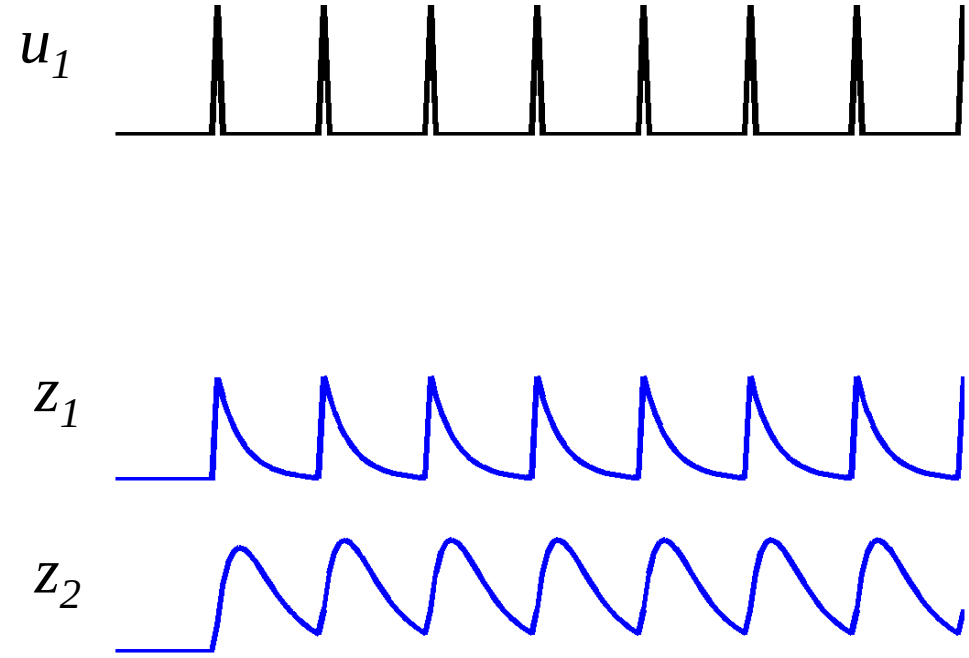
$$z_1(t) = \exp(-st)$$
$$z_2(t) = sa_{21}t\exp(-st)$$

$$a_{21} > 0$$



Neurodynamics: 2 nodes with input

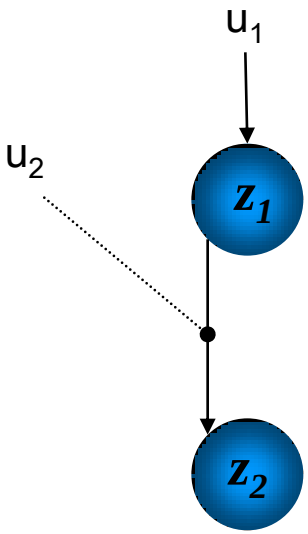


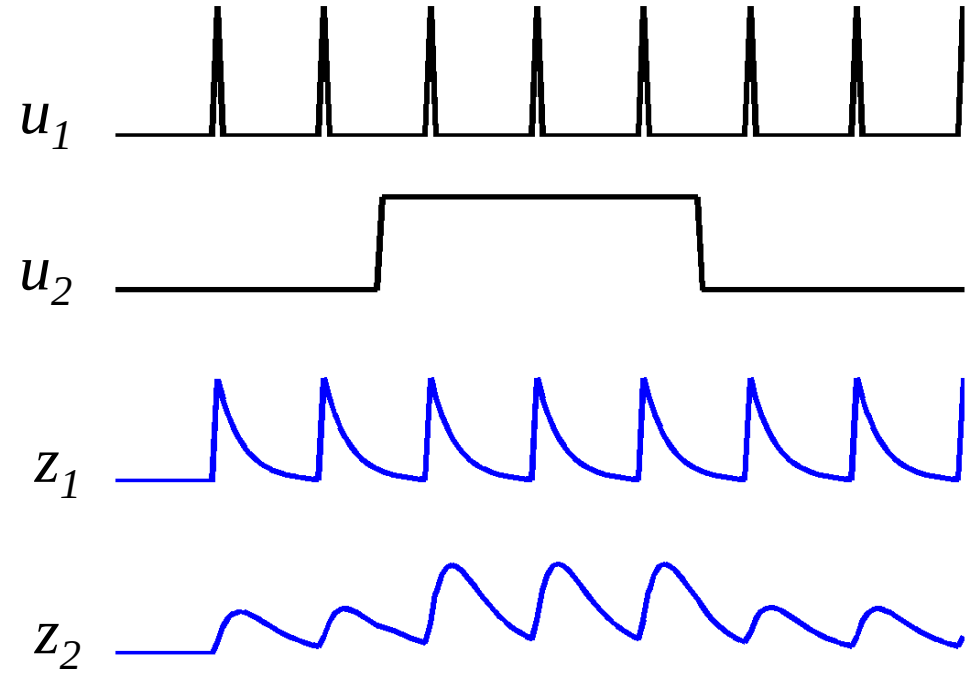


$$\begin{bmatrix} \dot{z}_1 \\ \dot{z}_2 \end{bmatrix} = s\begin{bmatrix} -1 & 0 \\ a_{21} & -1 \end{bmatrix}\begin{bmatrix} z_1 \\ z_2 \end{bmatrix} + \begin{bmatrix} c \\ 0 \end{bmatrix} u_1 \qquad a_{21} > 0$$

activity in z_2 is coupled to z_1 via coefficient a_{21}

Neurodynamics: positive modulation





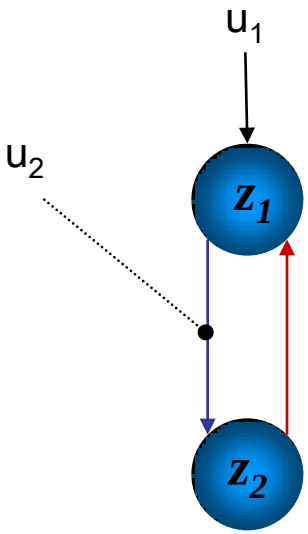
$$\begin{bmatrix} \dot{z}_1 \\ \dot{z}_2 \end{bmatrix} = s\begin{bmatrix} -1 & 0 \\ a_{21} & -1 \end{bmatrix}\begin{bmatrix} z_1 \\ z_2 \end{bmatrix} + u_2\begin{bmatrix} 0 & 0 \\ b_{21}^2 & 0 \end{bmatrix}\begin{bmatrix} z_1 \\ z_2 \end{bmatrix} + \begin{bmatrix} c \\ 0 \end{bmatrix}u_1$$

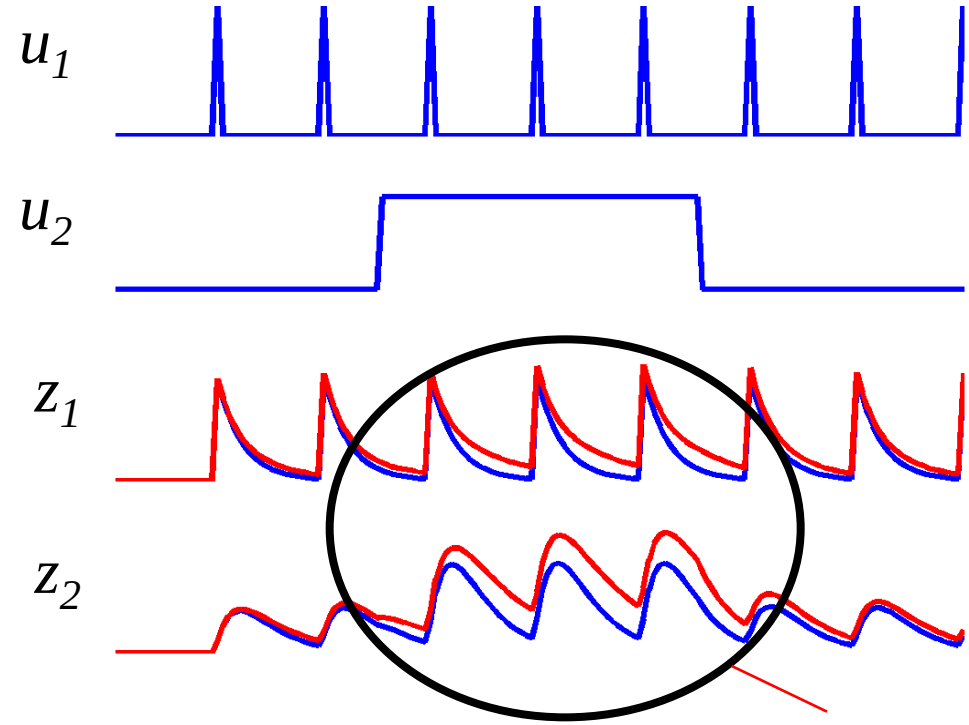
index, not squared

$b_{21}^2 > 0$

modulatory input u_2 activity through the coupling a_{21}

Neurodynamics: reciprocal connections



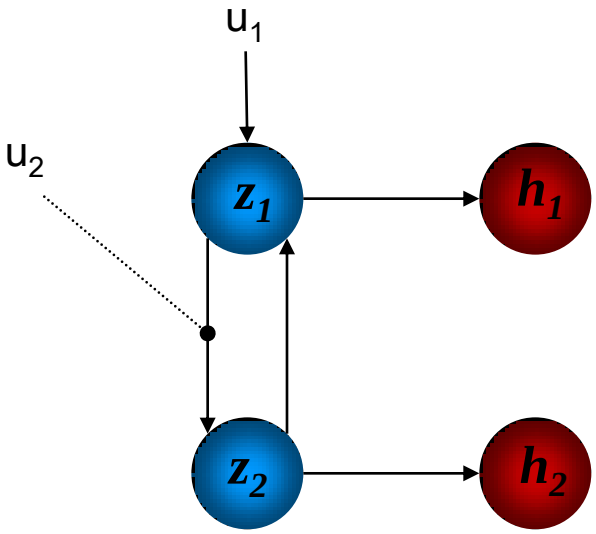


reciprocal connection disclosed by u_2

$$\begin{bmatrix}\dot{z}_1\\ \dot{z}_2\end{bmatrix} = s\begin{bmatrix}-1 & a_{12}\\ a_{21} & -1\end{bmatrix}\begin{bmatrix}z_1\\ z_2\end{bmatrix} + u_2\begin{bmatrix}0 & 0\\ b_{21}^2 & 0\end{bmatrix}\begin{bmatrix}z_1\\ z_2\end{bmatrix} + \begin{bmatrix}c\\ 0\end{bmatrix}u_1$$

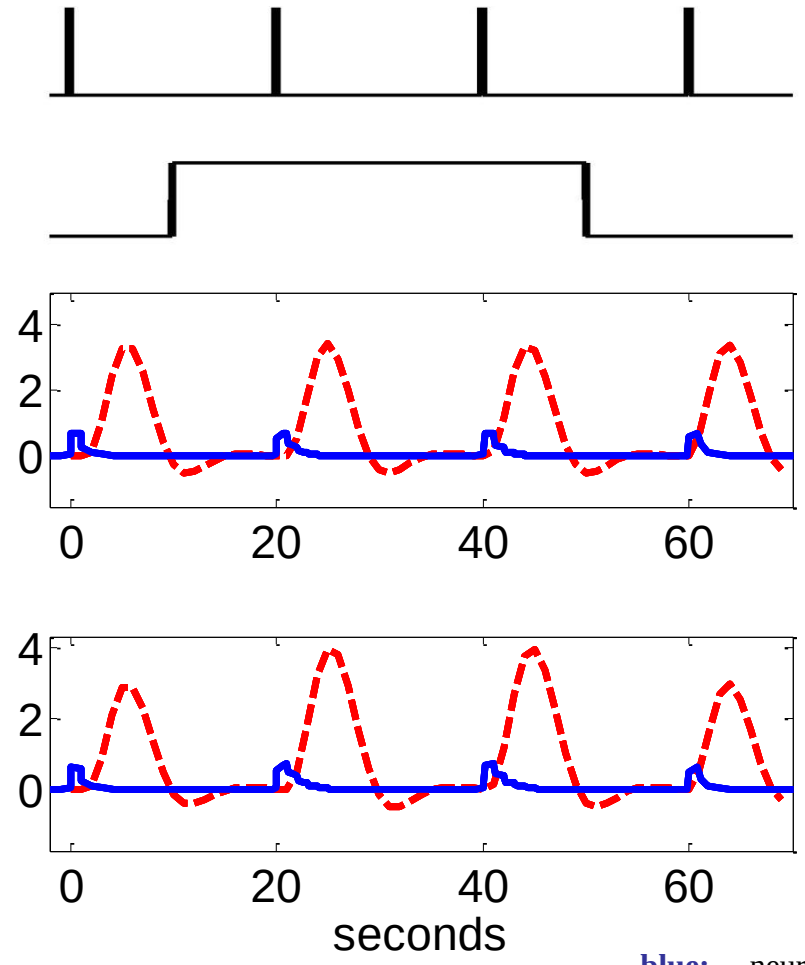
$$a_{12}, a_{21}, b_{21}^2 > 0$$

Haemodynamics: reciprocal connections



BOLD
(without noise)

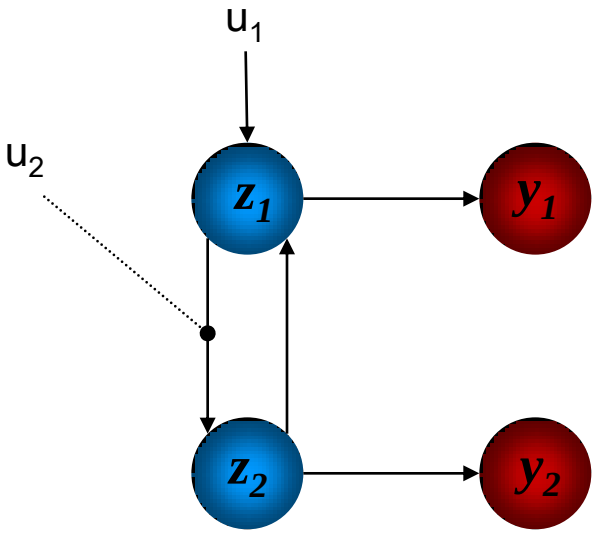
BOLD
(without noise)



blue: neuronal activity
red: bold response

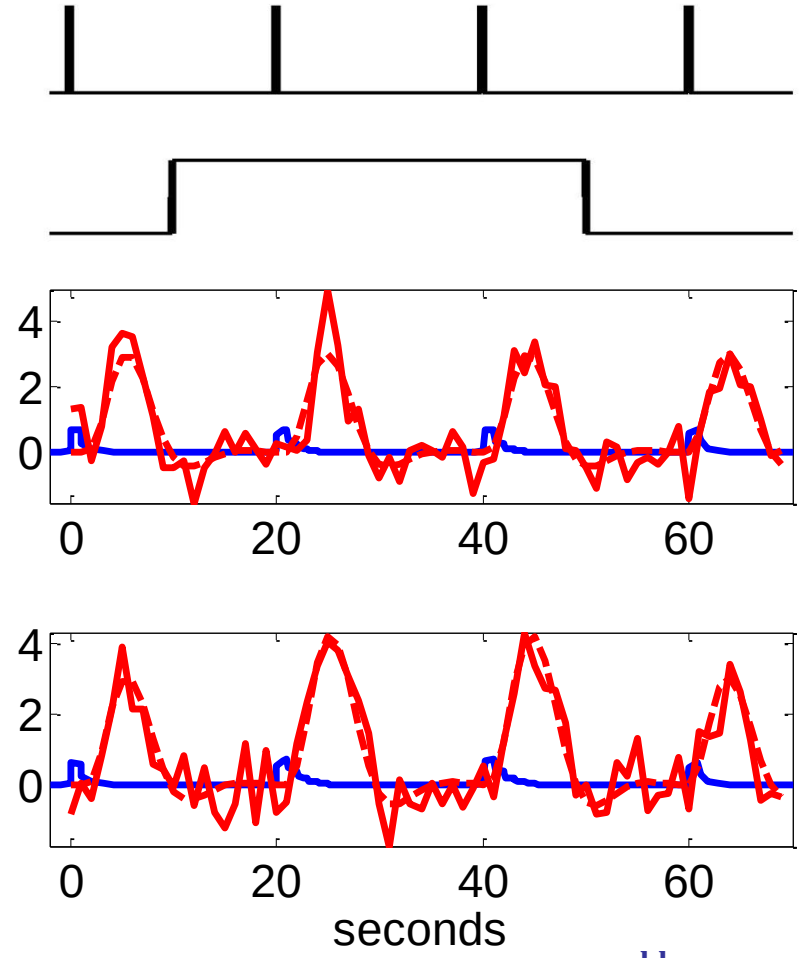
h(u,θ) represents the BOLD response (balloon model) to input

Haemodynamics: reciprocal connections



BOLD with **Noise added**

BOLD with **Noise added**



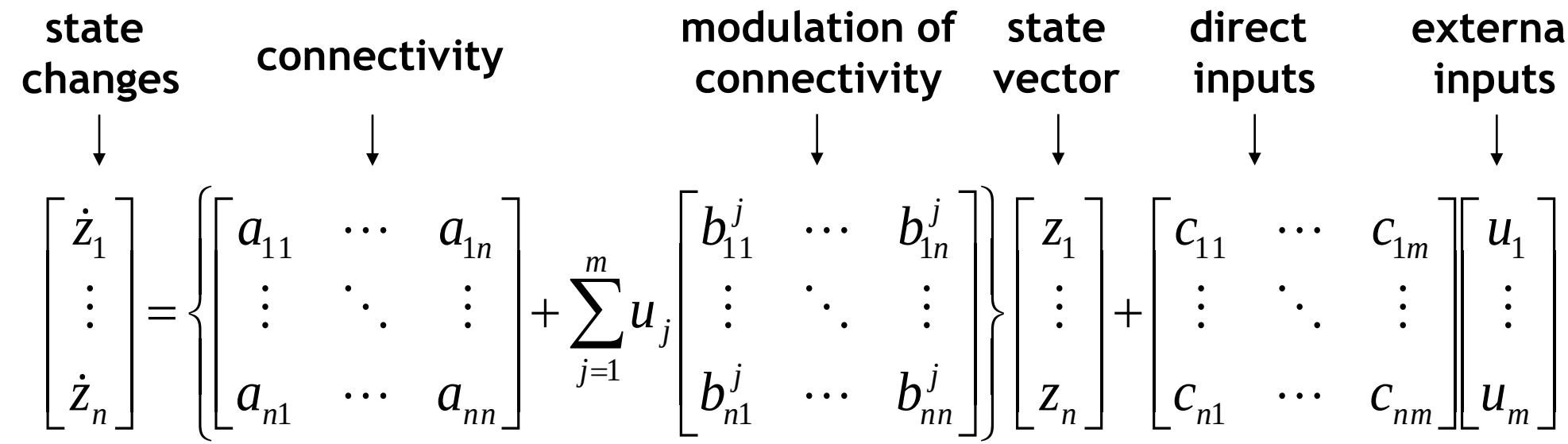
blue: neuronal activity

red: bold response

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

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

Bilinear state equation in DCM for fMRI



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

Conceptual overview

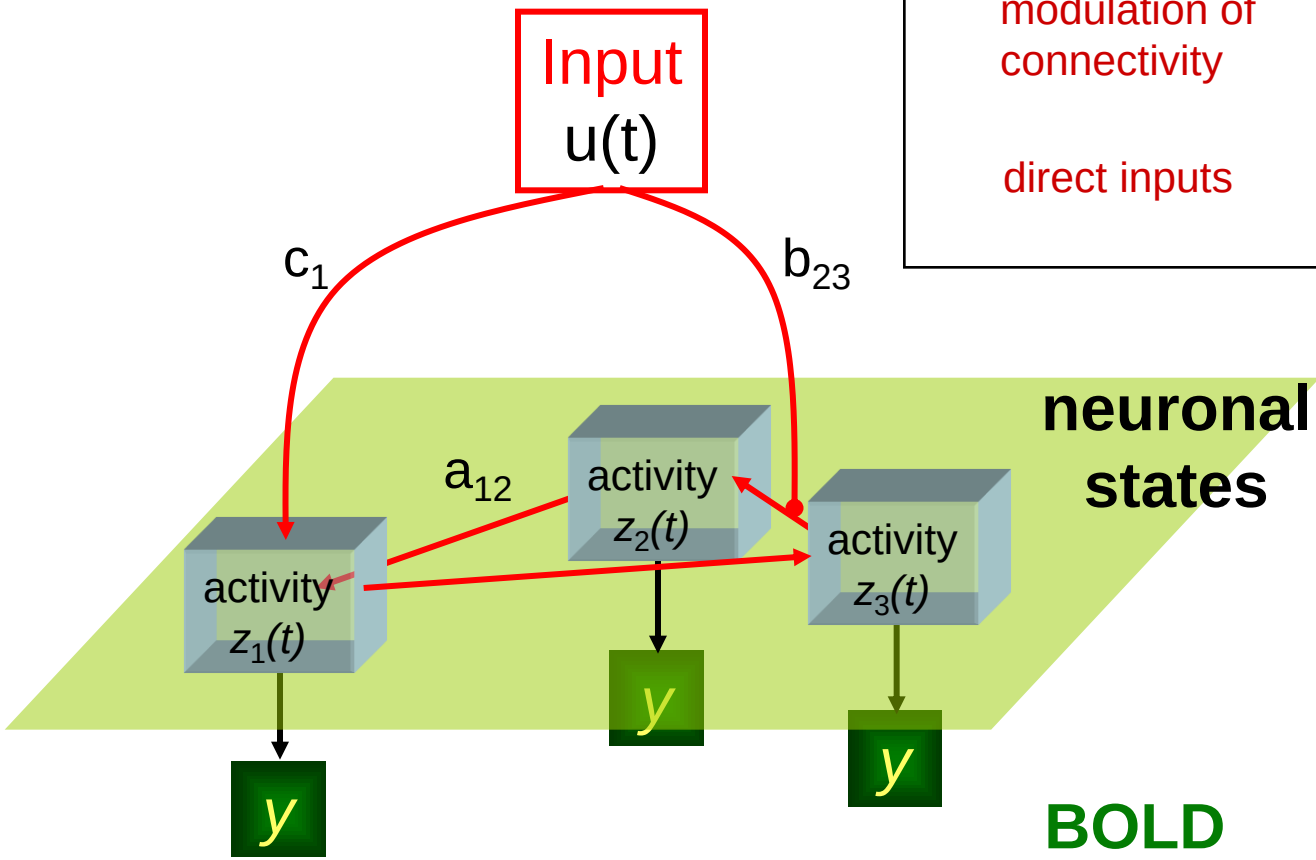
Neuronal state equation $\dot{z} = F(z, u, \theta^n)$

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



integration

z

λ

haemodynamic model

y

Friston et al. 2003, *NeuroImage*

The hemodynamic “Balloon” model

BOLD signal
$y(t) = \lambda(v, q)$

changes in volume
$\tau \dot{v} = f - v^{1/\alpha}$

changes in dHb
$\tau \dot{q} = f\, E(f, \rho)/\rho - v^{1/\alpha} q/v$

flow induction
$\dot{f} = s$

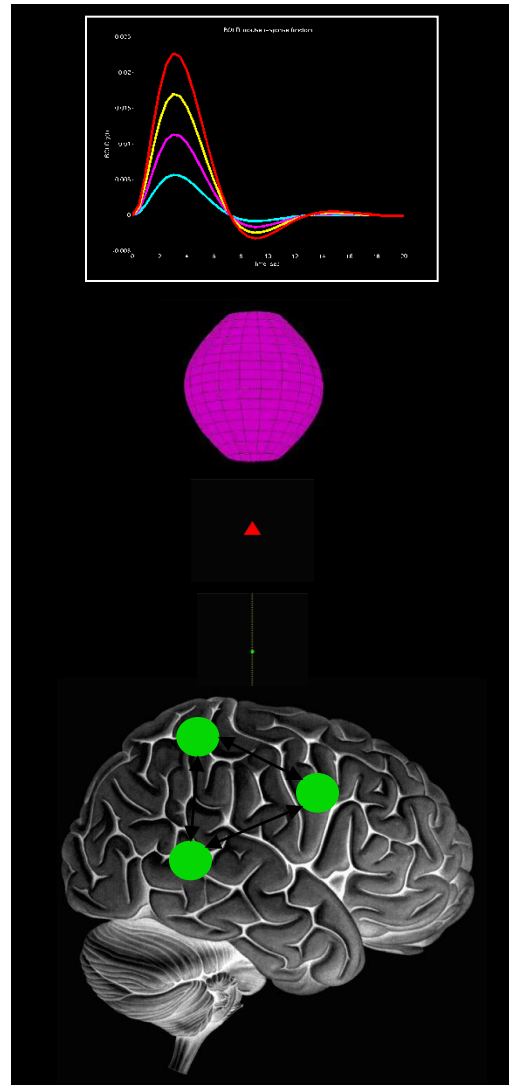
vasodilatory signal
$\dot{s} = z - \kappa s - \gamma(f - 1)$

neuronal input
$z(t)$

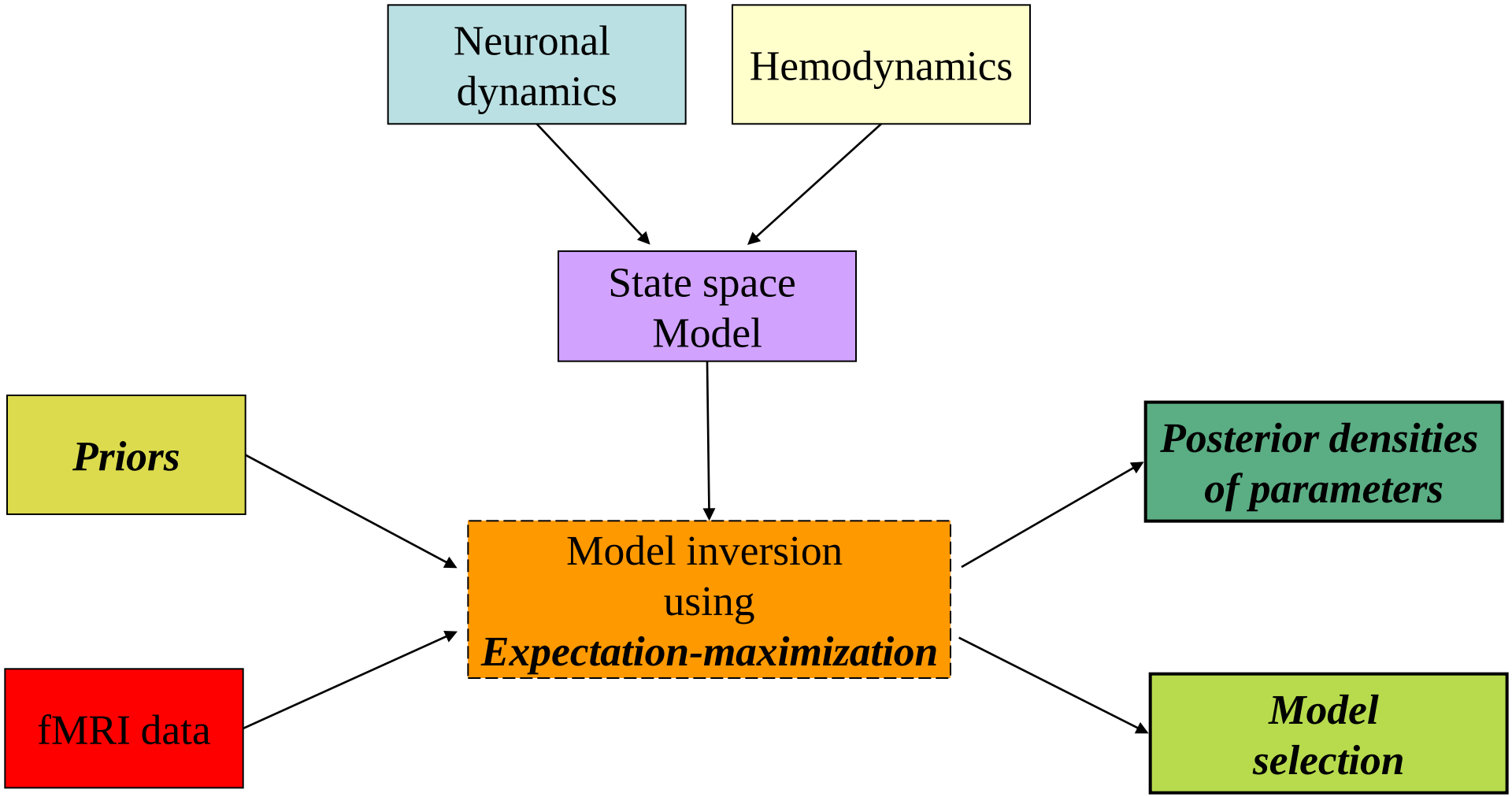
Region-specific HRFs!

5 haemodynamic parameters

Friston et al. 2000, *NeuroImage*
Stephan et al. 2007, *NeuroImage*



DCM roadmap


Neuronal dynamics
Hemodynamics
State space Model
Priors
fMRI data
Model inversion using Expectation-maximization
Posterior densities of parameters
Model selection

Estimation: Bayesian framework

Models of
•Haemodynamics in a single region
•Neuronal interactions

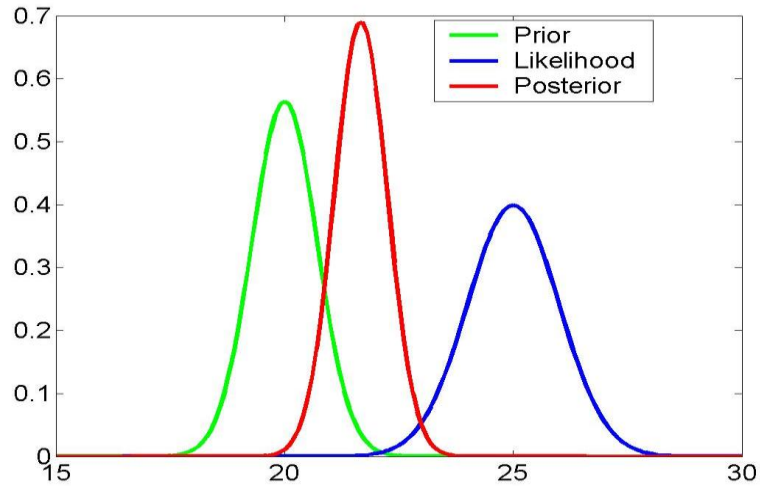
Constraints on
•Haemodynamic parameters
•Connections

likelihood term $p(y \mid \theta)$

$p(\theta)$ prior

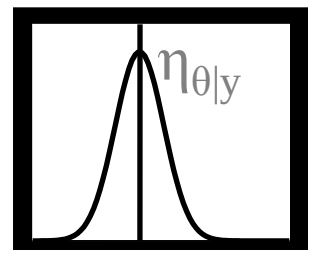
posterior $p(\theta \mid y) \propto p(y \mid \theta)\, p(\theta)$

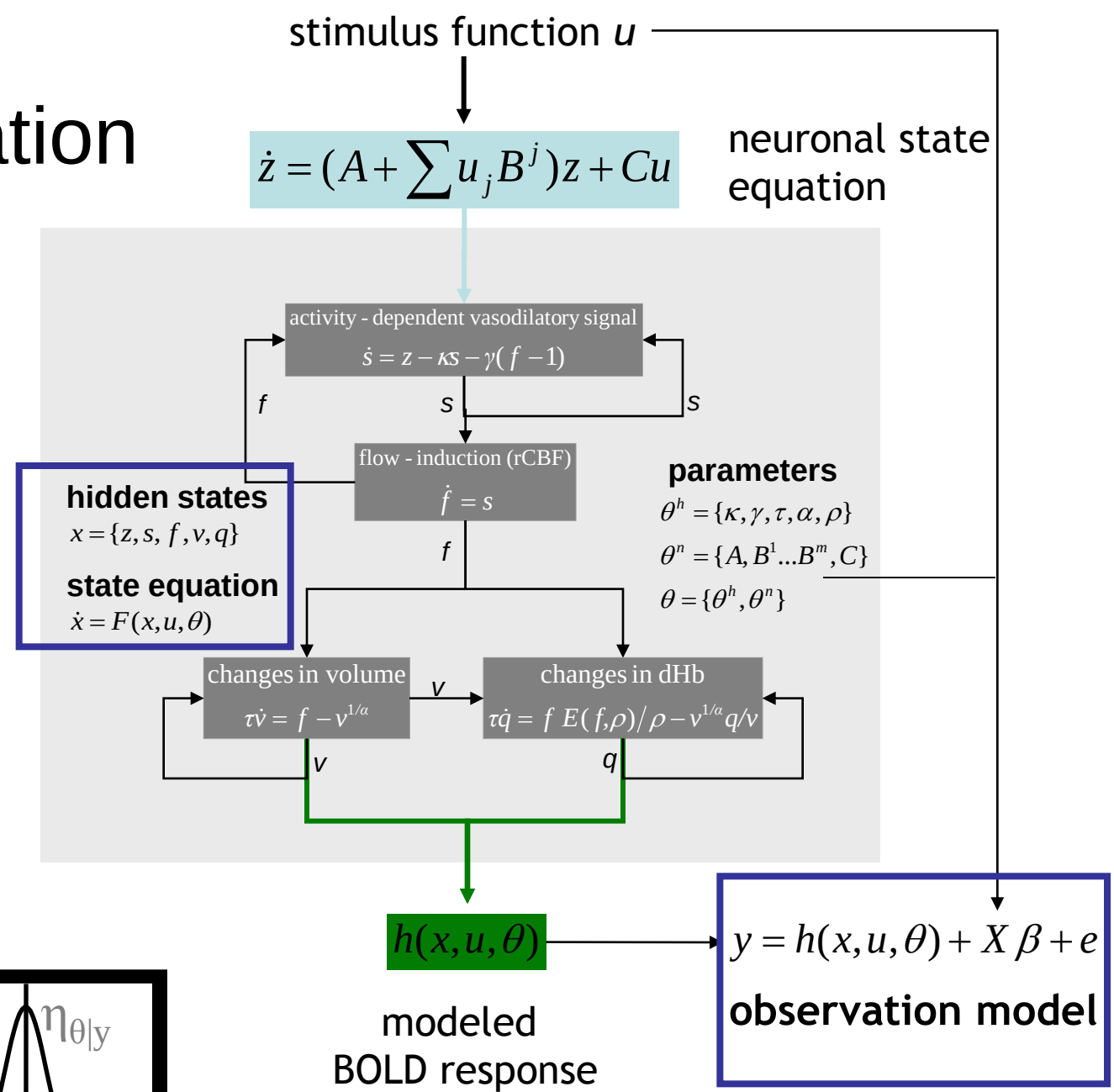
Bayesian estimation



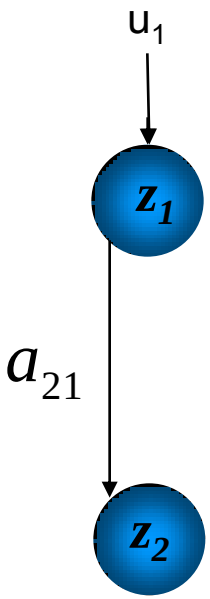
Overview: parameter estimation

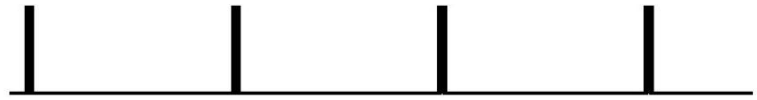
- Specify model (neuronal and haemodynamic level)
- Make it an observation model by adding measurement error *e* and confounds *X* (e.g. drift).
- Bayesian parameter estimation using expectation-maximization.
- Result:
  (Normal) posterior parameter distributions, given by mean $\eta_{\theta|y}$ and Covariance $C_{\theta|y}$.



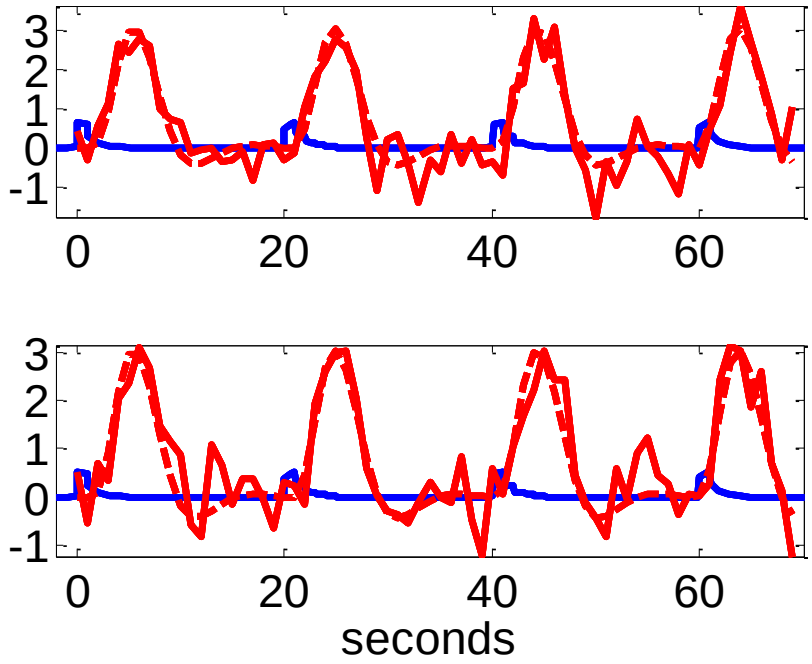


Parameter estimation: an example

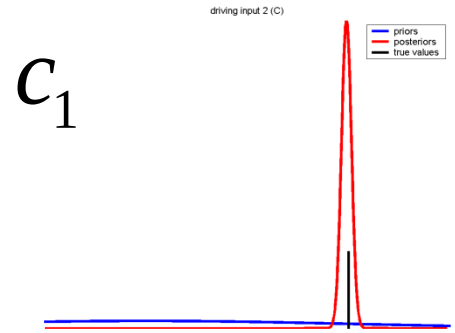




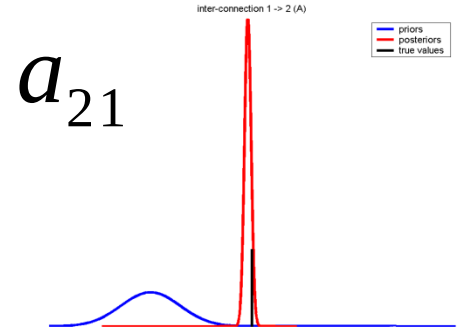
Simulated response



Input coupling, c_1



Forward coupling, a_{21}

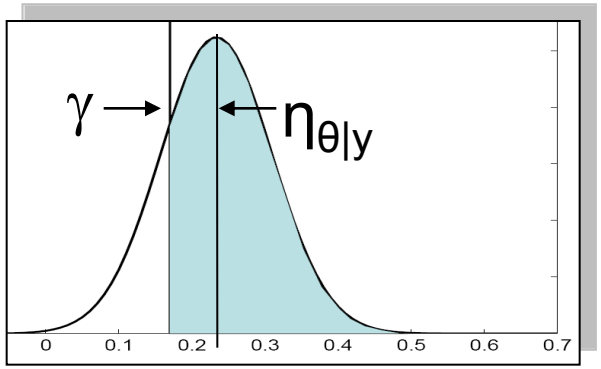


Prior density — Posterior density — true values

Inference about DCM parameters

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:

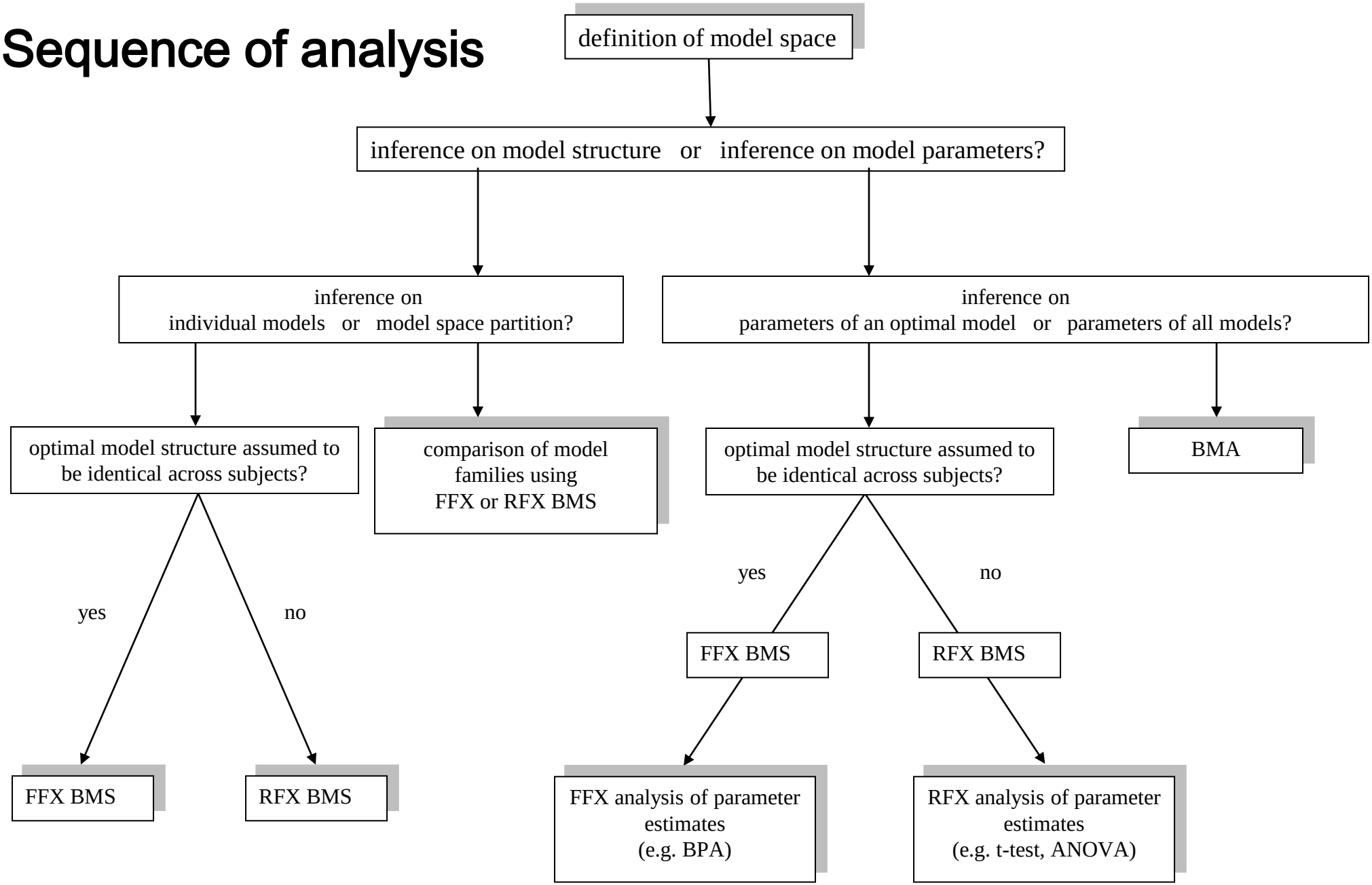


Classical frequentist test across groups

- 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

Bayesian parameter averaging

Sequence of analysis



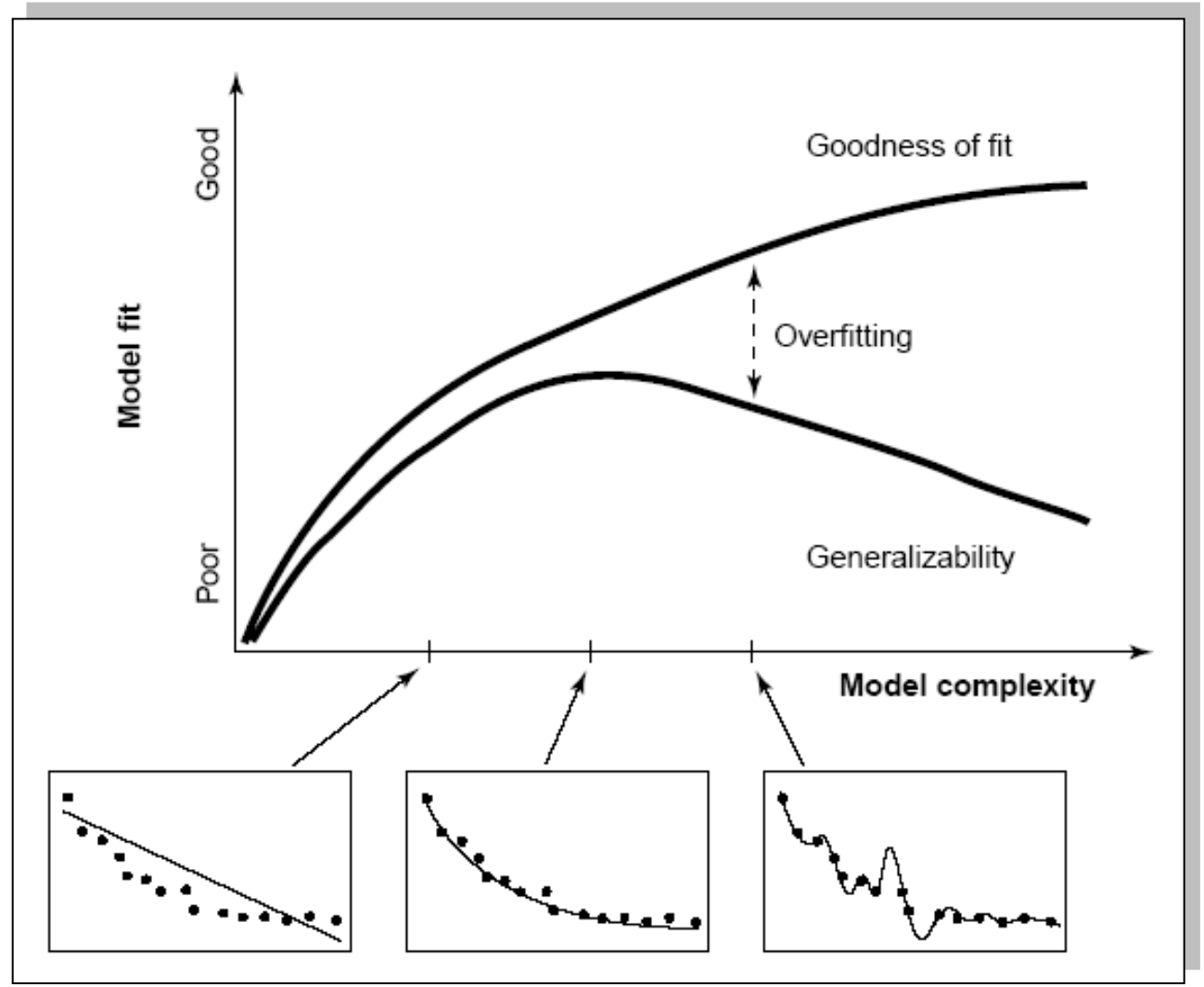
Stephan et al. 2010, *NeuroImage*

Model comparison and selection

Given competing hypotheses on structure & functional mechanisms of a system, which model is the best?

Which model represents the best balance between model fit and model complexity?

For which model m does $p(y|m)$ become maximal?



Pitt & Miyung (2002) *TICS*

Approximations to the model evidence in DCM

Logarithm is a monotonic function → Maximizing log model evidence = Maximizing model evidence

Log model evidence = balance between fit and complexity

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

In SPM2 & SPM5, interface offers 2 approximations:

Akaike Information Criterion: $AIC = \log p(y \mid \theta, m) - p$

Bayesian Information Criterion: $BIC = \log p(y \mid \theta, m) - \frac{p}{2} \log N$

No. of parameters

No. of data points

→ AIC favours more complex models,
BIC favours simpler models.

Penny et al. 2004, *NeuroImage*

The negative free energy approximation

- The negative free energy *F* is a lower bound on the log model evidence:

$$F = \log p(y \mid m) - KL\left[q(\theta), p(\theta \mid y, m)\right]$$

F comprises the expected log likelihood and the Kullback-Leibler (KL) divergence between conditional and prior densities

The complexity term in *F*

- In contrast to AIC & BIC, the complexity term of the negative free energy *F* accounts for parameter interdependencies. Under gaussian assumptions:

$$KL\left[q(\theta), p(\theta \mid m)\right]$$
$$= \frac{1}{2}\ln\left|C_\theta\right| - \frac{1}{2}\ln\left|C_{\theta|y}\right| + \frac{1}{2}\left(\mu_{\theta|y} - \mu_\theta\right)^T C_\theta^{-1}\left(\mu_{\theta|y} - \mu_\theta\right)$$

- The complexity term of *F* is higher
  - the more independent the prior parameters
  - the more dependent the posterior parameters
  - the more the posterior mean deviates from the prior mean

- NB: SPM8 only uses *F* for model selection !

Bayes factors

For a given dataset, to compare two models, we compare their evidences.

positive value, [0; ∞[

$$B_{12} = \frac{p(y \mid m_1)}{p(y \mid m_2)}$$

B_{12}	$p(m_1 \mid y)$	Evidence
1 to 3	50-75%	weak
3 to 20	75-95%	positive
20 to 150	95-99%	strong
≥ 150	≥ 99%	Very strong

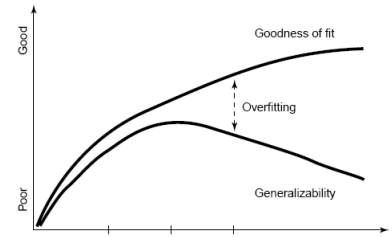
Kass & Raftery 1995, *J. Am. Stat. Assoc.*

Kass & Raftery classification:

or their log evidences

$$\ln(B_{12}) \approx F_1 - F_2$$

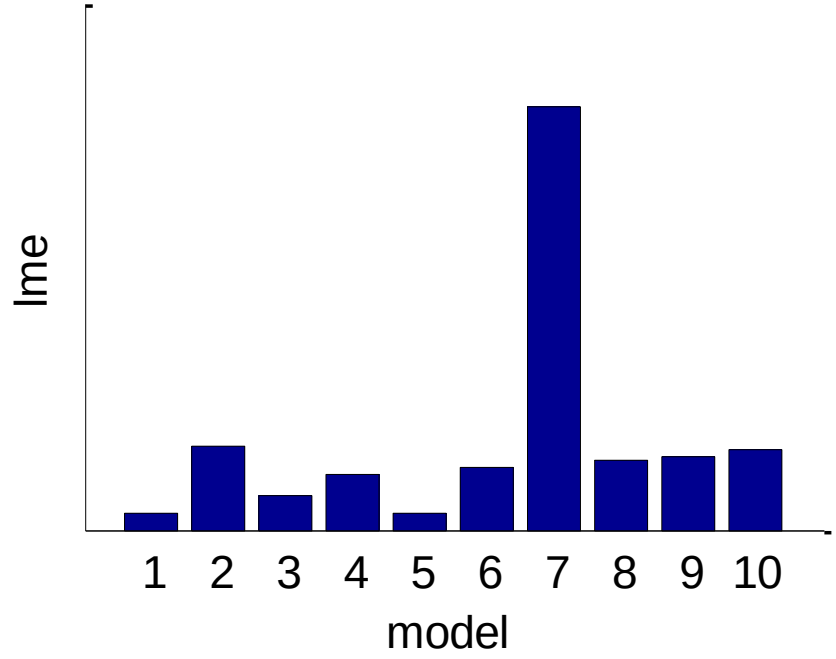
Inference on model space



Model evidence: The optimal balance of fit and complexity

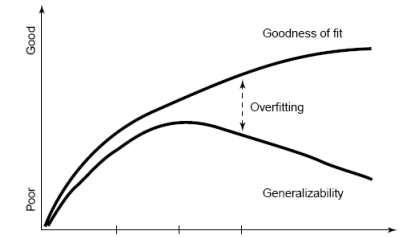
Comparing models

- Which is the best model?



Inference on model space

Model evidence: The optimal balance of fit and complexity



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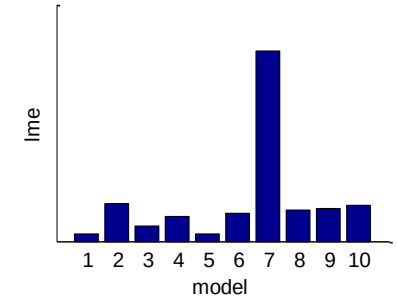
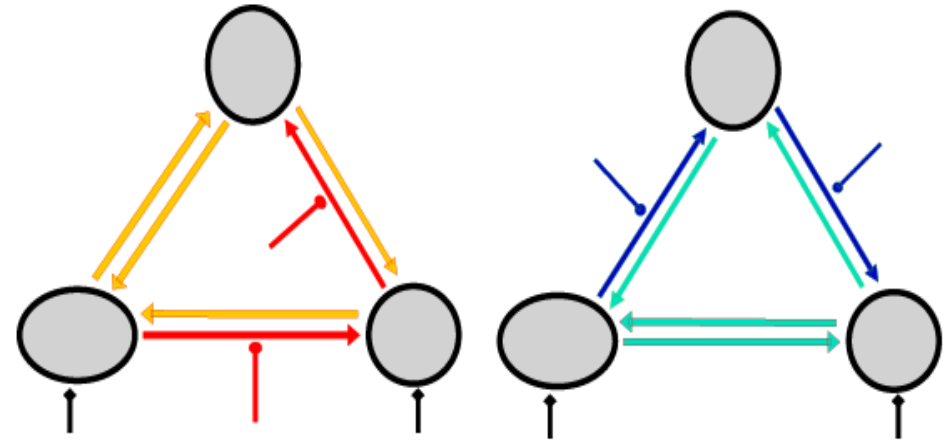


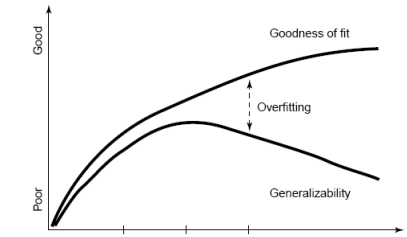
Comparing families of models

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



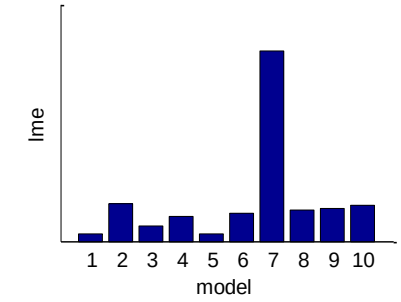
Inference on model space

Model evidence: The optimal balance of fit and complexity



Comparing models

- Which is the best model?



Comparing families of models

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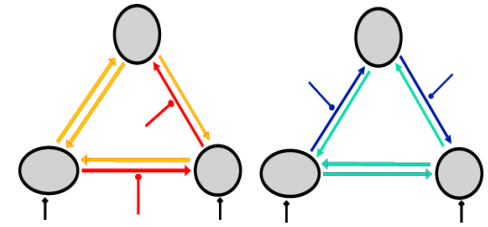
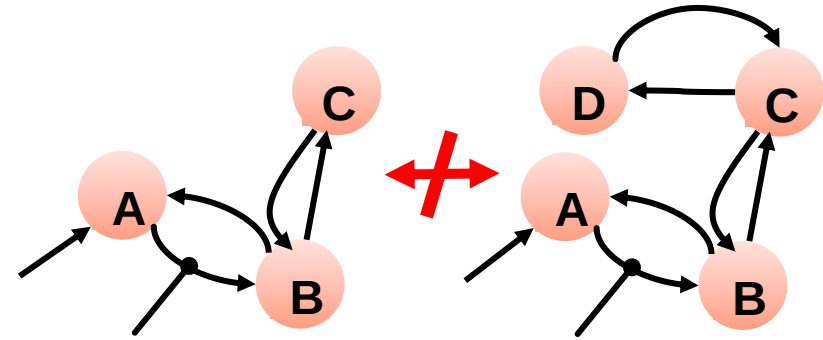


Only compare models with the same data



To recap... so, DCM….

- enables one to **infer hidden neuronal processes** from fMRI data
- 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 can change and the method extended to other modalities e.g. ERPs, LFPs)

Overview

Brain connectivity: types & definitions

Anatomical connectivity
Functional connectivity
Effective connectivity

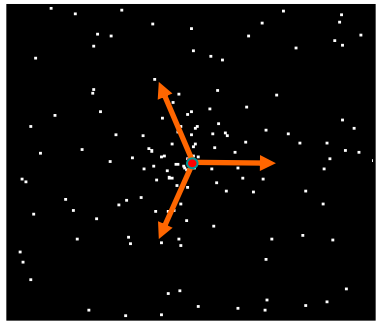
Dynamic causal models (DCMs)

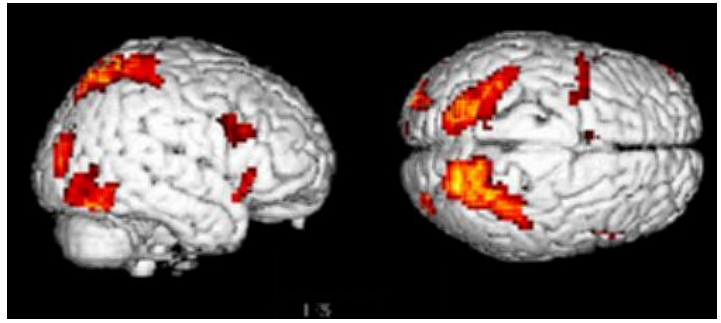
Neuronal model
Hemodynamic model
Estimation: Bayesian framework

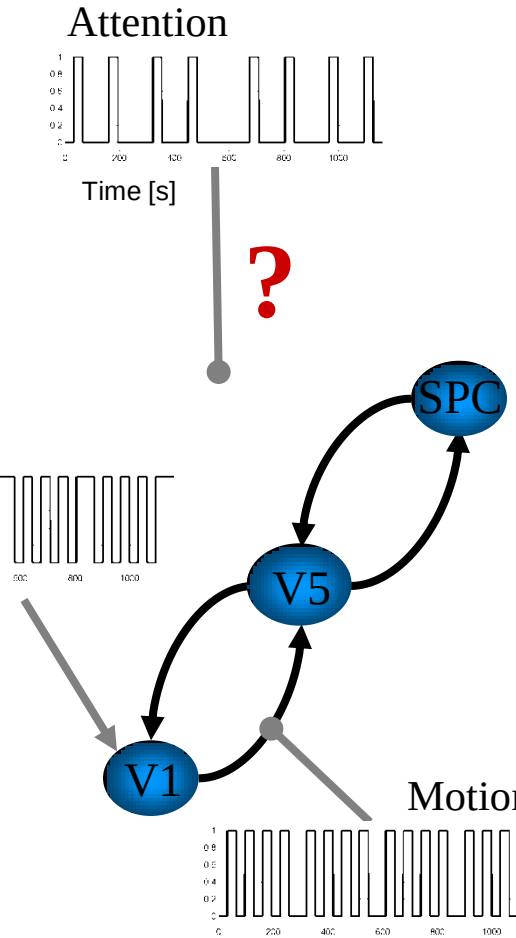
Applications & extensions of DCM to fMRI data

Attention to motion in the visual system

We used this model to assess the site of ***attention modulation*** during *visual motion processing* in an fMRI paradigm reported by *Büchel & Friston.*





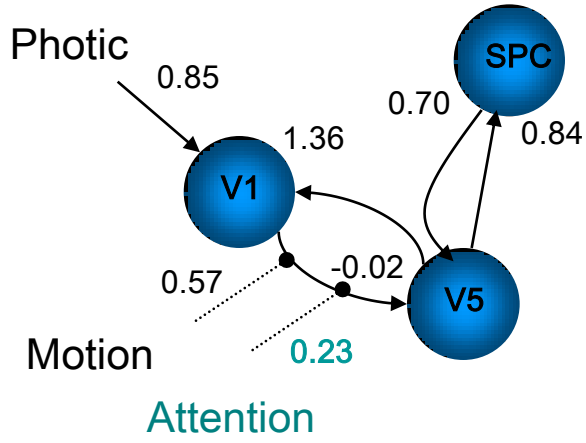


- fixation only
- observe static dots + photic → V1
- observe moving dots + motion → V5
- task on moving dots + attention → V5 + parietal cortex

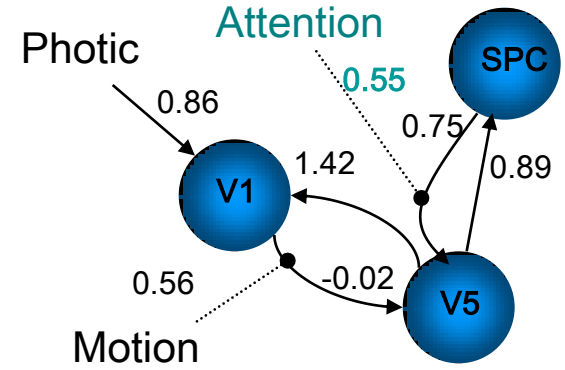
Friston et al. 2003,
NeuroImage

Comparison of two simple models

Model 1:
attentional modulation
of V1→V5



Model 2:
attentional modulation
of SPC→V5



Bayesian model selection:

Model 1 better than model 2

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

→ Decision for model 1:

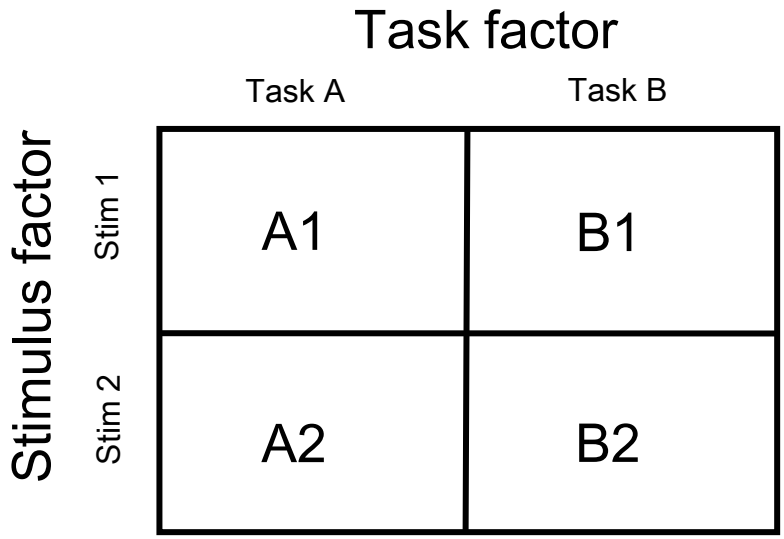
in this experiment, attention primarily modulates V1→V5

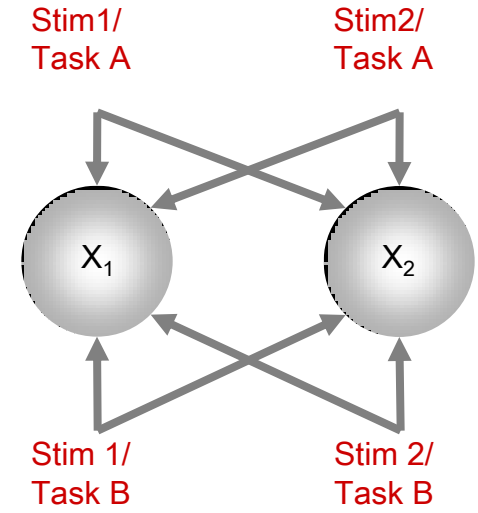
Planning a DCM-compatible study

- Suitable experimental design:
  - any design that is suitable for a GLM
  - preferably multi-factorial (e.g. 2 x 2)
    - e.g. one factor that varies the driving (sensory) input
    - and one factor that varies the contextual input

- Hypothesis and model:
  - Define specific *a priori* hypothesis
  - Which parameters are relevant to test this hypothesis?
  - If you want to verify that intended model is suitable to test this hypothesis, then use simulations
  - Define criteria for inference
  - What are the alternative models to test?

Multifactorial design: explaining interactions with DCM

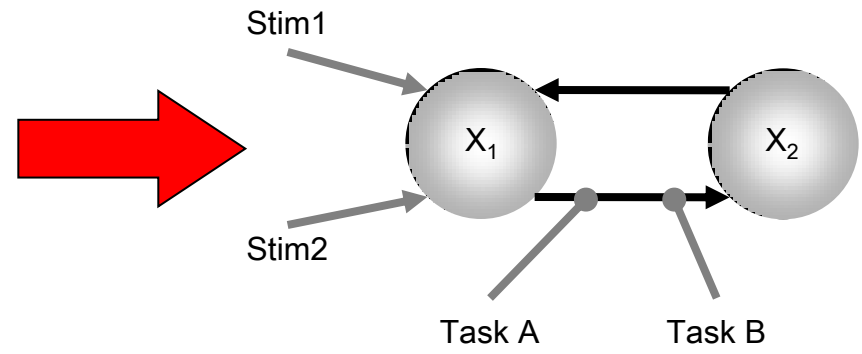




GLM

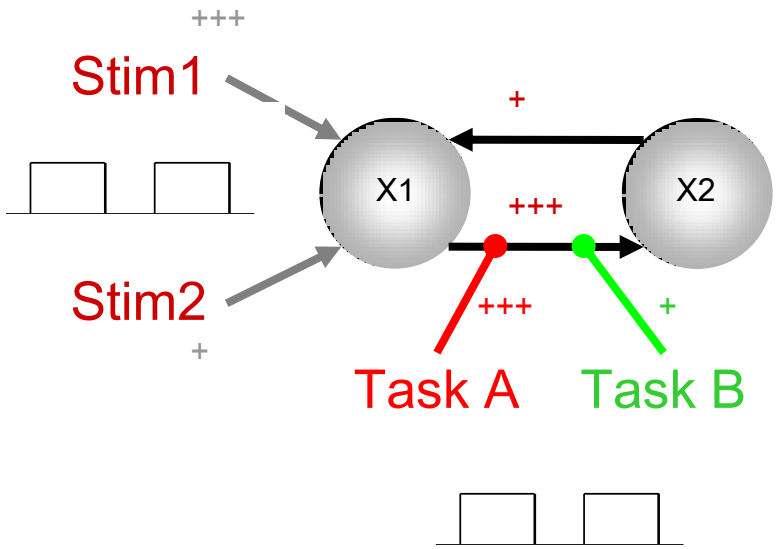
Let's assume that an SPM analysis shows a main effect of stimulus in X_1 and a stimulus × task interaction in X_2.

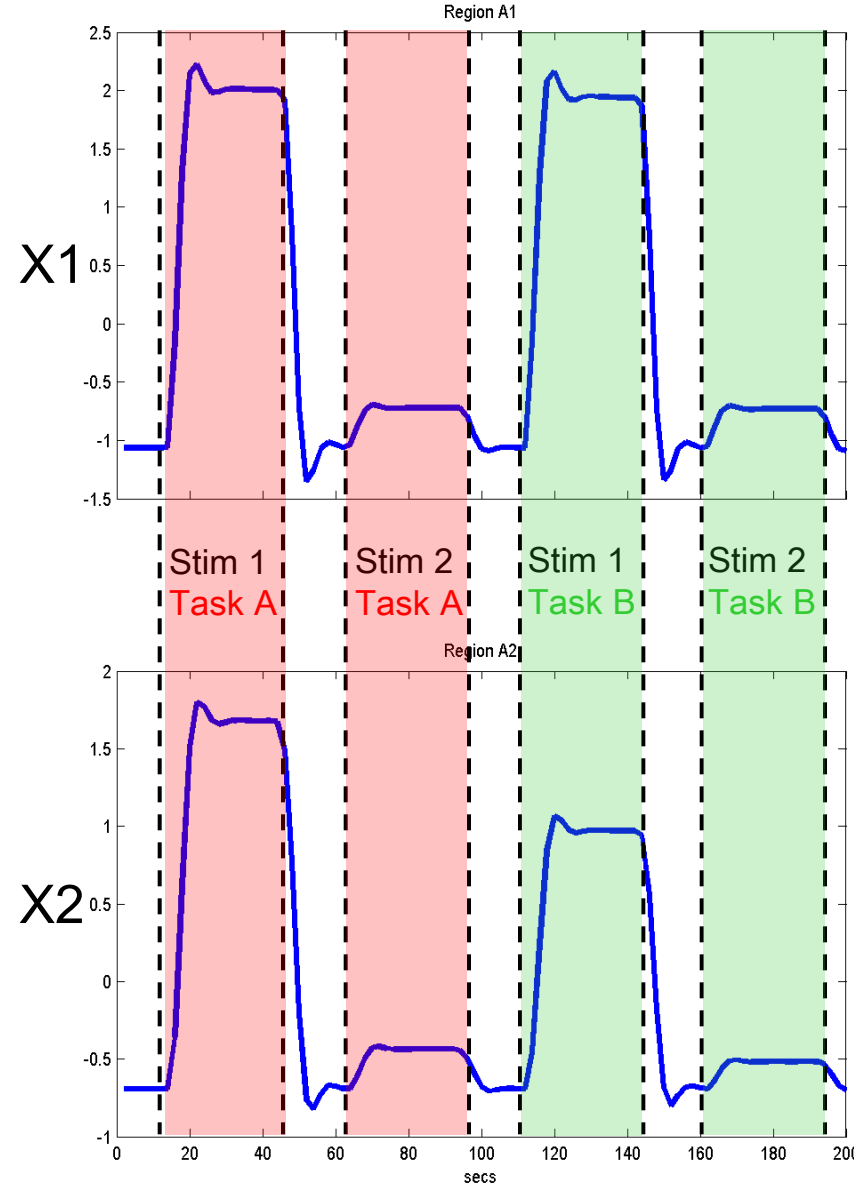
How do we model this using DCM?

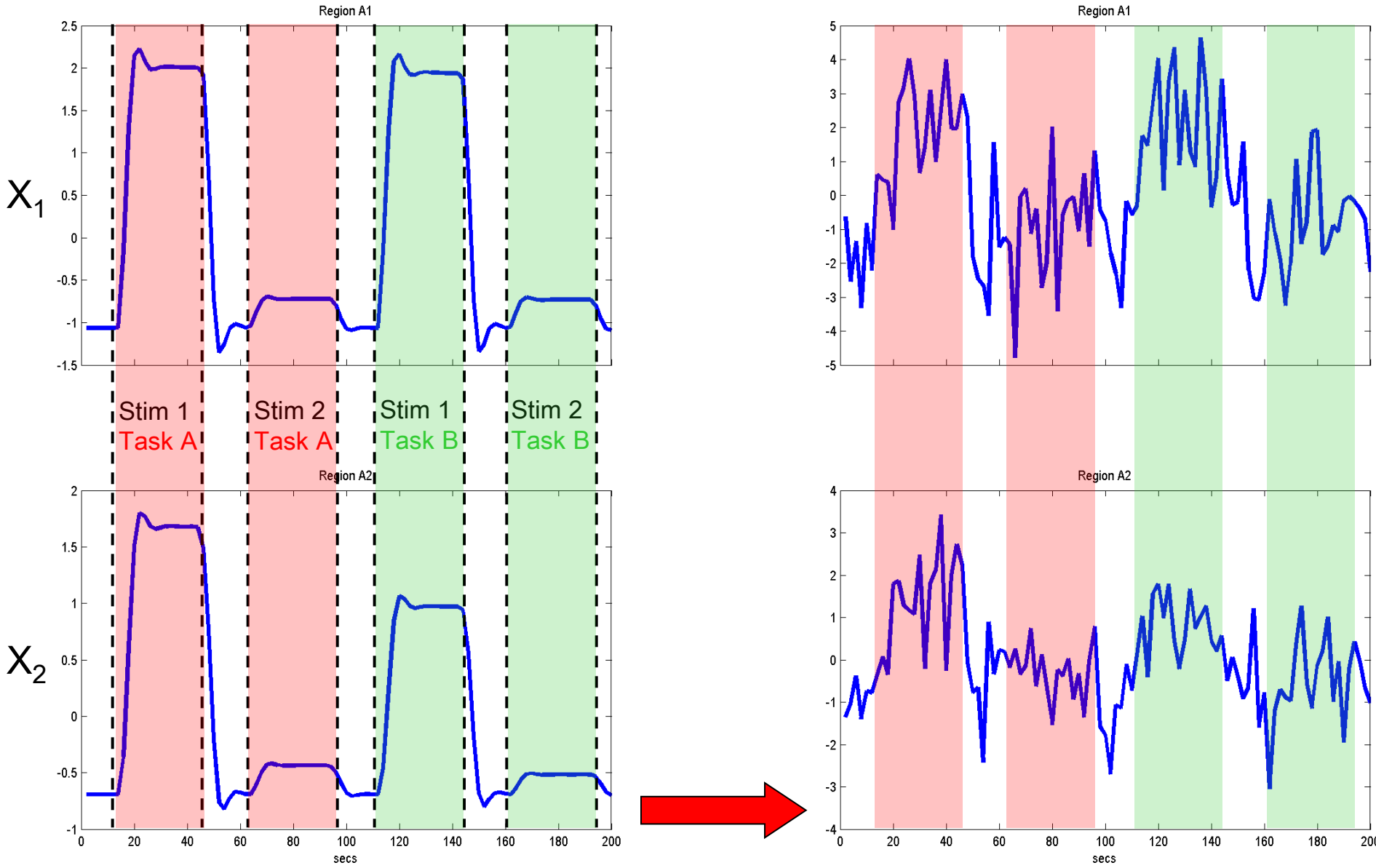


DCM

Simulated data





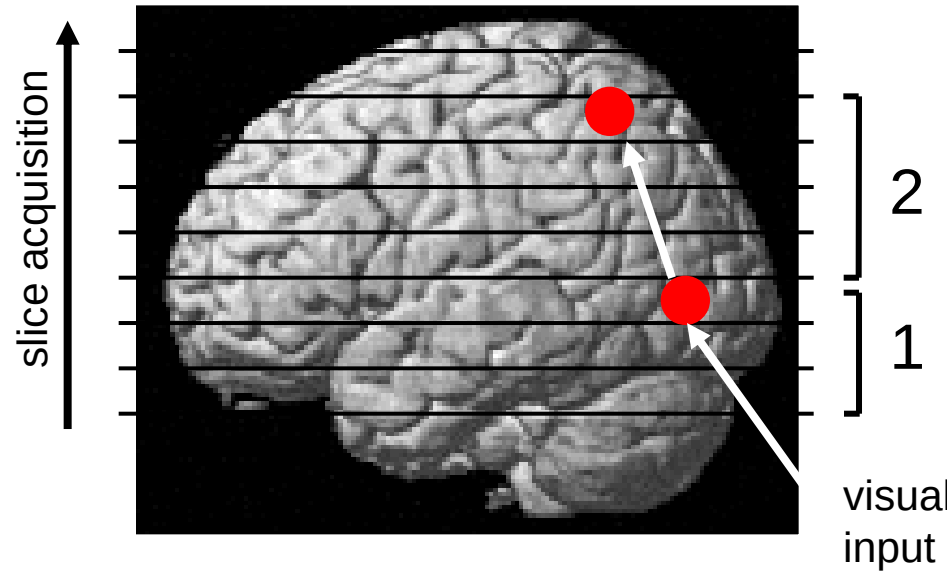


plus added noise (SNR=1)

Slice timing model

- potential timing problem in DCM:

  temporal shift between regional time series because of multi-slice acquisition



- Solution:
  - Modelling of (known) slice timing of each area.

Slice timing extension now allows for any slice timing differences!

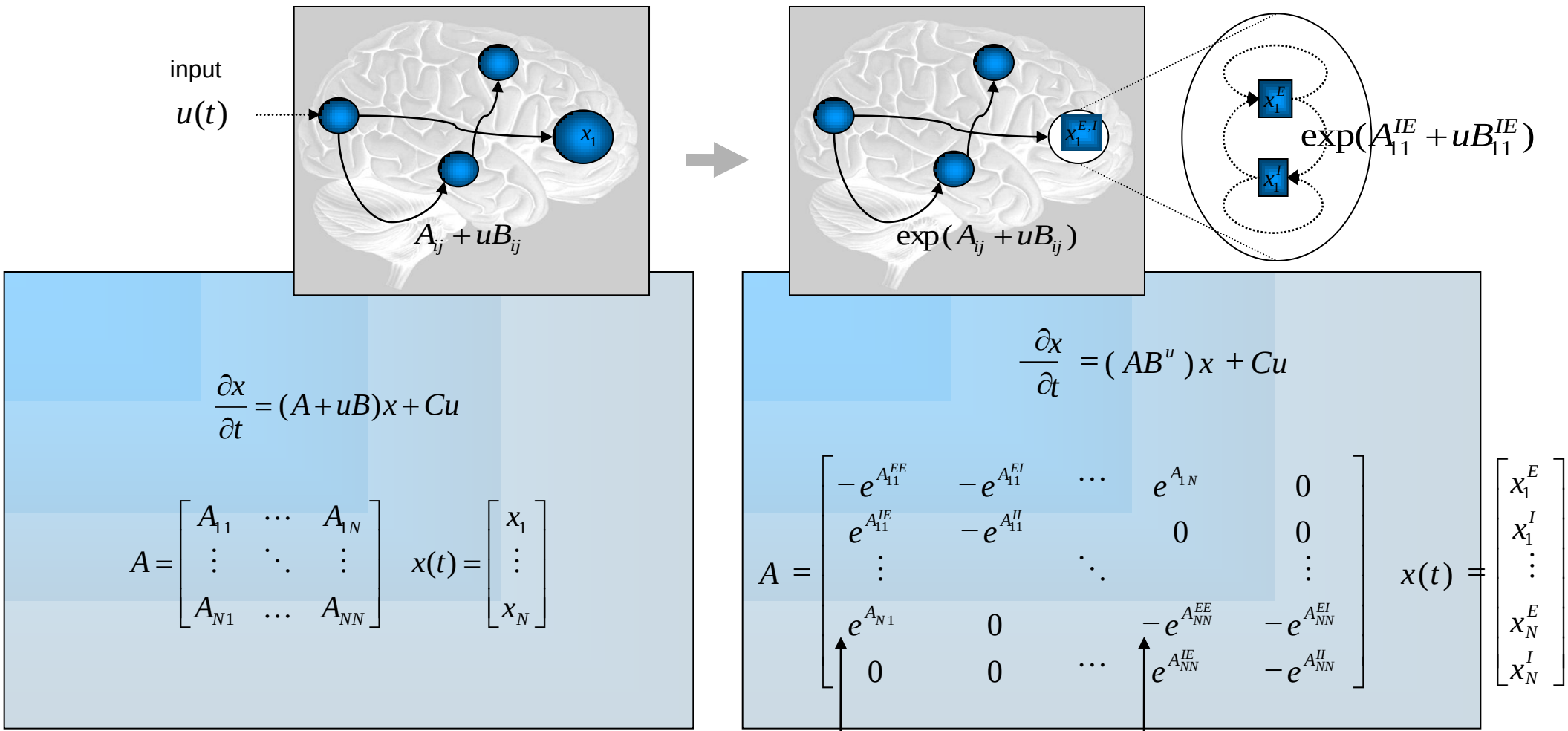
Long TRs (> 2 sec) no longer a limitation.

(Kiebel et al., 2007)

Two-state model

Single-state DCM

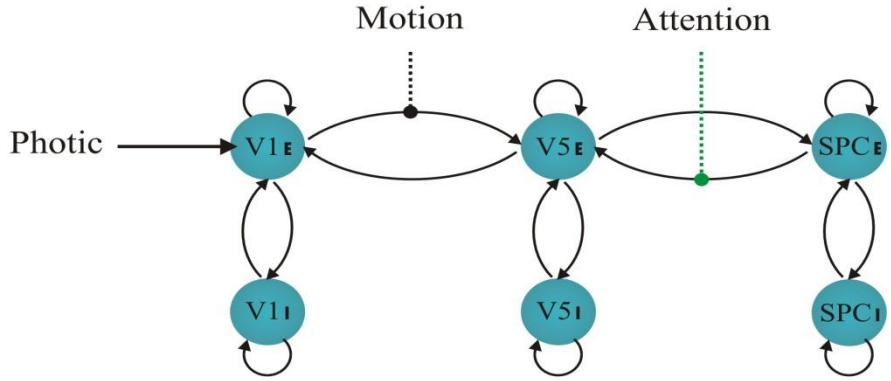
Two-state DCM



Example: Two-state Model Comparison

DCM for *Büchel & Friston*

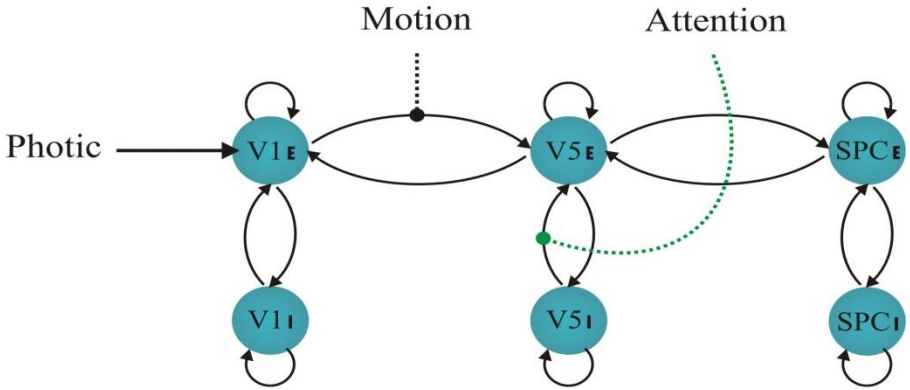
Model 1 - BCW



Attention

$$A = \begin{bmatrix} -EE_{V1} & -EI_{V1} & E_{V1}E_{V5} & 0 & 0 & 0 \\ IE_{V1} & -II_{V1} & 0 & 0 & 0 & 0 \\ E_{V5}E_{V1} & 0 & -EE_{V5} & -EI_{V5} & E_{V5}E_{SPC} & 0 \\ 0 & 0 & IE_{V5} & -II_{V5} & 0 & 0 \\ 0 & 0 & E_{SPC}E_{V5} & 0 & -EE_{SPC} & -EI_{SPC} \\ 0 & 0 & 0 & 0 & IE_{SPC} & -II_{SPC} \end{bmatrix}$$

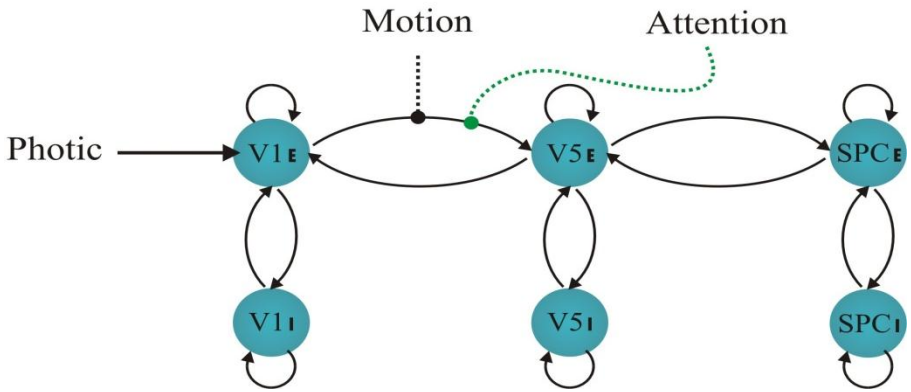
Model 2 - Intr



Attention

$$A = \begin{bmatrix} -EE_{V1} & -EI_{V1} & E_{V1}E_{V5} & 0 & 0 & 0 \\ IE_{V1} & -II_{V1} & 0 & 0 & 0 & 0 \\ E_{V5}E_{V1} & 0 & -EE_{V5} & -EI_{V5} & E_{V5}E_{SPC} & 0 \\ 0 & 0 & IE_{V5} & -II_{V5} & 0 & 0 \\ 0 & 0 & E_{SPC}E_{V5} & 0 & -EE_{SPC} & -EI_{SPC} \\ 0 & 0 & 0 & 0 & IE_{SPC} & -II_{SPC} \end{bmatrix}$$

Model 3 - FWD

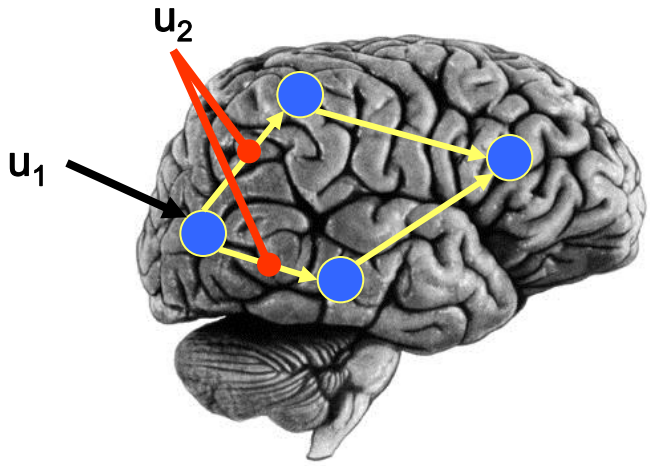


Attention

$$A = \begin{bmatrix} -EE_{V1} & -EI_{V1} & E_{V1}E_{V5} & 0 & 0 & 0 \\ IE_{V1} & -II_{V1} & 0 & 0 & 0 & 0 \\ E_{V5}E_{V1} & 0 & -EE_{V5} & -EI_{V5} & E_{V5}E_{SPC} & 0 \\ 0 & 0 & IE_{V5} & -II_{V5} & 0 & 0 \\ 0 & 0 & E_{SPC}E_{V5} & 0 & -EE_{SPC} & -EI_{SPC} \\ 0 & 0 & 0 & 0 & IE_{SPC} & -II_{SPC} \end{bmatrix}$$

Nonlinear DCM for fMRI

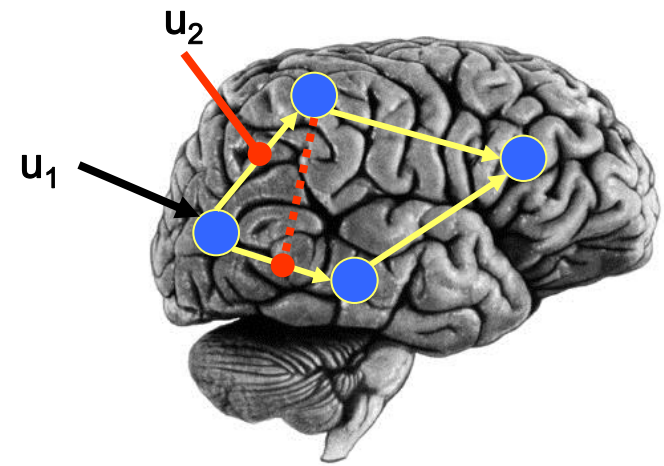
bilinear DCM



Bilinear state equation

$$\frac{dx}{dt} = \left(A + \sum_{i=1}^{m} u_i B^{(i)} \right) x + Cu$$

nonlinear DCM



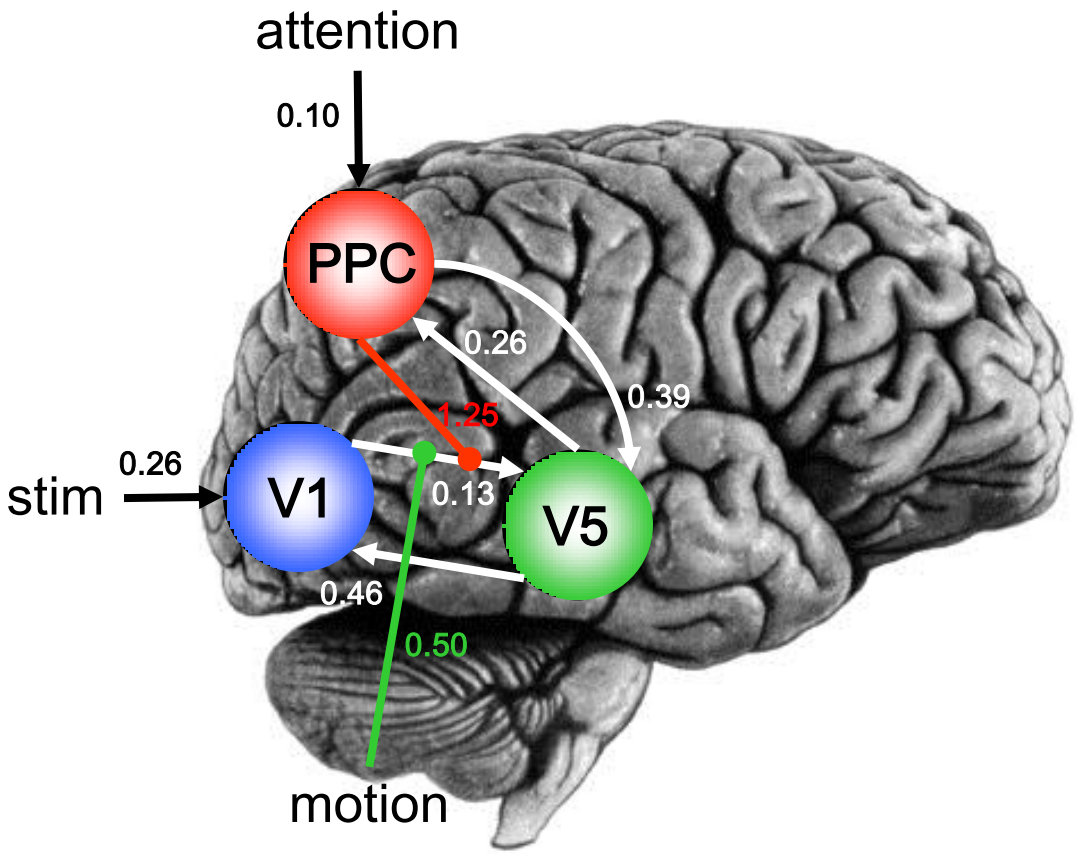
Nonlinear state equation

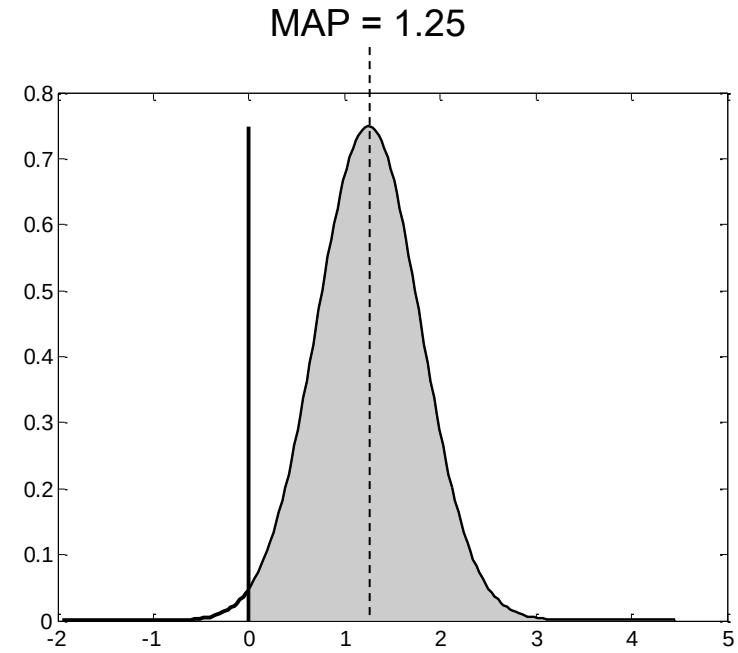
$$\frac{dx}{dt} = \left(A + \sum_{i=1}^{m} u_i B^{(i)} + \sum_{j=1}^{n} x_j D^{(j)} \right) x + Cu$$

Here DCM can model activity-dependent changes in connectivity; how connections are enabled or gated by activity in one or more areas.

Nonlinear DCM for fMRI

Can V5 activity during attention to motion be explained by allowing activity in PPC to modulate the V1-to-V5 connection?



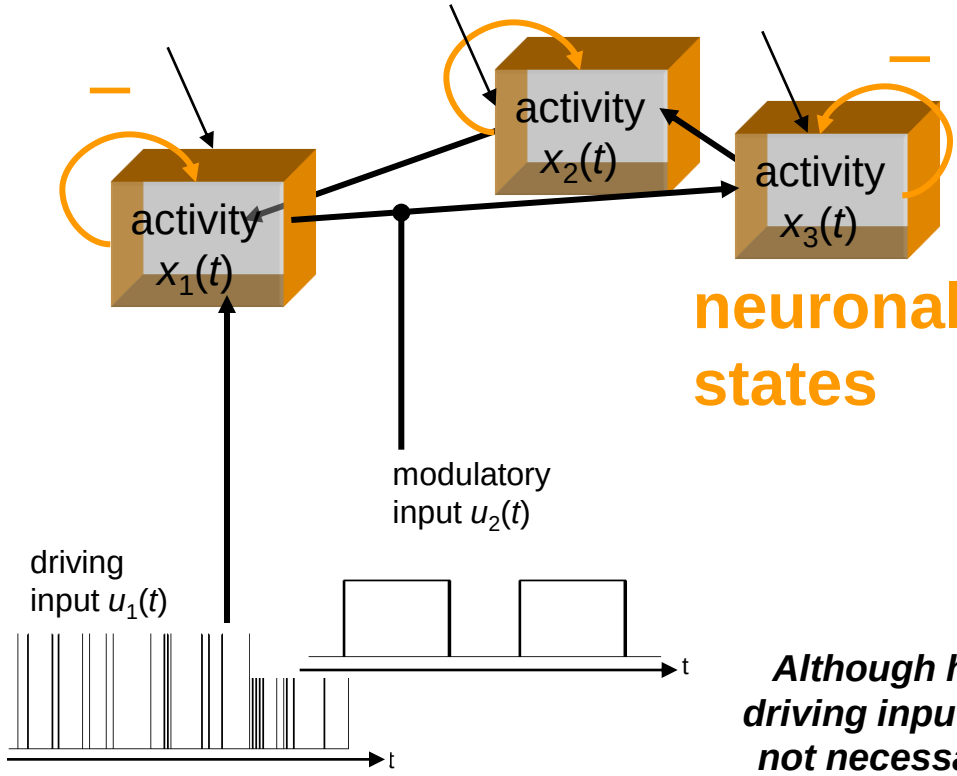


$$p(D^{PPC}_{V5,V1} > 0 \mid y) = 99.1\%$$

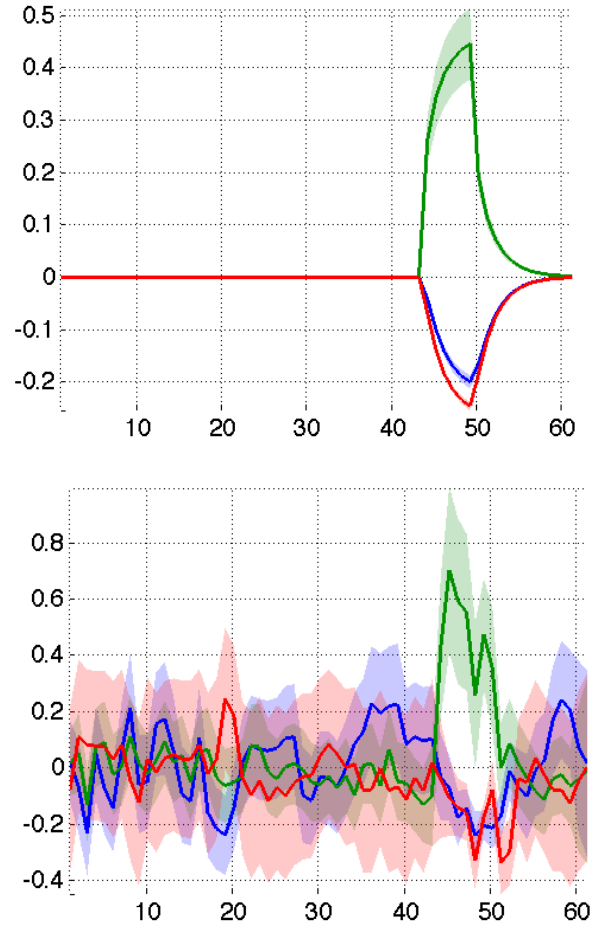
Stephan et al. 2008, *NeuroImage*

Stochastic DCMs

Stochastic innovations: variance hyperparameter



Although here driving inputs are not necessary...



Inversion: Generalised filtering (under the Laplace assumption)

Daunizeau et al, 2009

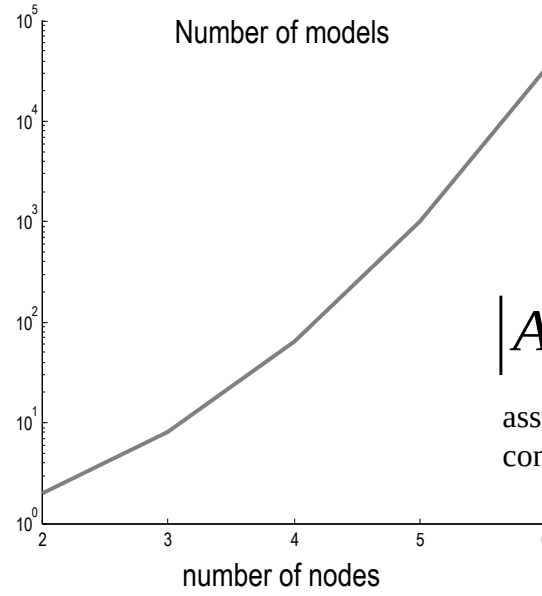
Friston et al, 2008

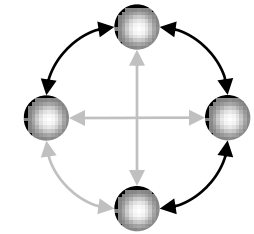
Bayesian Model Selection for large model spaces

- for less constrained model spaces, search methods are needed

- fast model scoring via the Savage-Dickey density ratio:

$$\ln p\left(y \mid m_i\right)$$
$$\approx \ln q\left(\theta_i = 0 \mid m_i\right) - \ln p\left(\theta_i = 0 \mid m_F\right)$$

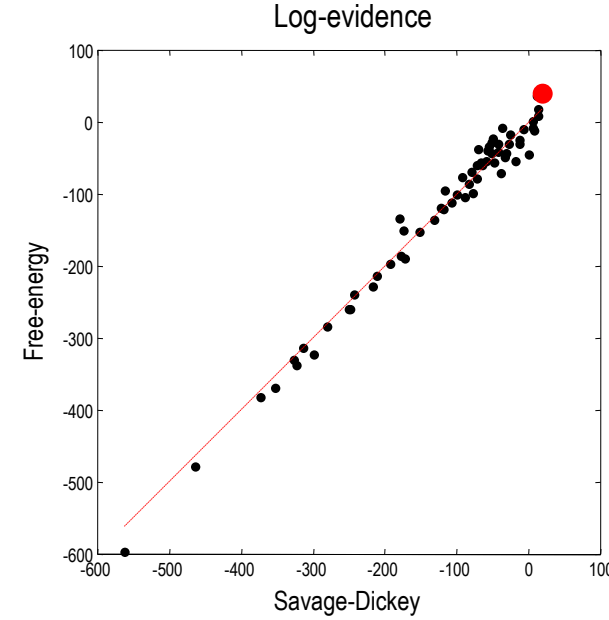




$$\left|A\left(m_i\right)\right| = 2^{n(n-1)/2}$$

assuming reciprocal connections

Life easier for more exploratory approachs!



Friston et al. 2011, *NeuroImage*
Friston & Penny 2011, *NeuroImage*

The evolution of DCM in SPM

- DCM is not one specific model, but a framework for Bayesian inversion of dynamical systems

- The default implementation in SPM is evolving over time:
- better numerical routines for inversion
- changes in prior to cover new variants

To enable replication of your results, you should state which SPM version you are using!

Some introductory references

- **The first DCM paper:** Dynamic Causal Modelling (2003). Friston et al. *NeuroImage* 19:1273-1302.
- **Physiological validation of DCM for fMRI:** Identifying neural drivers with functional MRI: an electrophysiological validation (2008). David et al. *PLoS Biol.* 6 2683–2697
- **Hemodynamic model:** Comparing hemodynamic models with DCM (2007). Stephan et al. *NeuroImage* 38:387-401
- **Nonlinear DCMs:**Nonlinear Dynamic Causal Models for FMRI (2008). Stephan et al. *NeuroImage* 42:649-662
- **Two-state model:** Dynamic causal modelling for fMRI: A two-state model (2008). Marreiros et al. *NeuroImage* 39:269-278
- **Group Bayesian model comparison:** Bayesian model selection for group studies (2009). Stephan et al. *NeuroImage* 46:1004-10174
- **10 Simple Rules for DCM** (2010). Stephan et al. *NeuroImage 52*.

Thank you for your attention!!!