Dynamic causal modeling

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Methods and Models for fMRI analysis, Lecture
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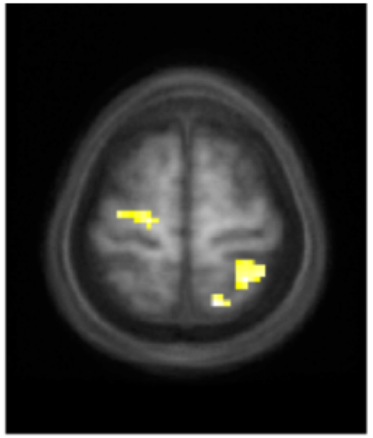
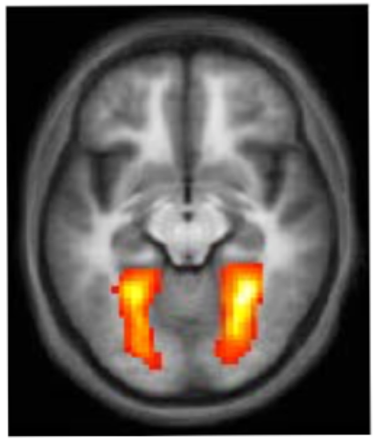
FROM FUNCTIONAL SEGREGATION TO FUNCTIONAL INTEGRATION

localization of brain activity
functional segregation



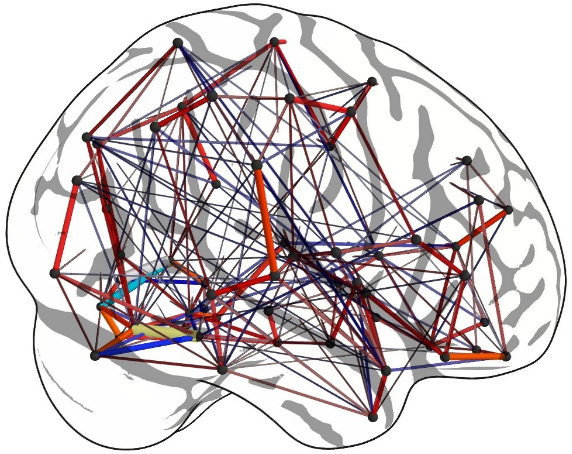
u_1



$u_1 \times u_2$

"Where in the brain does my experimental manipulation have an effect?"

analysis of brain connectivity
functional integration

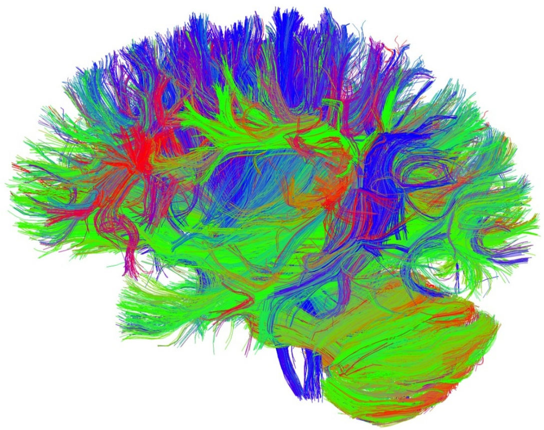


https://team.inria.fr/parietal/files/2013/02/pc_dag.jpg

"How do brain regions interact with each other? How does my experimental manipulation propagate through the network?"

DIFFERENT FORMS OF BRAIN CONNECTIVITY

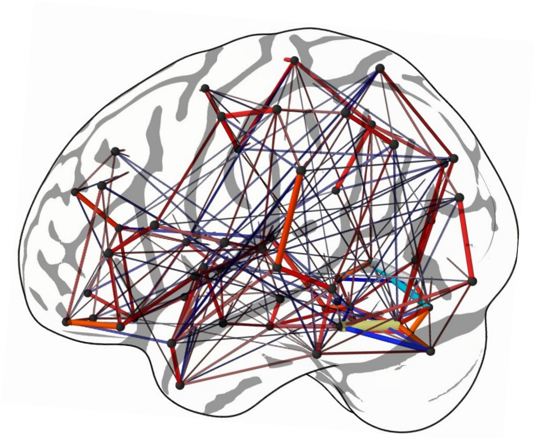
structural connectivity



https://optimalsurgerytle.weebly.com/imaging-and-dataset.html

- presence of anatomical/ physical connections
- Diffusion weighted imaging (DWI), tractography, tracer studies

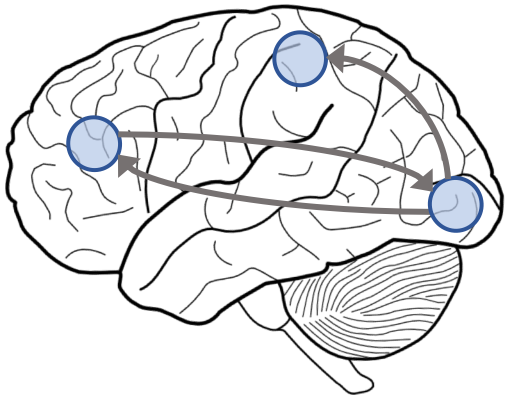
functional connectivity



https://team.inria.fr/parietal/files/2013/02/pc_dag.jpg

- statistical dependencies between regional time series
- correlations, Independent Component Analysis (ICA)

effective connectivity



http://www.clker.com/cliparts/e/5/Q/i/e/o/brain-line-drawing-md.png

- directed influences between neuronal populations
- Dynamic causal modeling (DCM)

adapted from: Sporns, 2007, *Scholarpedia*

DYNAMIC CAUSAL MODELING

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Available online at www.sciencedirect.com
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NeuroImage 19 (2003) 1273–1302

NeuroImage
www.elsevier.com/locate/ynimg

Dynamic causal modelling

K.J. Friston,* L. Harrison, and W. Penny

The Wellcome Department of Imaging Neuroscience, Institute of Neurology, Queen Square, London WC1N 3BG, UK

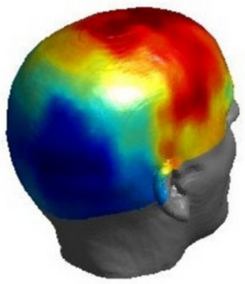
Received 18 October 2002; revised 7 March 2003; accepted 2 April 2003

- Dynamic causal modeling (DCM) for functional magnetic resonance imaging (fMRI) data was introduced in 2003 by Karl Friston, Lee Harrison and Will Penny (NeuroImage 19:1273-1302)
- Allows effective (directed) connectivity analyses based on fMRI data

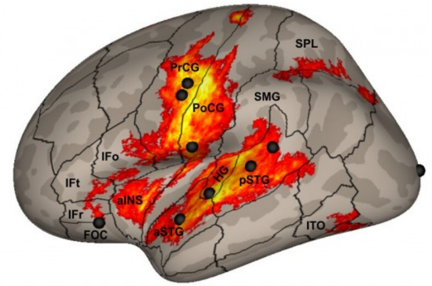
Friston et al., 2003, *NeuroImage*

Dynamic causal modeling

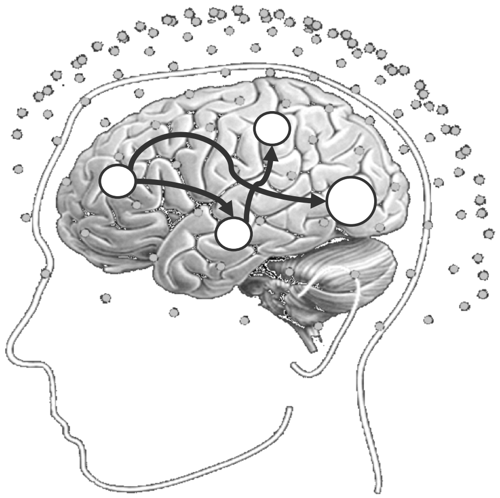
EEG, MEG



fMRT



http://sites.bu.edu/guentherlab/



Forward model:

Predicting measured activity

$$y = g(x, \theta) + \varepsilon$$

Model inversion:

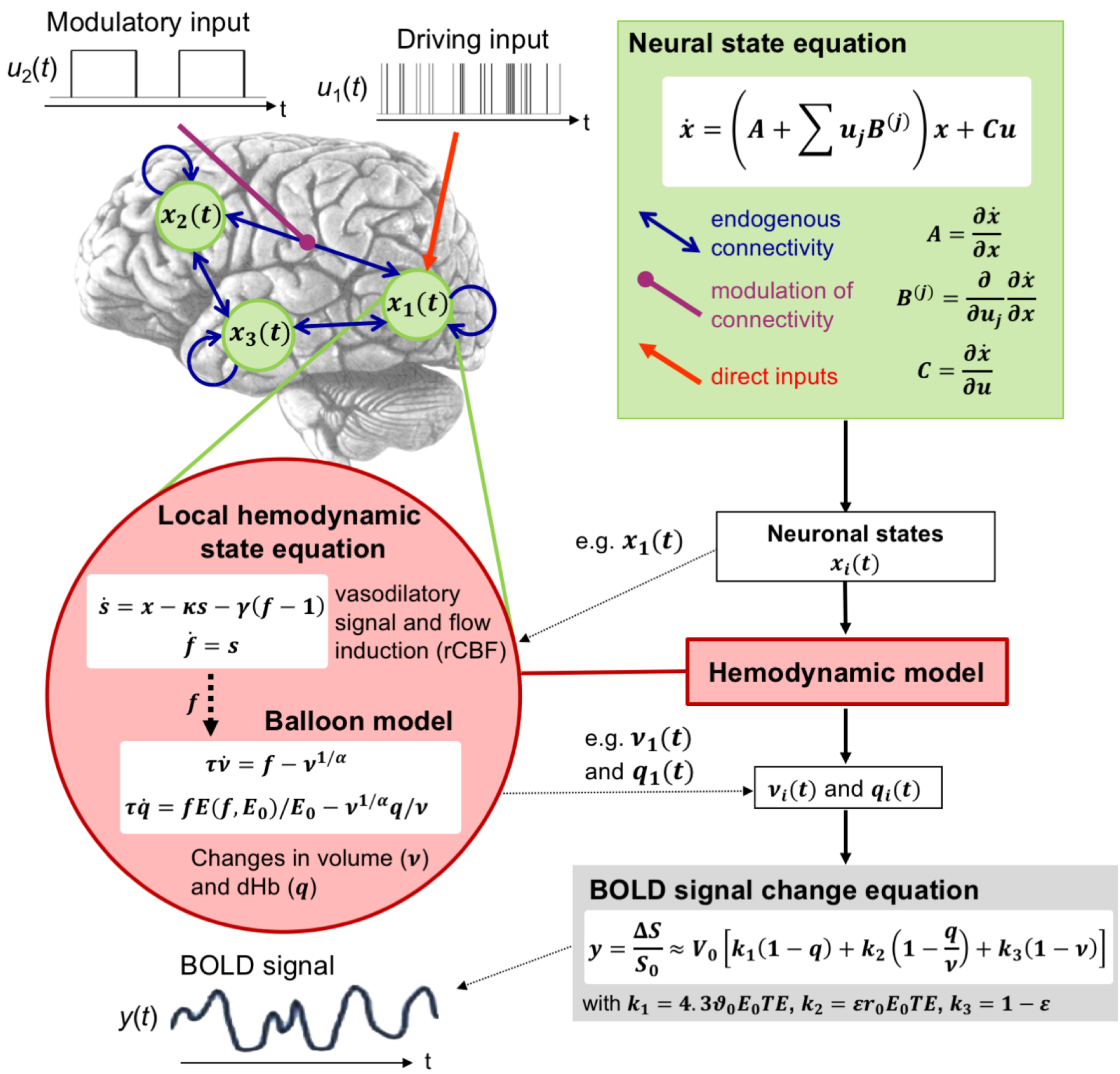
Estimating neuronal mechanisms

$$\frac{dx}{dt} = f(x, u, \theta) + \omega$$

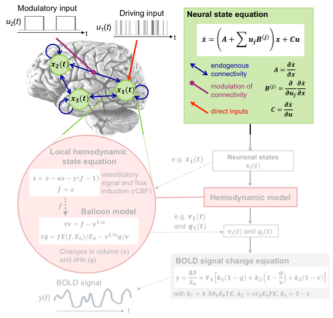
Friston et al., 2003, *NeuroImage*; David et al., 2006, *NeuroImage*

THEORY

DCM FOR FMRI (OVERVIEW)



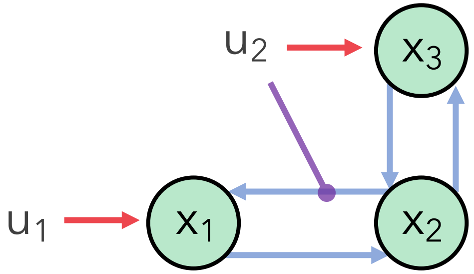
Friston et al., 2003, *NeuroImage*; Stephan et al., 2015, *Neuron*



Neuronal state equation

$$\frac{dx}{dt} = f(x,u) \approx \boxed{f(x_0,0) + \overset{A}{\frac{\partial f}{\partial x}}x + \overset{C}{\frac{\partial f}{\partial u}}u + \overset{B}{\frac{\partial^2 f}{\partial x \partial u}}ux} + \frac{\partial^2 f}{\partial x^2}\frac{x^2}{2} + \cdots$$

bilinear model



Friston et al., 2003, *NeuroImage*; Stephan et al., 2008, *NeuroImage*

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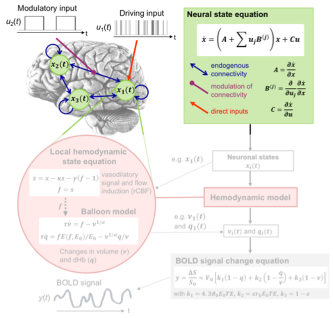
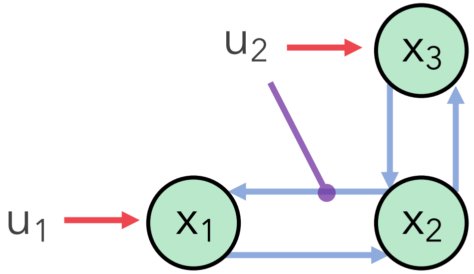
Neuronal state equation

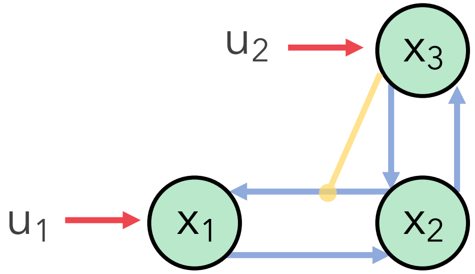


$$\frac{dx}{dt} = f(x,u) \approx f(x_0,0) + \overbrace{\frac{\partial f}{\partial x}}^{A}x + \overbrace{\frac{\partial f}{\partial u}}^{C}u + \overbrace{\frac{\partial^2 f}{\partial x \partial u}}^{B}ux + \overbrace{\frac{\partial^2 f}{\partial x^2}}^{D}\frac{x^2}{2} + \cdots$$

bilinear model

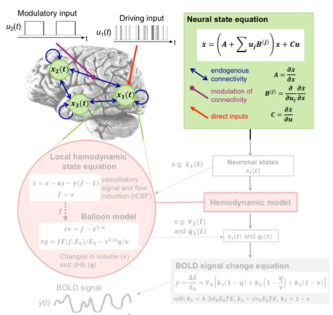
nonlinear model



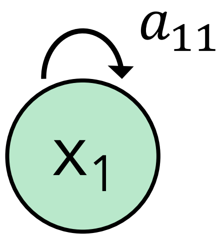


Friston et al., 2003, *NeuroImage*; Stephan et al., 2008, *NeuroImage*

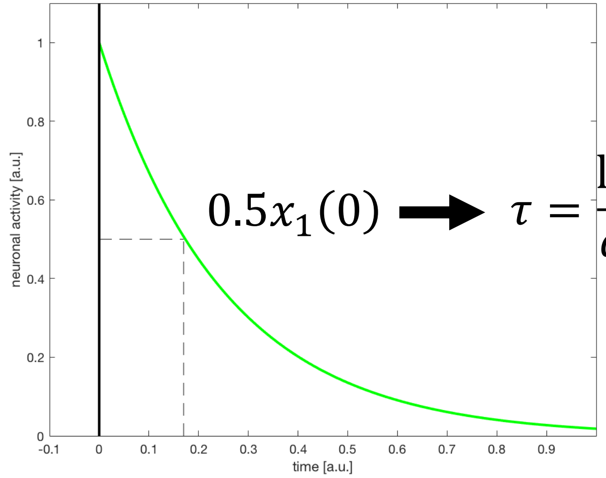
Neuronal state equation



DCM effective connectivity parameters are rate constants

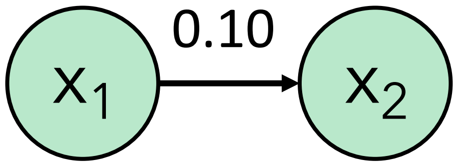


$$\frac{dx_1}{dt} = a_{11} x_1 \longrightarrow x_1(t) = x_1(0) \cdot exp(a_{11} t)$$

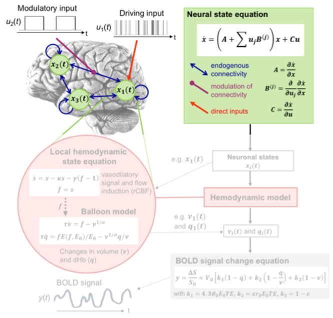


$$0.5 x_1(0) \longrightarrow \tau = \frac{\ln 2}{a_{11}}$$

Friston et al., 2003, *NeuroImage*

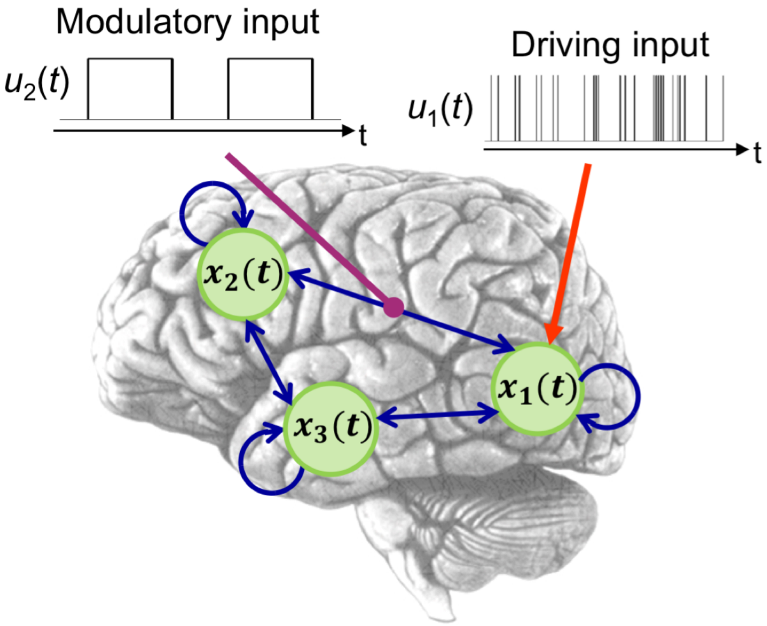


If $\text{region}_1 \rightarrow \text{region}_2$ is $0.10 s^{-1}$, this means that, per unit time, the increase in activity in region_2 corresponds to 10% of the current activity in region_1



Neuronal state equation

Interim summary: bilinear neuronal state equation



Friston et al., 2003, *NeuroImage*

State change

External inputs

Current state

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

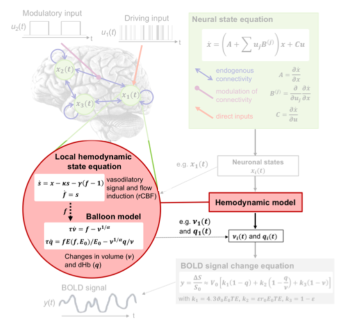
$$\theta = \{A, B^{(1)}, \ldots, B^{(m)}, C\}$$

Endogenous connectivity

Modulatory connectivity

Driving inputs

HEMODYNAMIC MODEL



Neuronal dynamics only indirectly observable via hemodynamic response

↑ neuronal activity

↑ blood flow

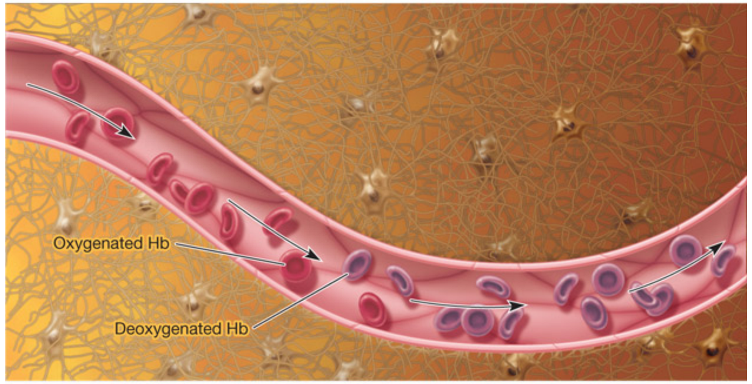
↑ oxygenated Hb

↑ T2*

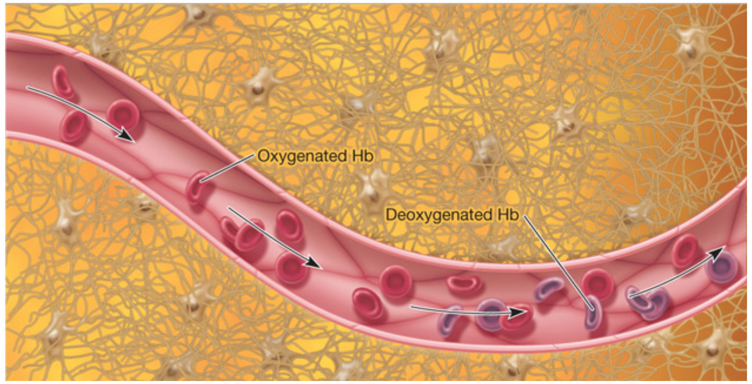
↑ fMRI signal

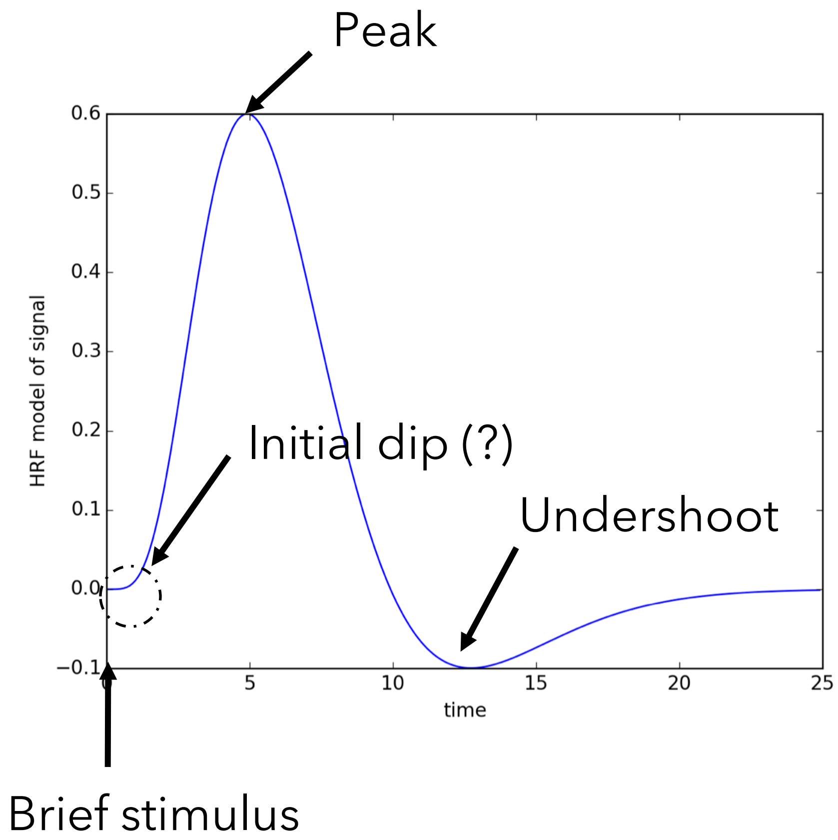
Huettel et al., 2004, *NeuroImage*

Rest



Activity





HEMODYNAMIC MODEL

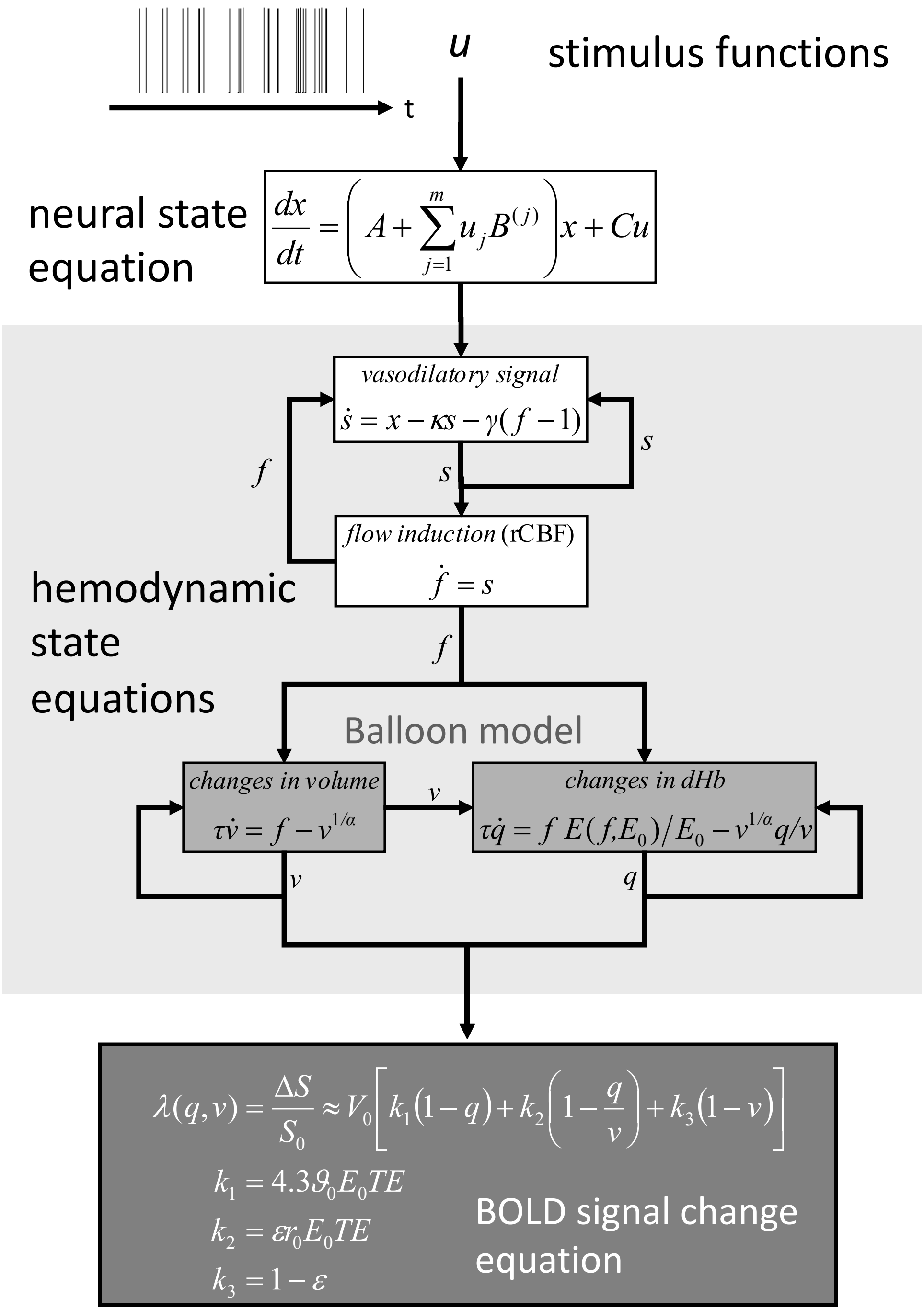
6 hemodynamic parameters:

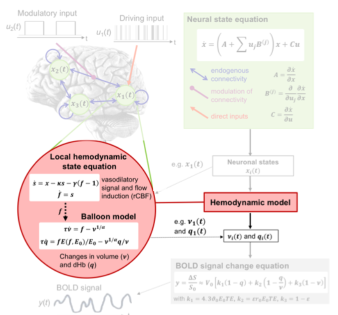
$$\theta^h = \{\kappa, \gamma, \tau, \alpha, \rho, \varepsilon\}$$

Important for model fitting, but typically of no interest for statistical inference.

Hemodynamic parameters are computed separately for each region → region specific HRFs!

Friston et al., 2003, *NeuroImage*; Stephan et al., 2007, *NeuroImage*





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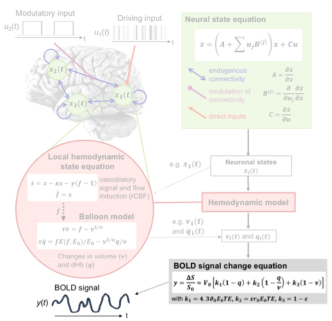
BOLD SIGNAL CHANGE EQUATION



Resting blood volume — Deoxyhemoglobin content — Blood volume

$$\lambda(q,v) = \frac{\Delta S}{S_0} \approx V_0\left[k_1(1-q) + k_2\left(1-\frac{q}{v}\right) + k_3(1-v)\right]$$

$$k_1 = 4.3\vartheta_0 E_0 TE$$

$$k_2 = \varepsilon r_0 E_0 TE$$

$$k_3 = 1 - \varepsilon$$

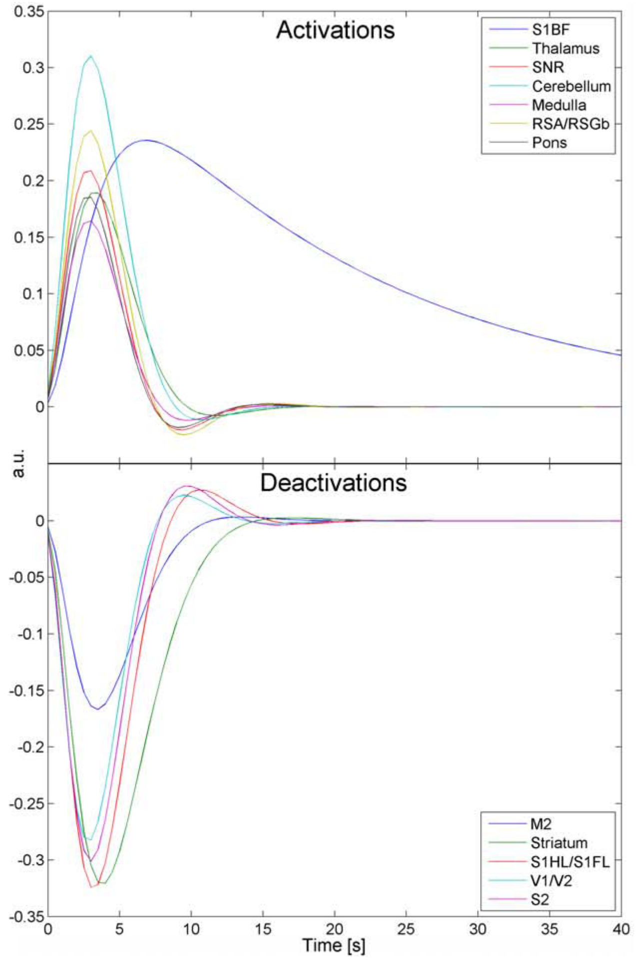
$$V_0 = 0.04$$

$$E_0 = 0.4$$

At 1.5 Tesla	At 3 Tesla	At 7 Tesla
$\vartheta_0 = 40.3\ \mathrm{s}^{-1}$	$\vartheta_0 = 80.3\ \mathrm{s}^{-1}$	$\vartheta_0 = 188\ \mathrm{s}^{-1}$
$r_0 = 25\ \mathrm{s}^{-1}$	$r_0 = 110\ \mathrm{s}^{-1}$	$r_0 = 340\ \mathrm{s}^{-1}$
$TE \approx 0.04\ \mathrm{s}$	$TE \approx 0.035\ \mathrm{s}$	$TE \approx 0.025\ \mathrm{s}$
$\varepsilon \approx 1.28$	$\varepsilon \approx 0.47$	$\varepsilon \approx 0.026$

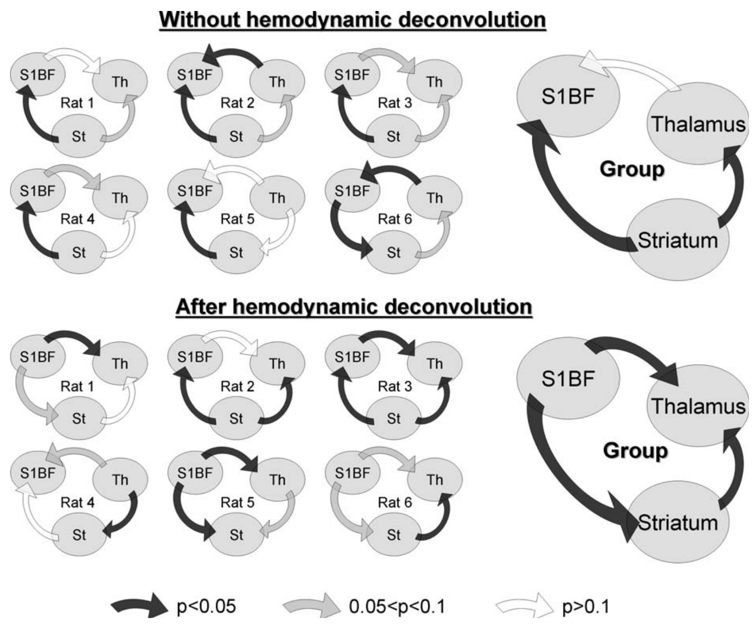
Friston et al., 2003, *NeuroImage*; Stephan et al., 2007, *NeuroImage*

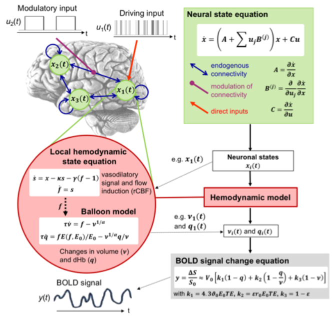
Hemodynamics are important



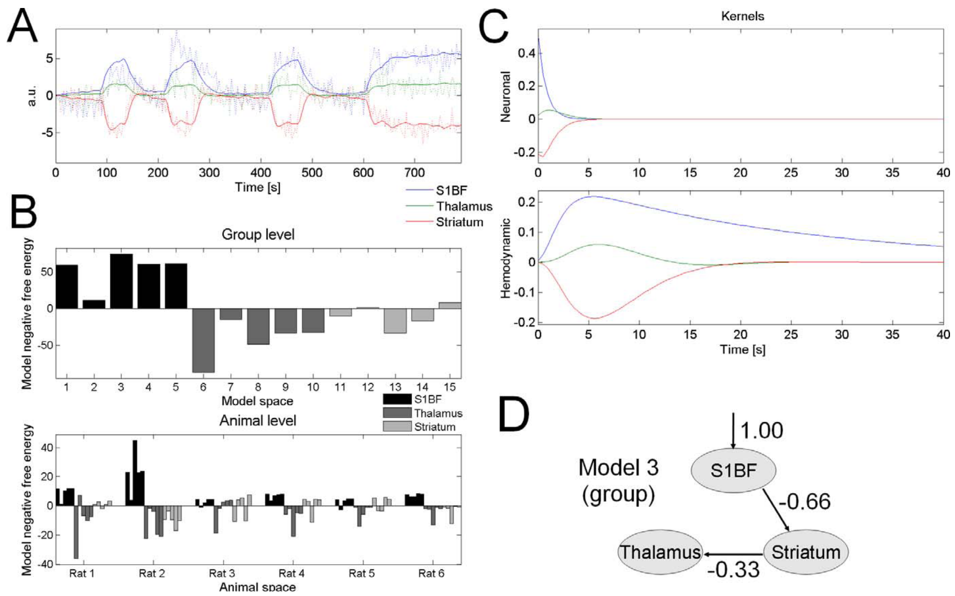
David et al., 2008, *PLoS Biol.*

Granger causality





DCM



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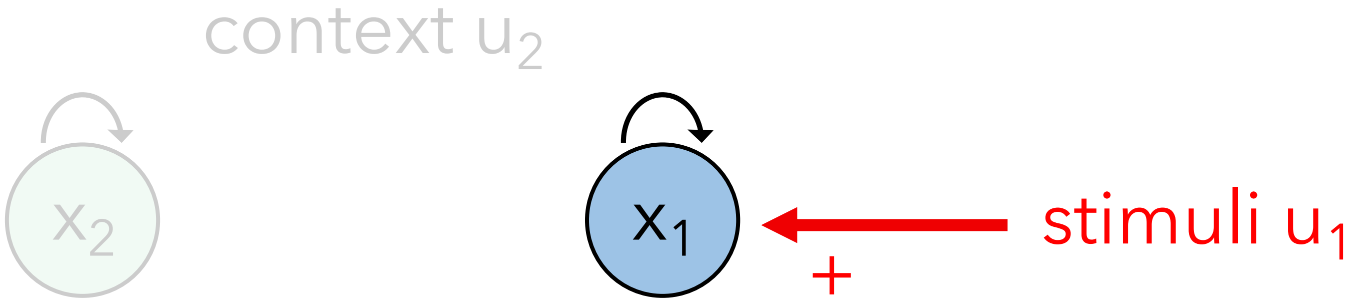
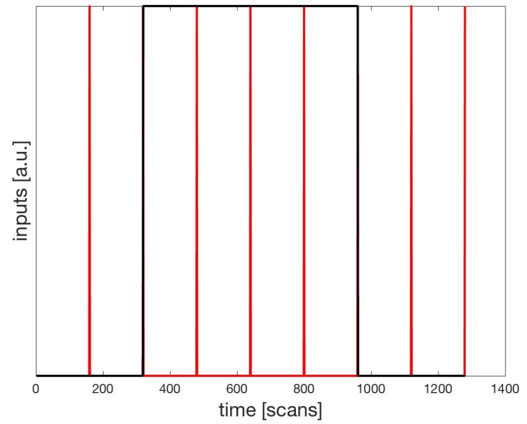
SIMULATIONS

What can DCM explain?

Example: single node



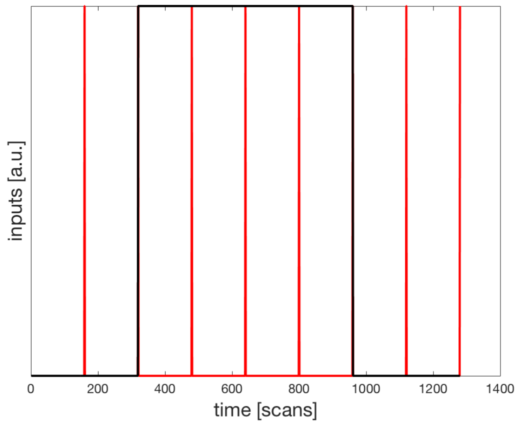


?

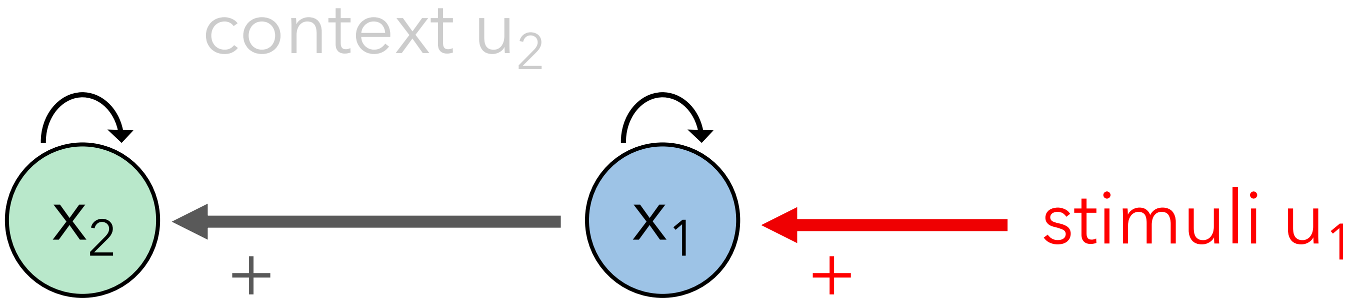
$$\frac{dx}{dt} = Ax + u_2 B^{(2)} x + C u_1$$

$$\begin{bmatrix} \dot{x}_1 \\ \dot{x}_2 \end{bmatrix} = \begin{bmatrix} a_{11} & 0 \\ 0 & a_{22} \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + u_2 \begin{bmatrix} 0 & 0 \\ 0 & 0 \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + \begin{bmatrix} c_{11} & 0 \\ 0 & 0 \end{bmatrix} \cdot \begin{bmatrix} u_1 \\ u_2 \end{bmatrix}$$

What can DCM explain?



Example: two connected nodes

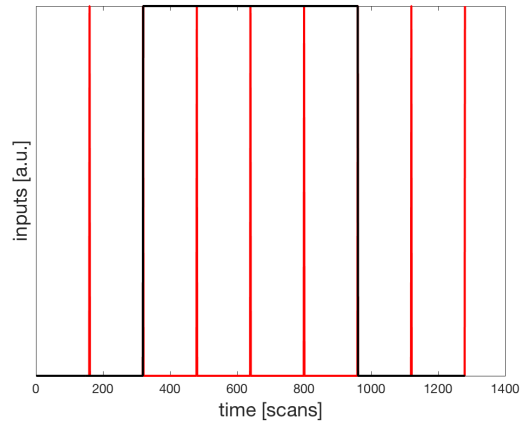


?

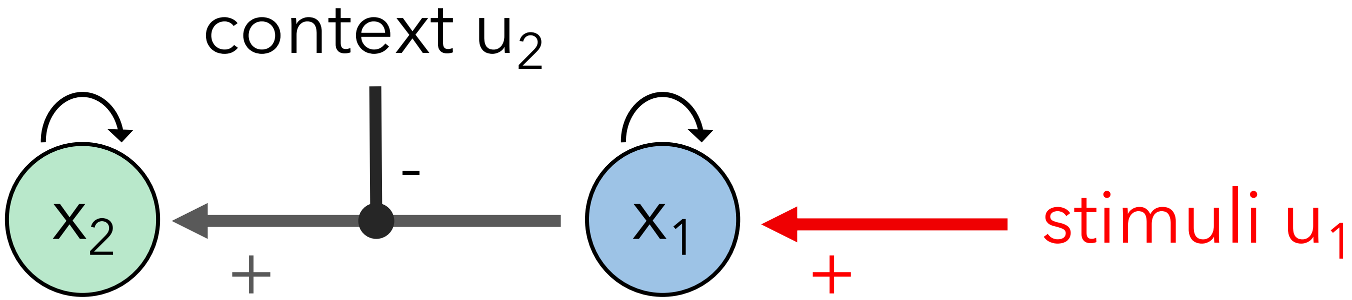
$$\frac{dx}{dt} = Ax + u_2 B^{(2)} x + C u_1$$

$$\begin{bmatrix} \dot{x}_1 \\ \dot{x}_2 \end{bmatrix} = \begin{bmatrix} a_{11} & 0 \\ a_{21} & a_{22} \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + u_2 \begin{bmatrix} 0 & 0 \\ 0 & 0 \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + \begin{bmatrix} c_{11} & 0 \\ 0 & 0 \end{bmatrix} \cdot \begin{bmatrix} u_1 \\ u_2 \end{bmatrix}$$

WHAT CAN DCM EXPLAIN?



Example: modulation of connection

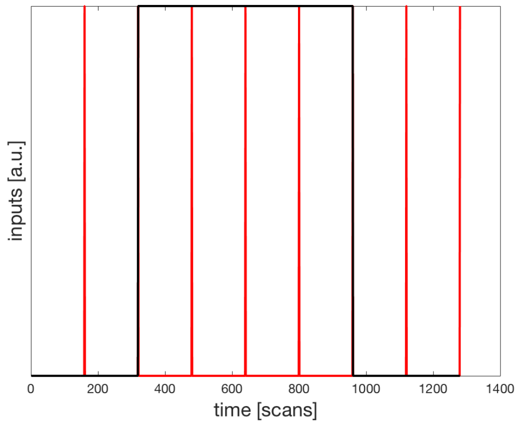


?

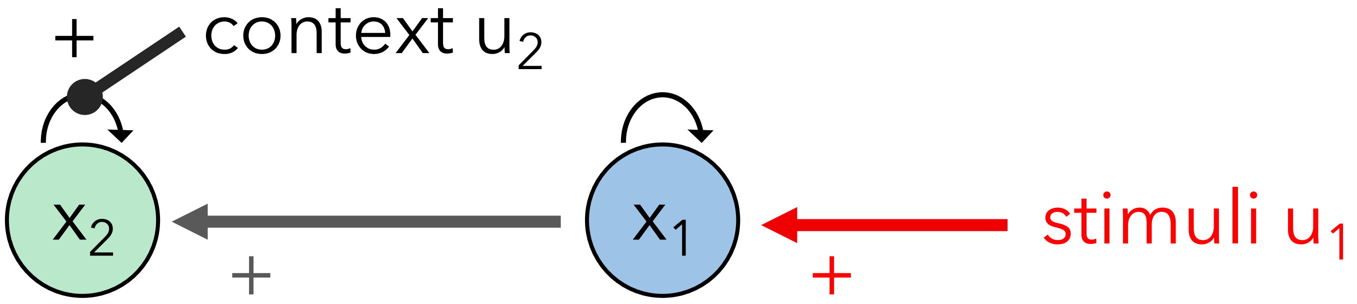
$$\frac{dx}{dt} = Ax + u_2 B^{(2)} x + C u_1$$

$$\begin{bmatrix} \dot{x}_1 \\ \dot{x}_2 \end{bmatrix} = \begin{bmatrix} a_{11} & 0 \\ a_{21} & a_{22} \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + u_2 \begin{bmatrix} 0 & 0 \\ b_{21}^{(2)} & 0 \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + \begin{bmatrix} c_{11} & 0 \\ 0 & 0 \end{bmatrix} \cdot \begin{bmatrix} u_1 \\ u_2 \end{bmatrix}$$

What can DCM explain?



Example: modulation of inhibitory self-connection



?

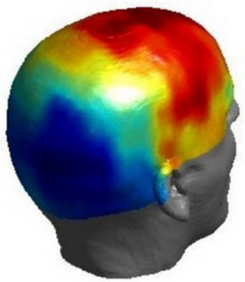
$$\frac{dx}{dt} = Ax + u_2 B^{(2)} x + C u_1$$

$$\begin{bmatrix} \dot{x}_1 \\ \dot{x}_2 \end{bmatrix} = \begin{bmatrix} a_{11} & 0 \\ a_{21} & a_{22} \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + u_2 \begin{bmatrix} 0 & 0 \\ 0 & b_{22}^{(2)} \end{bmatrix} \cdot \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} + \begin{bmatrix} c_{11} & 0 \\ 0 & 0 \end{bmatrix} \cdot \begin{bmatrix} u_1 \\ u_2 \end{bmatrix}$$

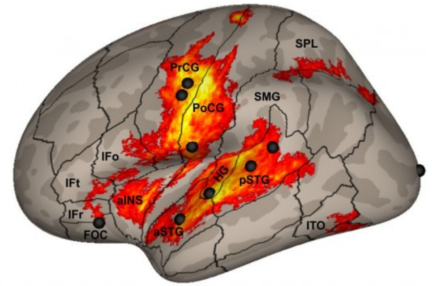
MODEL INVERSION / INFERENCE

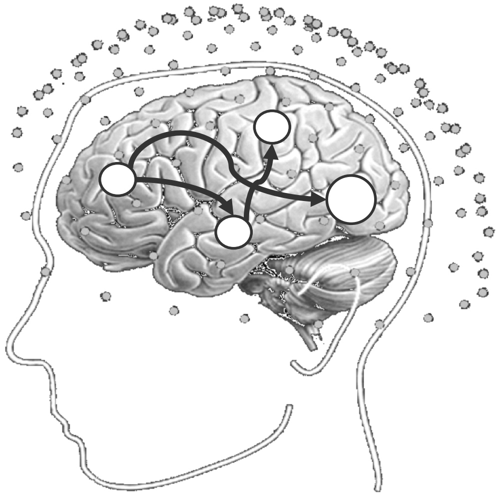
Dynamic causal modeling

EEG, MEG



fMRT





Forward model:
Predicting measured activity

$$p(y|\vartheta,m)$$

likelihood

Model inversion:
Estimating neuronal mechanisms

$$p(\vartheta|y,m)$$

posterior

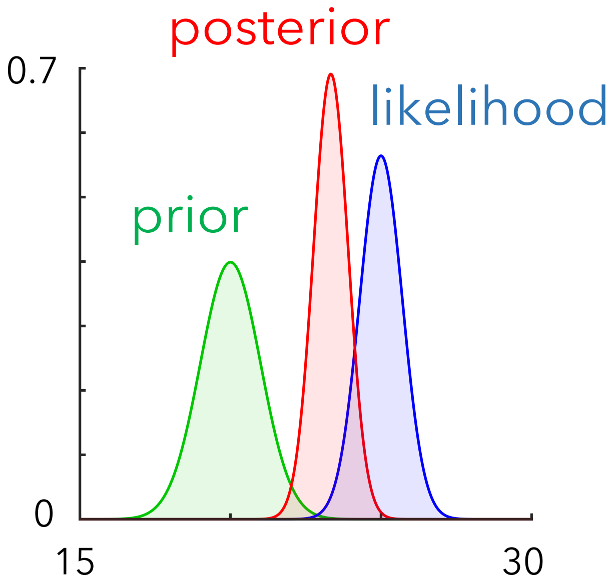
Friston et al., 2003, *NeuroImage*; David et al., 2006, *NeuroImage*

BAYES THEOREM

Reverend Thomas Bayes
(1702-1761)

Bayes theorem gives a recipe for evaluating the posterior density by combining new data (likelihood) and prior knowledge

$$\underbrace{p(\theta|y,m)}_{\text{posterior}} = \frac{\overbrace{p(y|\theta,m)}^{\text{likelihood}}\overbrace{p(\theta|m)}^{\text{prior}}}{\underbrace{p(y|m)}_{\text{model evidence}}}$$

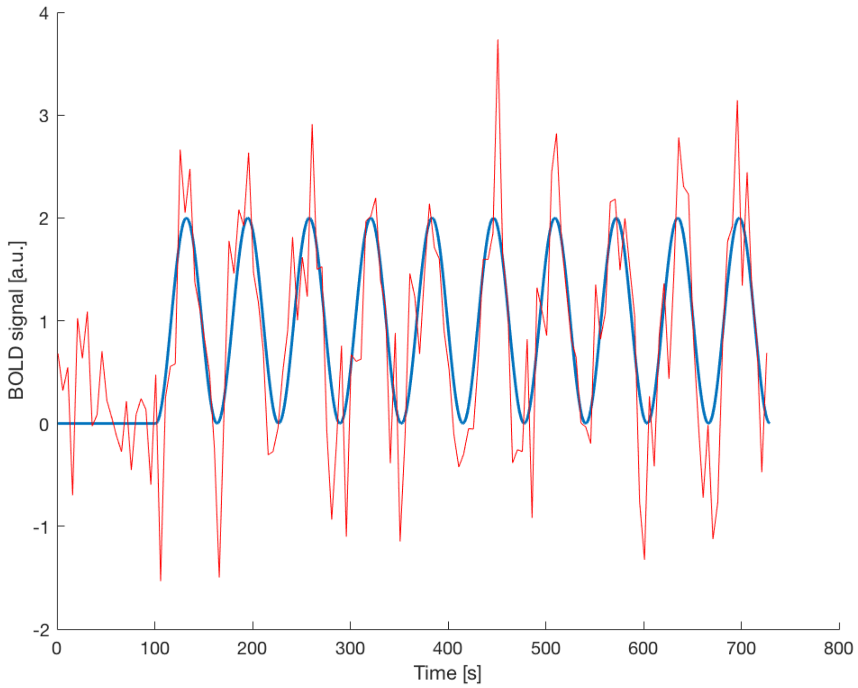


The posterior probability of the parameters is an optimal combination of our prior knowledge and the new data that we have acquired

Likelihood function

Assume data is normally distributed around the prediction from the dynamical model (Gaussian noise):

$$p(y(t)|\theta, m) = \mathcal{N}\left(y(t); g\left(\theta^n, \theta^h, u\right), \theta^\sigma\right)$$



LIKELIHOOD FUNCTION

Assume data is normally distributed around the prediction from the dynamical model (Gaussian noise):

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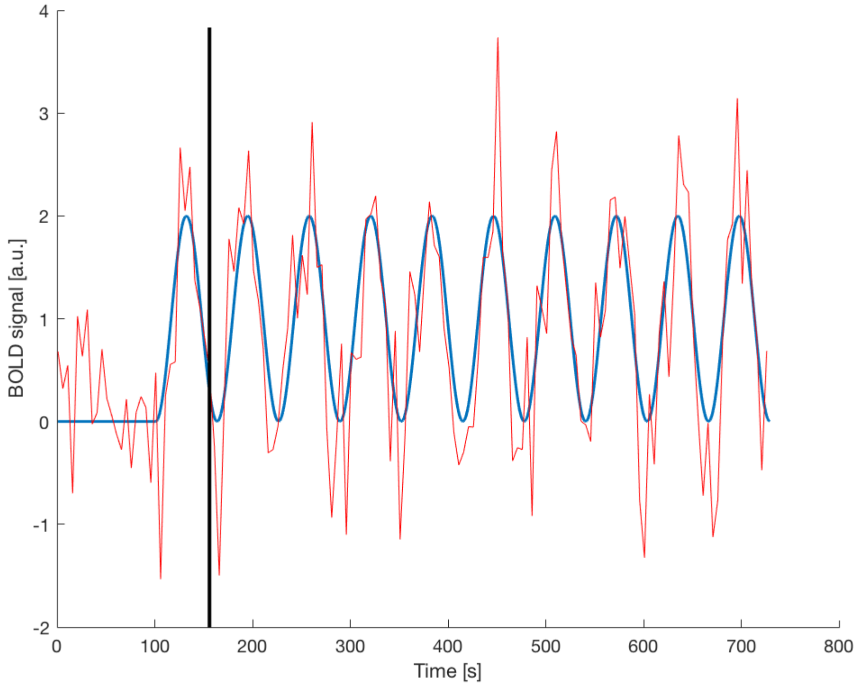

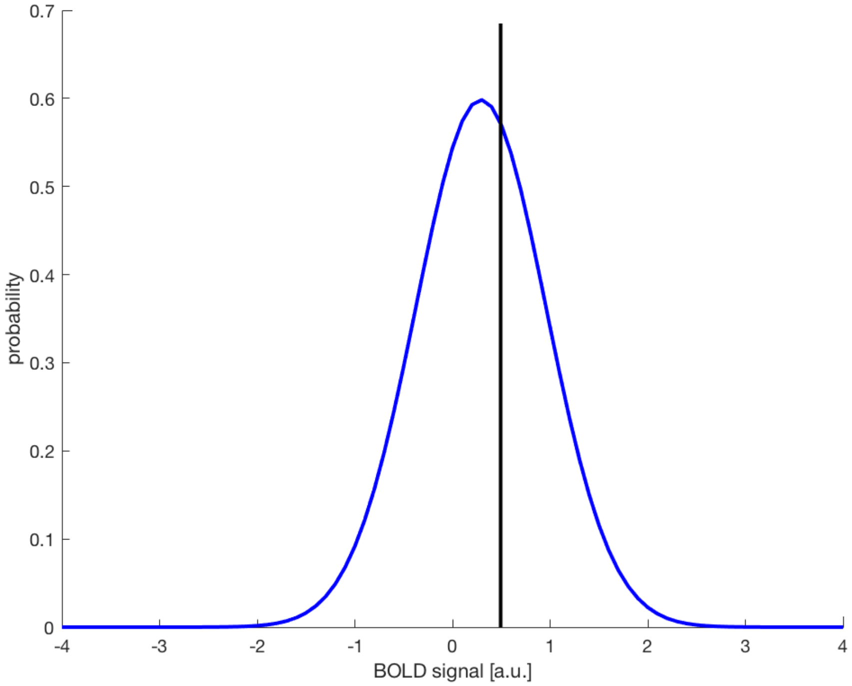

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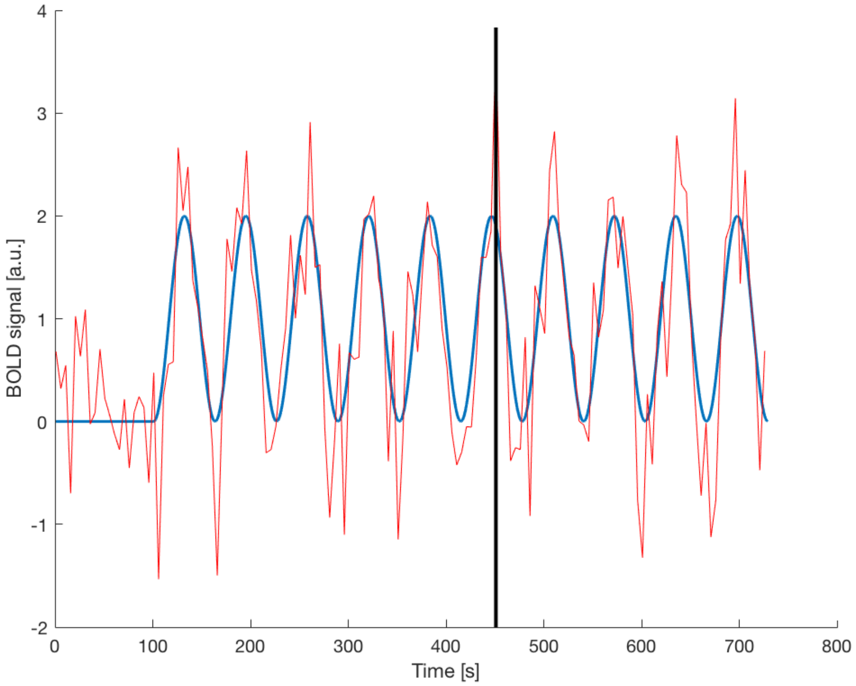
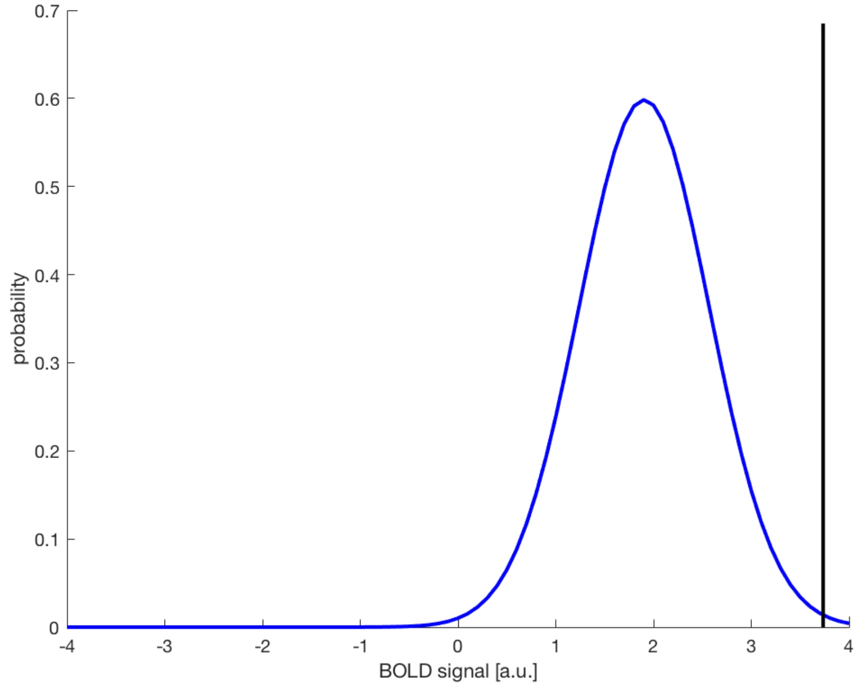
Assume data is normally distributed around the prediction from the dynamical model (Gaussian noise):

$$p(y(t)|\theta, m) = \mathcal{N}\left(y(t); g\left(\theta^n, \theta^h, u\right), \theta^\sigma\right)$$





PRIORS

Bayes theorem gives a recipe for evaluating the posterior density by combining new data (likelihood) and prior knowledge

$$p(\theta|y,m) = \frac{p(y|\theta,m)\overbrace{p(\theta|m)}^{\text{prior}}}{p(y|m)}$$

Neuronal parameters:

- self-connections: principled (to ensure that the system is stable)
- other parameters (between–region connections, modulation, inputs): shrinkage priors

Hemodynamic parameters:

- empirical

PRIORS

Types of priors:

- Explicit priors on *model parameters* (e.g., connection strengths)
- Implicit priors on *model functional form* (e.g., system dynamics)
- Choice of "interesting" *data features* (e.g., regional time-series vs. ICA analysis)

Role of priors (on model parameters):

- Resolving the *ill-posedness* of the inverse problem
- Avoiding *overfitting* (cf. generalization error)

Impact of priors:

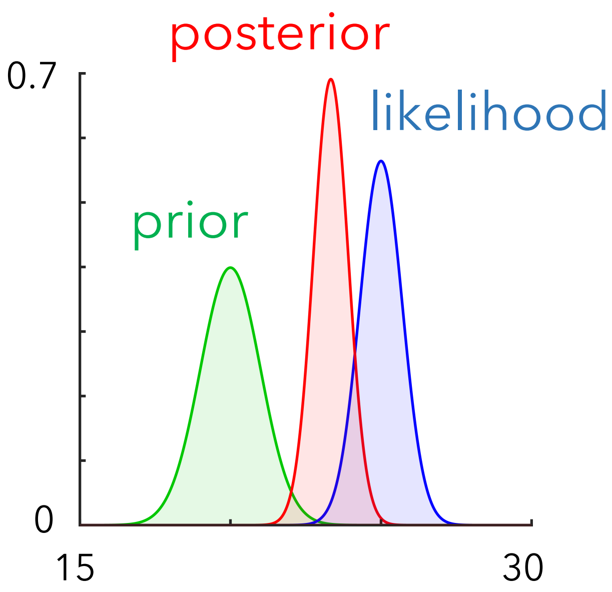
- On posterior distributions over parameters (cf. "shrinkage to the mean" effect)
- On model evidence (cf. "Occam's razor")
- On free-energy landscape (cf. Laplace approximation)

BAYES THEOREM

Reverend Thomas Bayes
(1702-1761)

Bayes theorem gives a recipe for evaluating the posterior density by combining new data (likelihood) and prior knowledge

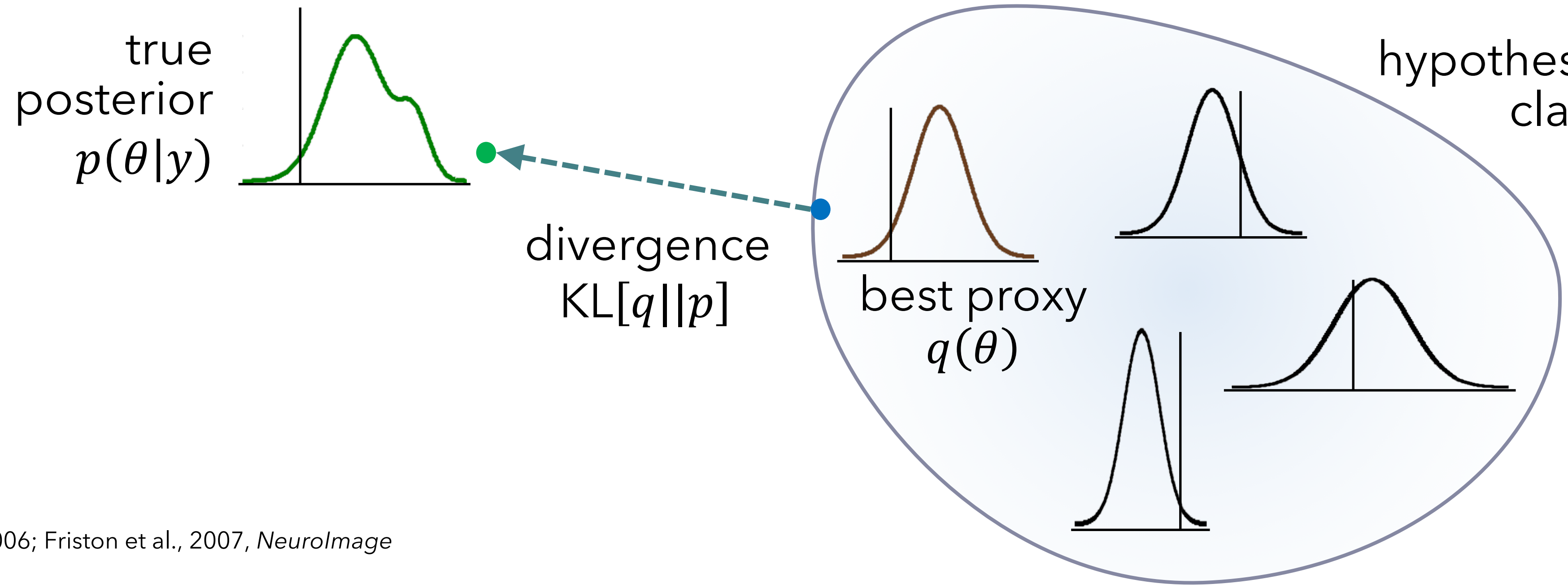
$$\underbrace{p(\theta|y,m)}_{\text{posterior}} = \frac{\overbrace{p(y|\theta,m)}^{\text{likelihood}}\overbrace{p(\theta|m)}^{\text{prior}}}{\underbrace{p(y|m)}_{\text{model evidence}}}$$



The posterior probability of the parameters is an optimal combination of our prior knowledge and the new data that we have acquired

Variational Bayes (VB)

Idea: find an approximate density $q(\theta)$ that is maximally similar to the true posterior $p(\theta|y)$. This is often done by assuming a particular form for q (fixed form VB) and then optimizing its sufficient statistics.



Bishop, 2006; Friston et al., 2007, *NeuroImage*

Negative free energy

$$\ln p(y) = \underbrace{\mathrm{KL}[q||p]}_{\substack{\textbf{divergence} \geq 0 \\ \text{(unknown)}}} + \underbrace{F(q,y)}_{\substack{\textbf{neg. free energy} \\ \text{(easy to evaluate} \\ \text{for a given } q)}}$$

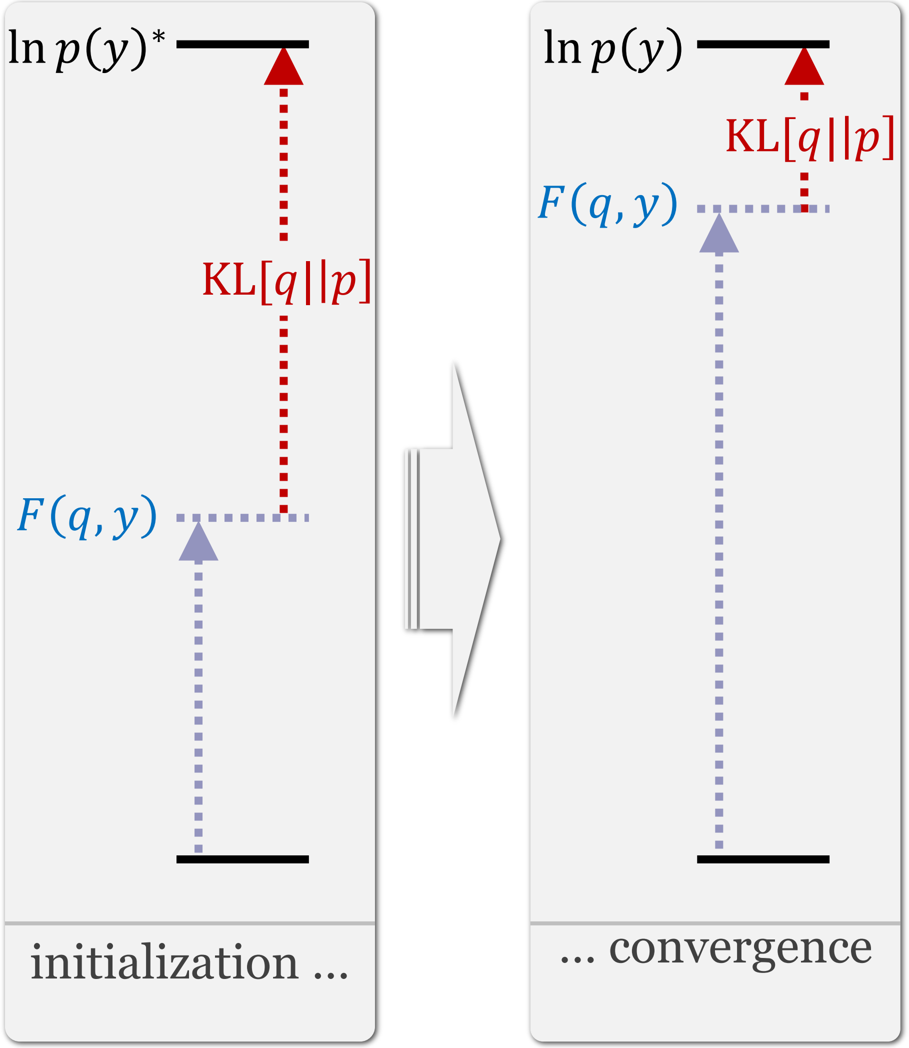
$F(q,y)$ is a functional with respect to the approximate posterior $q(\theta)$.

Maximizing $F(q,y)$ is equivalent to:

- minimizing $KL[q||p]$
- tightening $F(q,y)$ as a lower bound on the log model evidence

When $F(q,y)$ is maximized, $q(\theta)$ is our best estimate of the true posterior.

Bishop, 2006; Friston et al., 2007, *NeuroImage*



NEGATIVE FREE ENERGY – A CLOSER LOOK

The **negative free energy** represents a trade-off between the accuracy and complexity of a model:

$$F = \langle \log p(y|\theta, m) \rangle_q - KL[q(\theta) \| p(\theta|m)]$$

accuracy
(expected log likelihood)

complexity
(KL divergence between approximate posterior and prior)

In contrast to "simple" criteria (e.g., AIC & BIC), the complexity term of the negative free energy accounts for parameter interdependencies and is a much richer description:

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

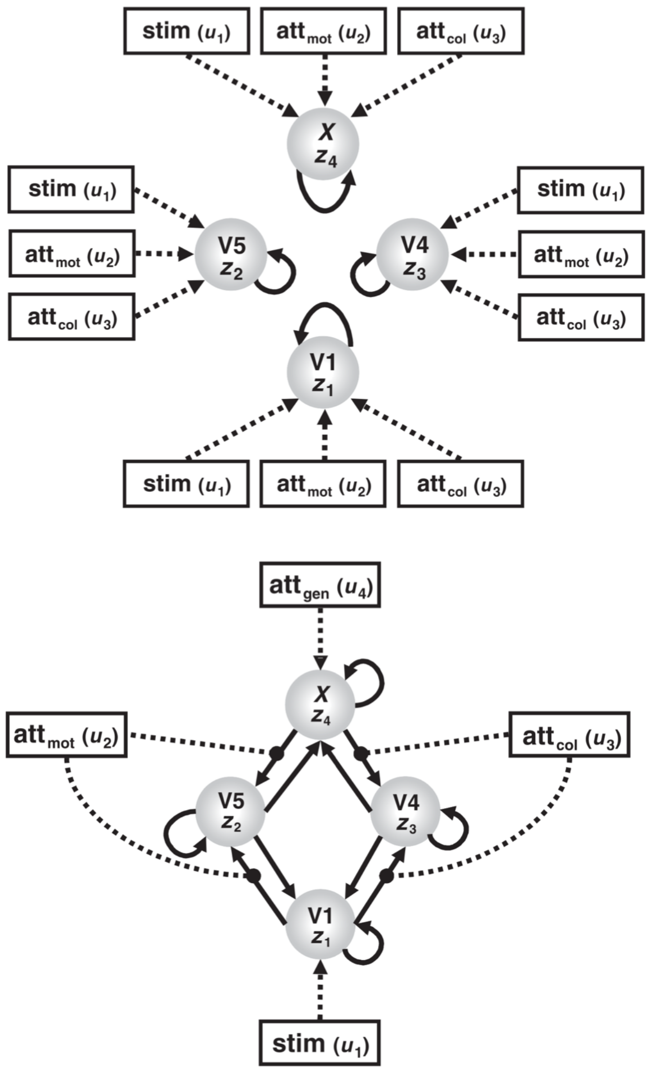
Bishop, 2006; Friston et al., 2007, *NeuroImage*

NOTE: GLM VS. DCM

DCM tries to model the same phenomena (i.e., local BOLD responses) as a GLM, just in a different way (via connectivity and its modulations).

No activation detected by a GLM → no motivation to include this region in a deterministic DCM.

However, a stochastic DCM (that incorporates a noise term in the neuronal state equation and can thus accounts for endogenous fluctuations) could be applied despite the absence of a local activation.



Stephan, 2004, *J. Anat.*

APPLICATIONS

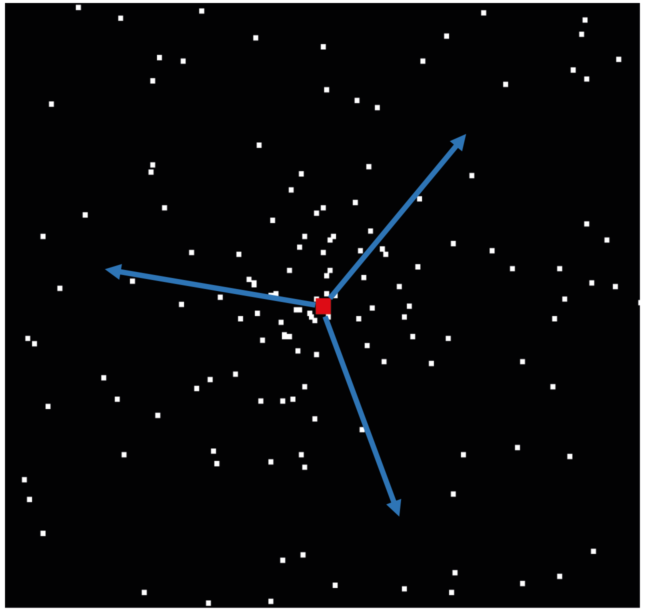
Simple example: Attention to motion

Stimuli: radially moving dots were presented.

Pre-scanning: 5x30s trials with 5 speed changes. Subjects were asked to detect the change in radial velocity.

Scanning: No actual speed changes. Conditions:

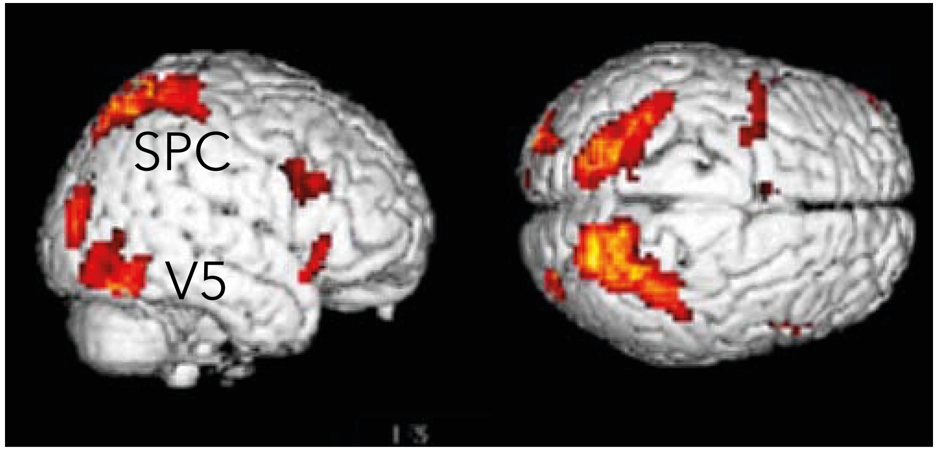
- F: fixation
- S: static dots
- M: moving dots
- A: attend moving dots



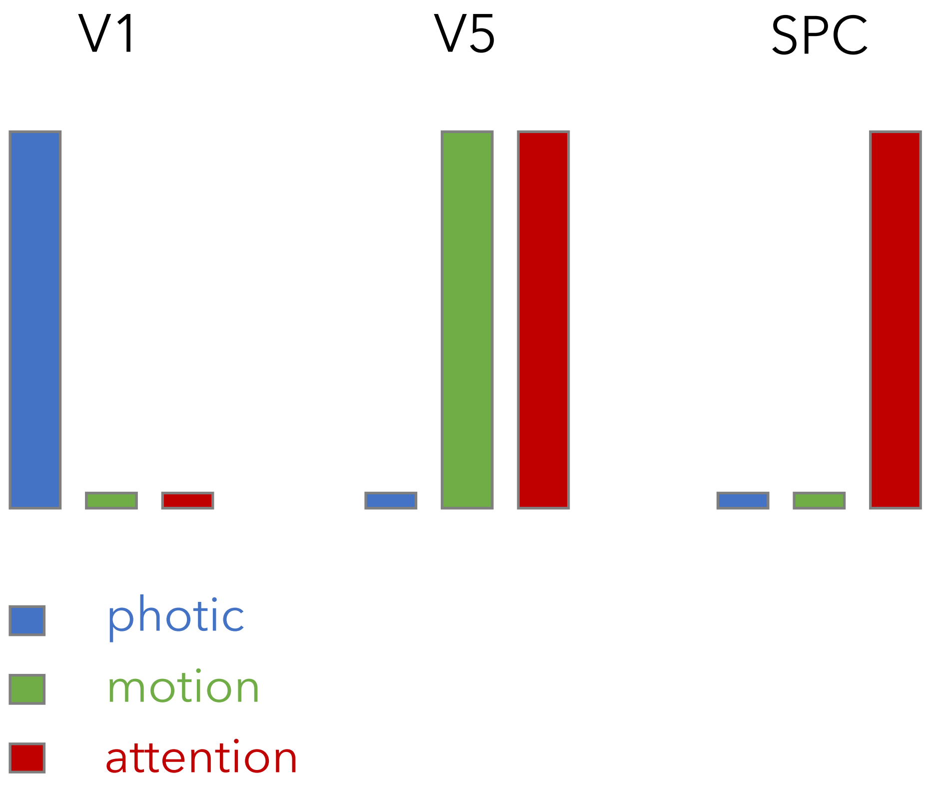
Büchel and Friston, 1997, *Cerebral Cortex*; Friston et al., 2003, *NeuroImage*

SIMPLE EXAMPLE: ATTENTION TO MOTION

Single-subject results: BOLD activation patterns

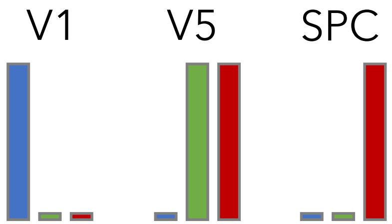


Linear contrast: attention > no attention

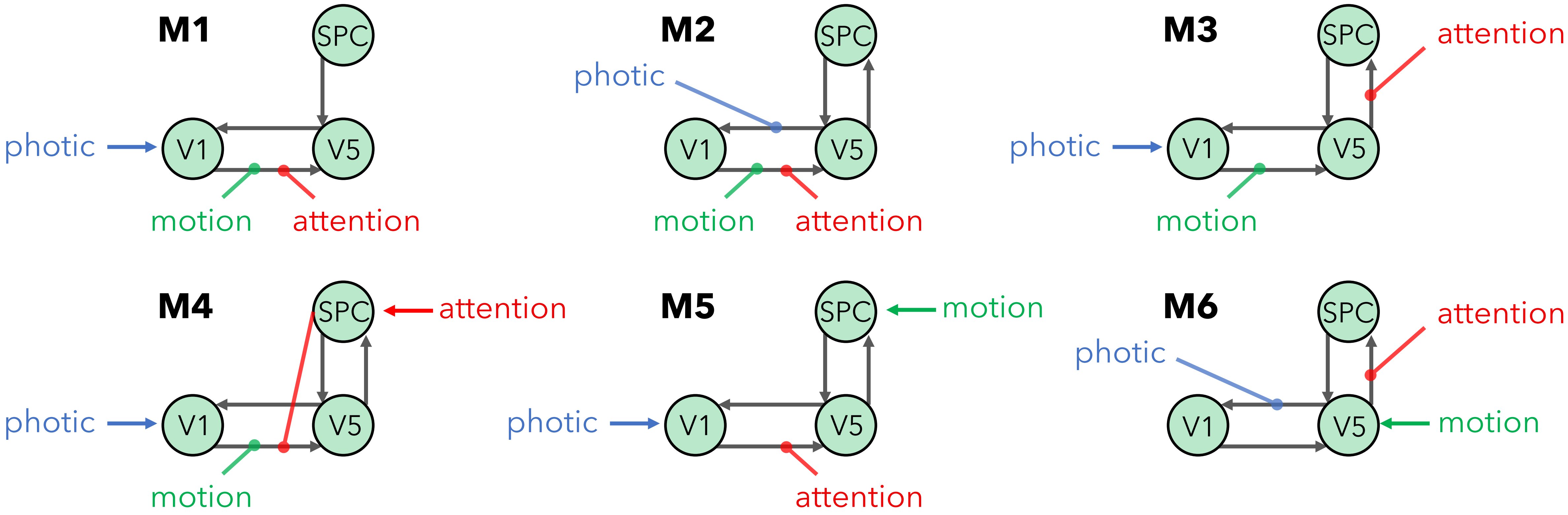


Büchel and Friston, 1997, *Cerebral Cortex*; Friston et al., 2003, *NeuroImage*

Simple example: Attention to motion



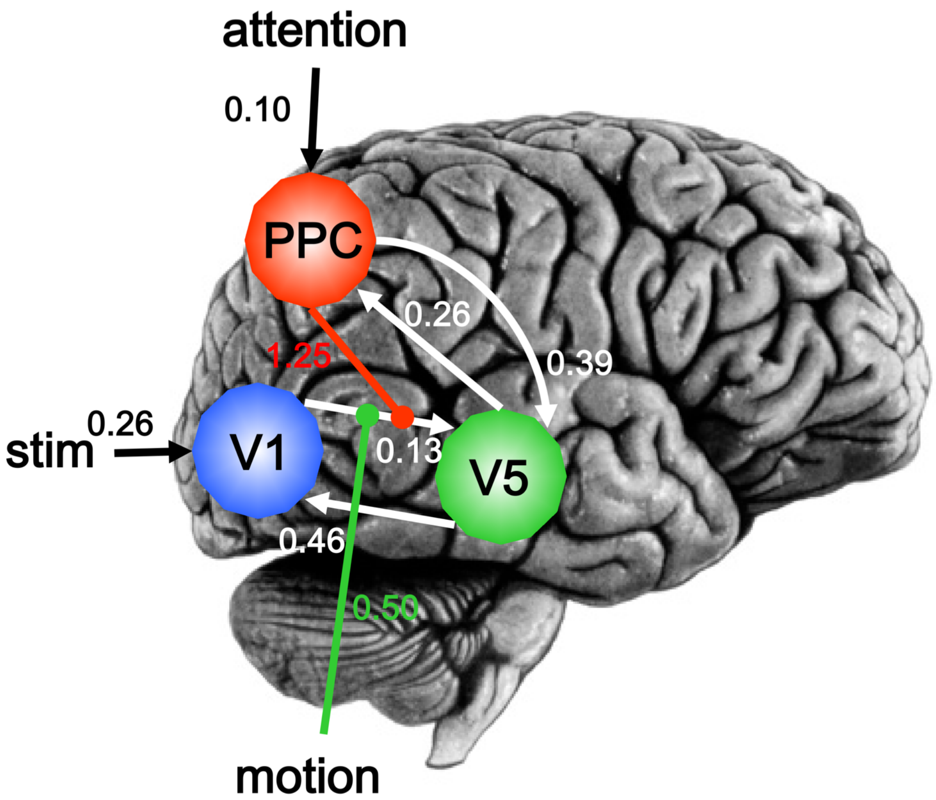
Model space definition – which models can explain the data (Quiz)?

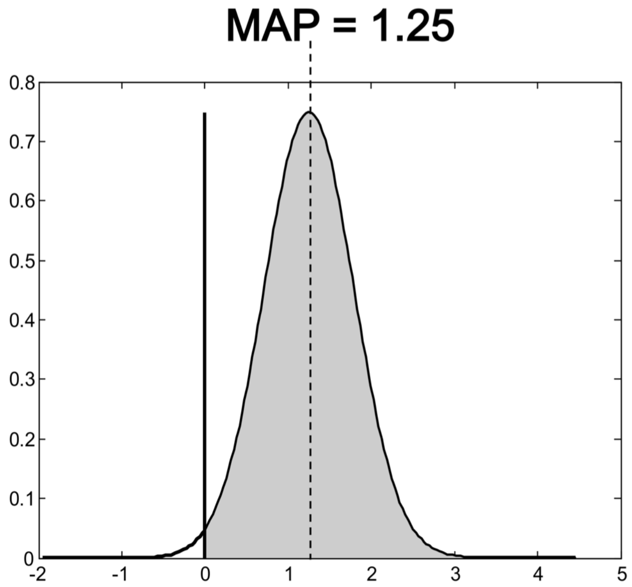


Büchel and Friston, 1997, *Cerebral Cortex*; Friston et al., 2003, *NeuroImage*

Simple example: Attention to motion

Single-subject results: DCM effective connectivity





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

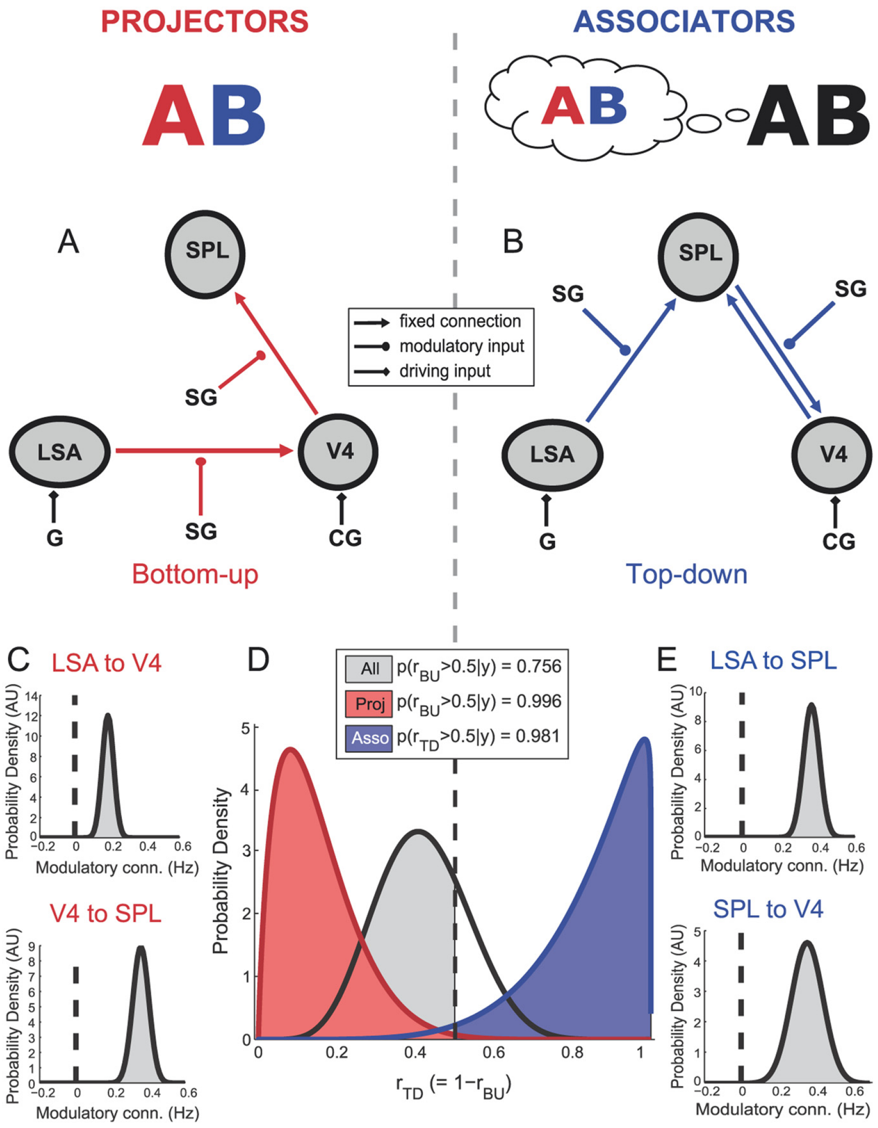
Büchel and Friston, 1997, *Cerebral Cortex*; Friston et al., 2003, *NeuroImage*; Stephan et al., 2008, *NeuroImage*

Applications of BMS and BMA

Individuals with different forms of color-grapheme synesthesia were tested and effective connectivity in the relevant neural circuits was assessed using DCM.

Bayesian model selection (BMS) as a formal approach to differential diagnosis in clinical applications

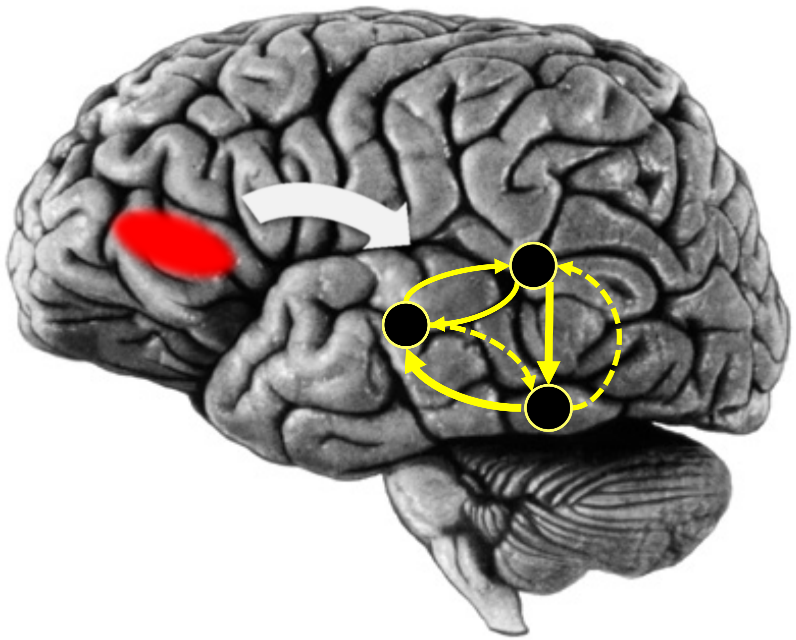
(Note: Here, different forms of synesthesia were tested. This is not a clinical condition, but simply a specific cognitive trait)



Van Leeuwen et al., 2011, *J. Neurosci.*

Generative embedding: Aphasia

Dissociating aphasic patients (N=11) and healthy controls (N=26)



Schofield et al., 2012, *J. Neurosci.*; Brodersen et al., 2011, *PLoS Comp. Biol.*

Generative embedding: Aphasia

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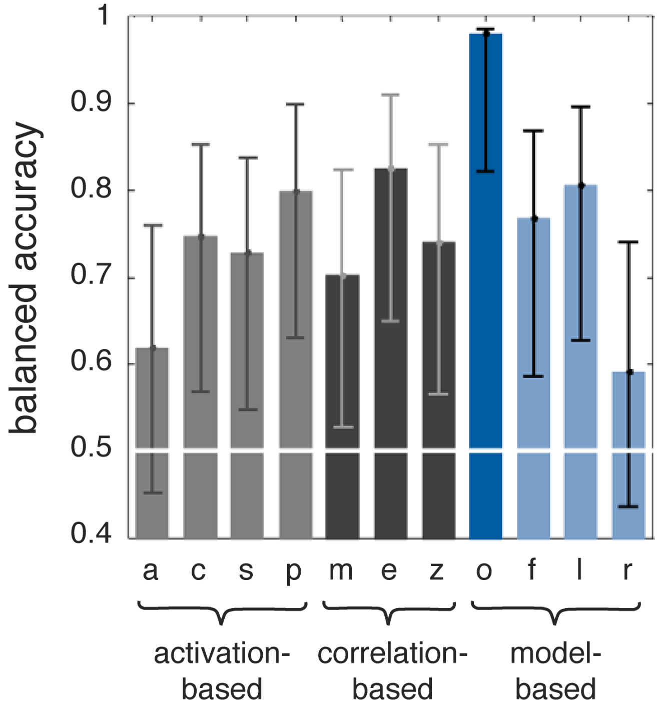

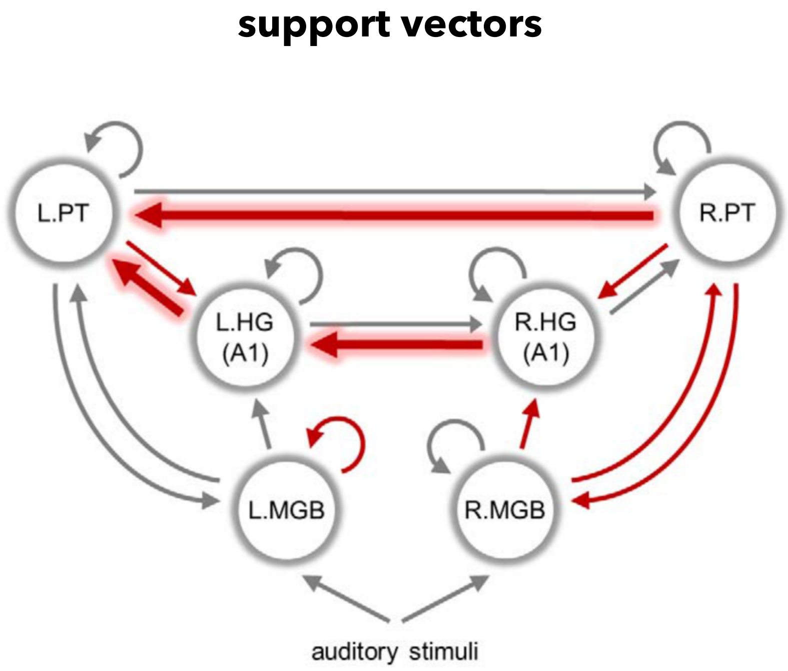

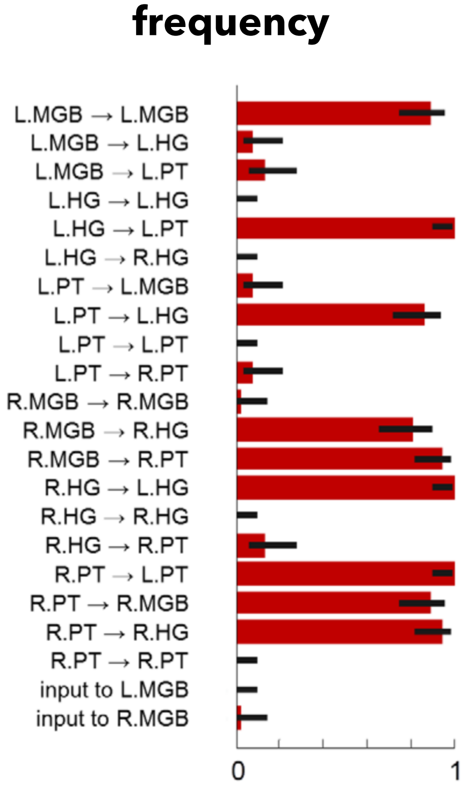

Schofield et al., 2012, *J. Neurosci.*; Brodersen et al., 2011, *PLoS Comp. Biol.*

GENERATIVE EMBEDDING: APHASIA

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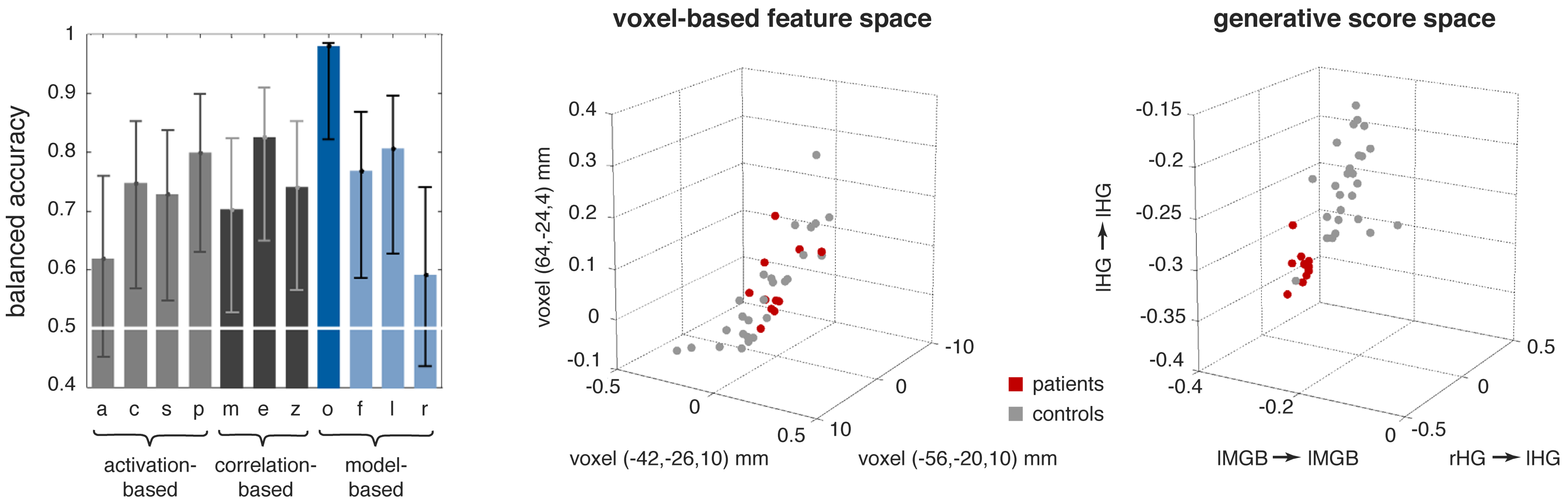
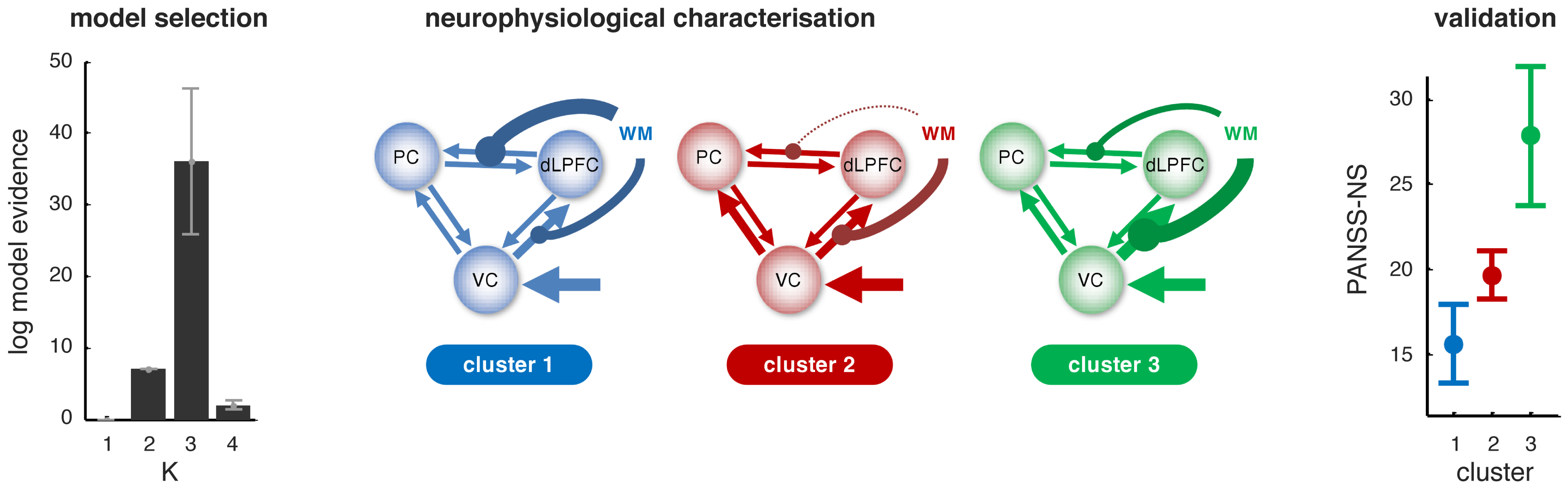


Schofield et al., 2012, *J. Neurosci.*; Brodersen et al., 2011, *PLoS Comp. Biol.*

Generative embedding: Schizophrenia

Detecting subgroups of patients in schizophrenia (N=41)

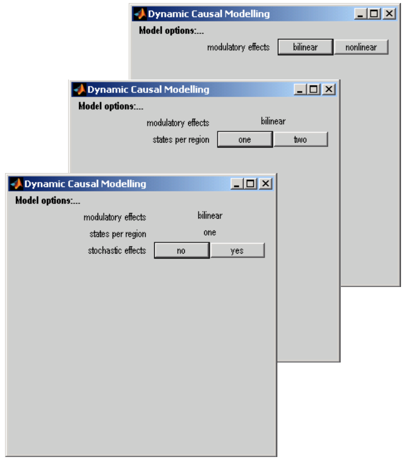


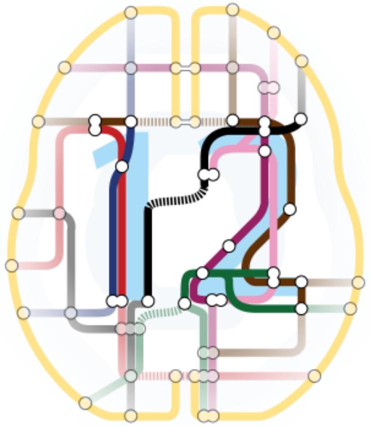
Deserno et al., 2012, *J. Neurosci.*; Brodersen et al., 2014, *NeuroImage: Clinical*

Evolution of DCM

Different variants and extensions **within** SPM

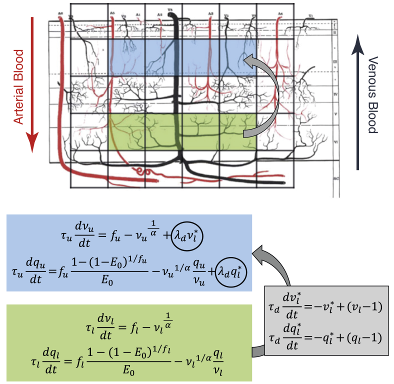
- bilinear vs. nonlinear
- single-state vs. two-state (per region)
- deterministic vs. stochastic
- time-series vs. cross-spectra

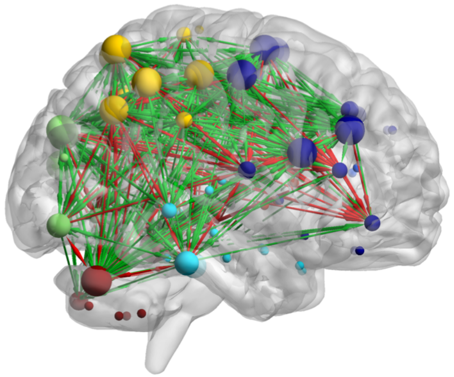




Different variants and extensions **outside** SPM

- DCM for layered BOLD
- Global optimization schemes for model inversion
- Hierarchical unsupervised generative embedding
- regression DCM (rDCM)



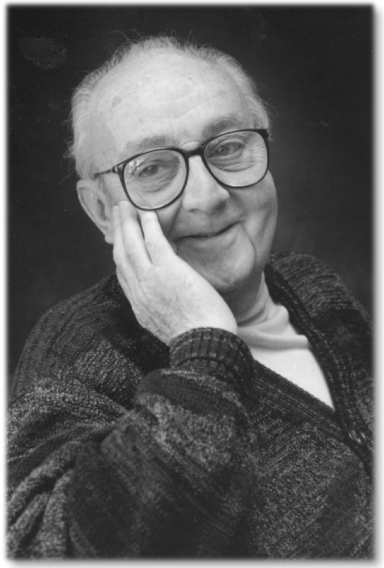


Friston et al., 2003, *NeuroImage*; Stephan et al., 2009, *NeuroImage*; Marreiros et al., 2008, *NeuroImage*; Daunizeau et al., 2009, *NeuroImage*; Friston et al., 2014, *NeuroImage*; Havlicek et al., 2017, *NeuroImage*; Heinzle et al., 2016, *NeuroImage*; Sengupta et al, 2015, *NeuroImage*; Lomakina et al., 2015, *NeuroImage*; Aponte et al., 2015, *J. Neurosci. Meth.*; Friston et al., 2016, *NeuroImage*; Raman et al., 2016, *J. Neurosci. Meth;* Frässle et al., 2017, 2018, *NeuroImage*

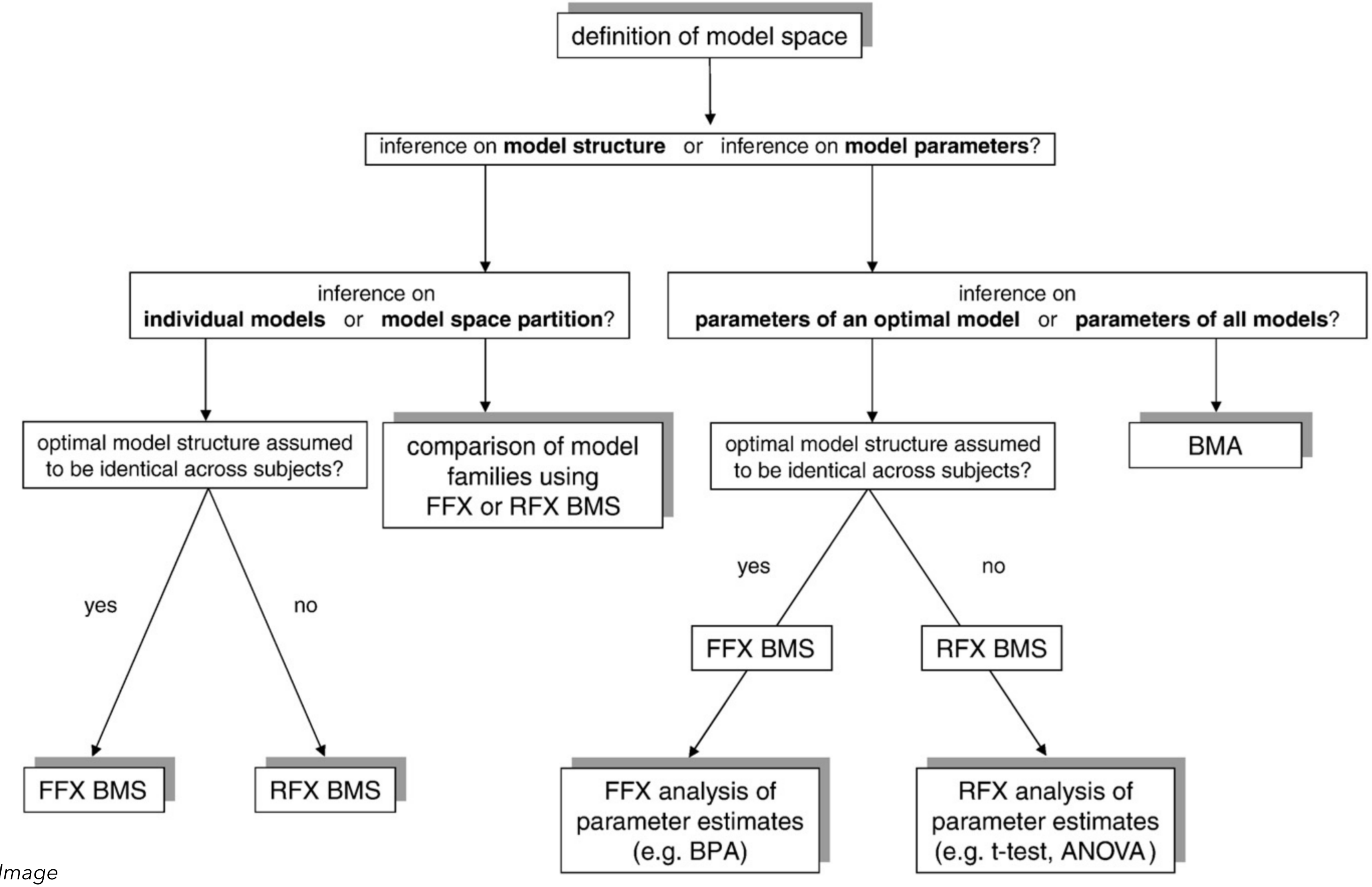
ALL MODELS ARE WRONG

BUT SOME ARE USEFUL

George Edward Pelham Box
(1919-2013)



SCHEMATIC OVERVIEW



Stephan et al., 2010, *NeuroImage*

Further Readings (1)

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Questions

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THANK YOU FOR YOUR ATTENTION !

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