Dynamic Causal Modelling for Steady State Responses

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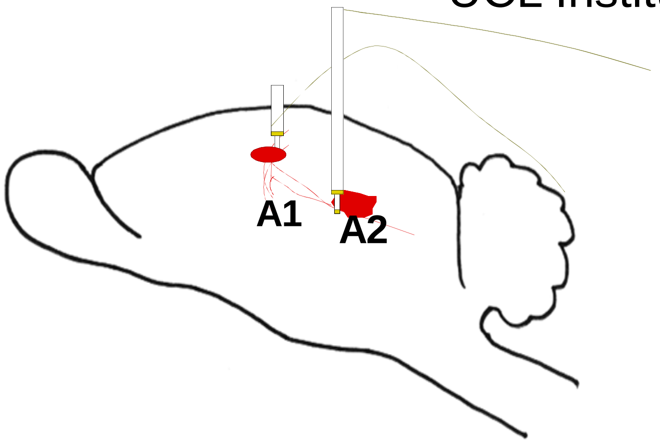
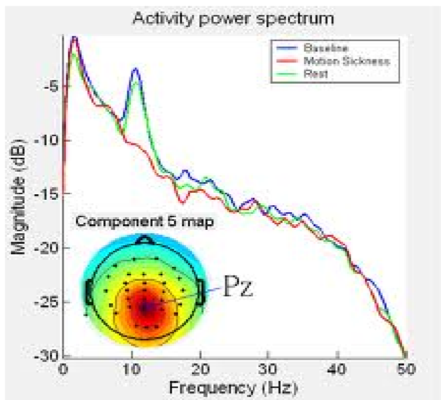
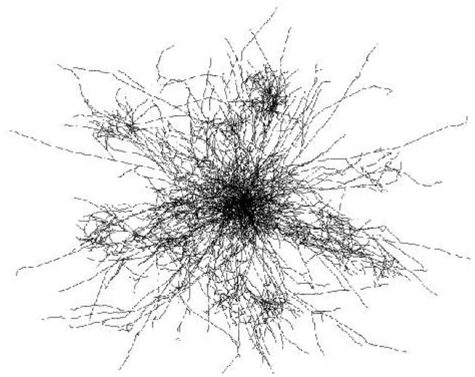




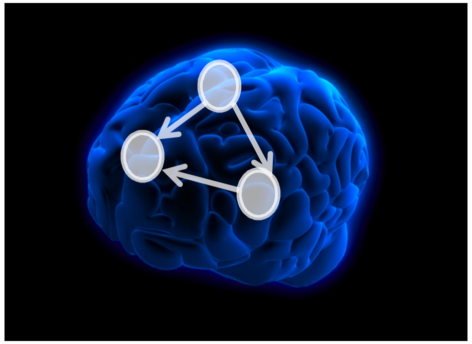


SPM Course London May 2012

Dynamic Causal Modelling for SSR

A framework which uses Bayesian techniques to fit differential equations to steady – state data. It allows for comparison between competing models of brain architecture and furnishes estimates for parameters that are not measured directly by exploiting electrophysiological data.

Although it is based on sophisticated models from computational neuroscience, its application is straightforward and does not require mathematical training.



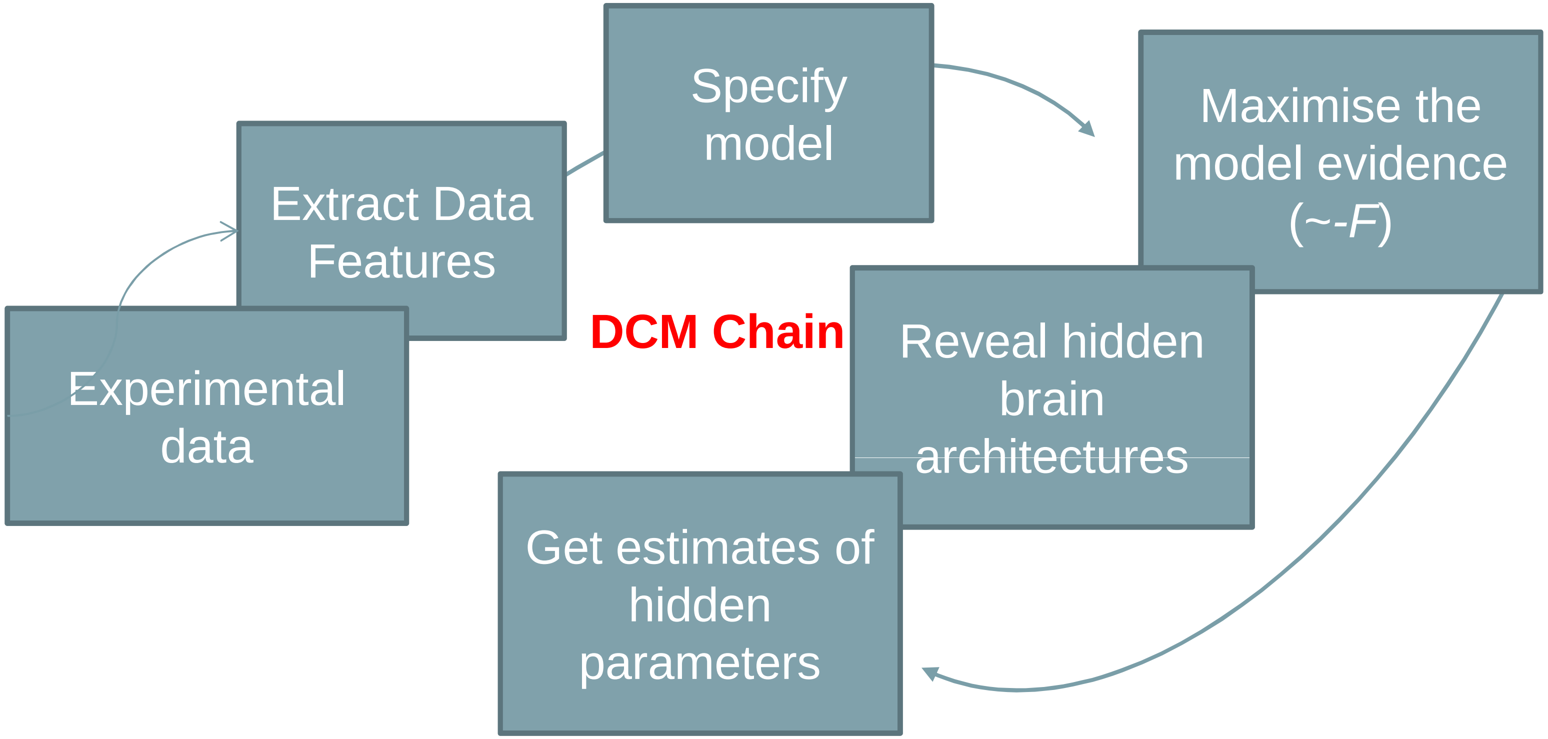
2. When should I use DCM for SSR?

-Brain activity retains similar statistical features (e.g.variance) and frequency content
across measurement period
-Cannot describe nonlinear coupling
between frequencies →next talk

Advantages:

➢ Summarize activity in a compact way -
no need to fit long time series (computationally expensive)
➢Describe brain function in terms of a
characteristic frequency associated with the task under study

3. Which steps do we take when using DCM for SSR?



4.Where has DCM for SSR been applied ?

- ❑DCM for SSR
  (Moran et al., Neuroimage, 2009)

- ❑ Anaesthesia:
  Anaesthetic Depth in Rodents
  (Moran et al., Plos One, 2011)
- ❑ Dopamine in working memory
  (Moran et al., Current Biol., 2011)
- ❑ Beta oscillations in PD
  (Moran et al., Plos CB, 2011)
  (Marreiros – yesterday's talk)
- ❑ Sleep and Coma
  (Boly et al., Science, 2011,
  J Neuro, 2012)
- ❑***Extension:***
  DCM for Neural Fields
  (Pinotsis et al., Neuroimage, 2011,2012)

Overview

1. Data Features

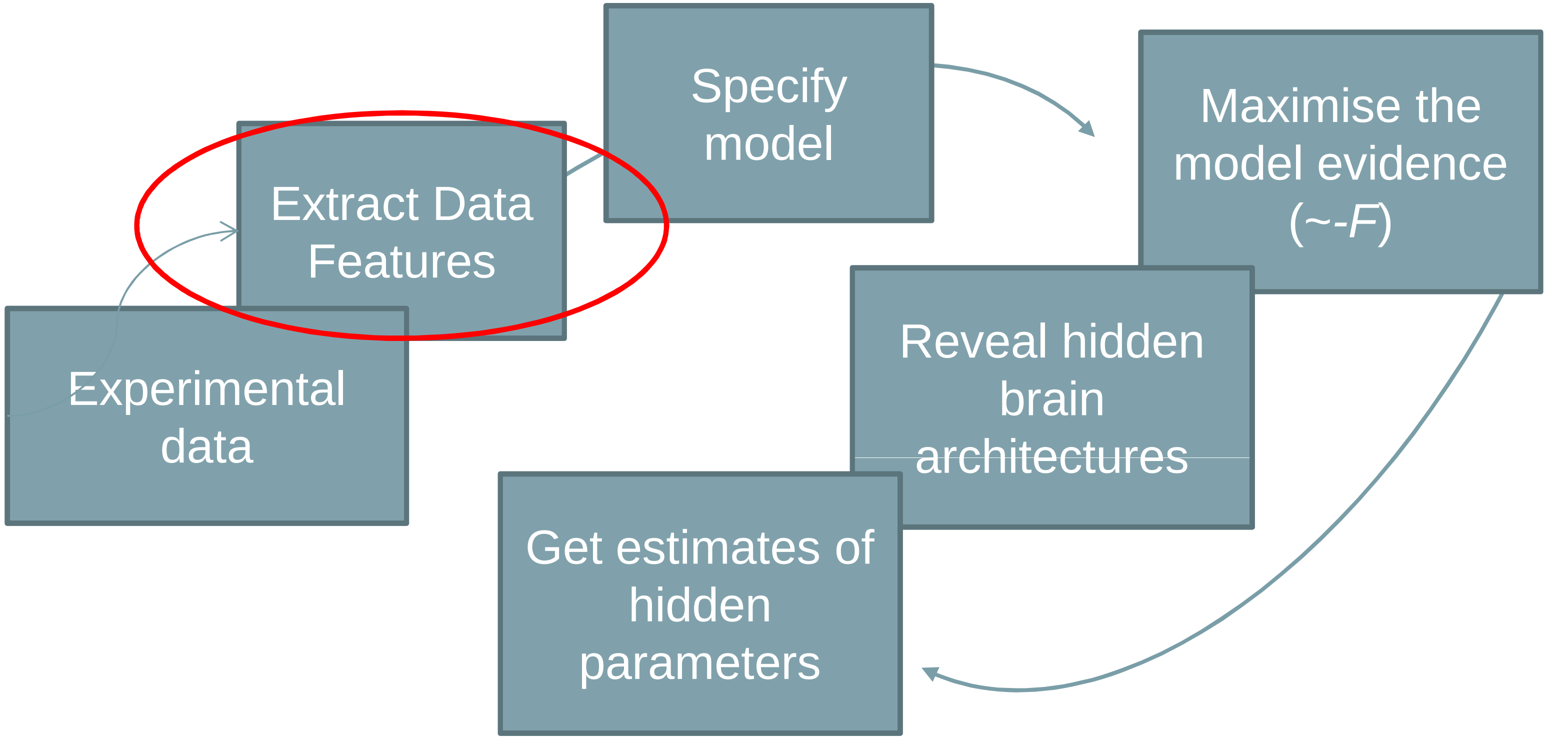
2. Generative Model

3. Bayesian Inversion: Parameter Estimates and Model Comparison

4. Example: Glutamate and GABA in Rodent Auditory Cortex

5. DCM for Current Source Density

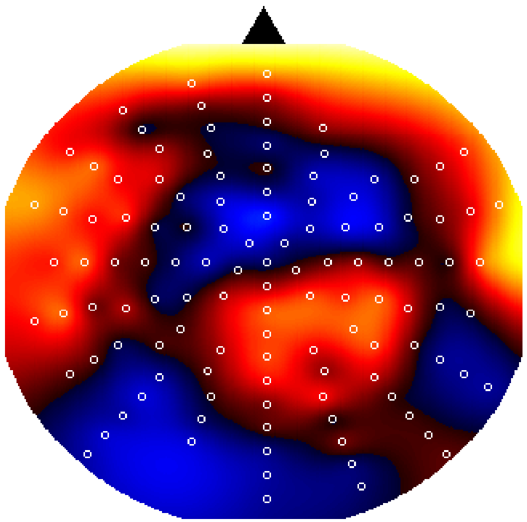
6. DCM for Neural Fields

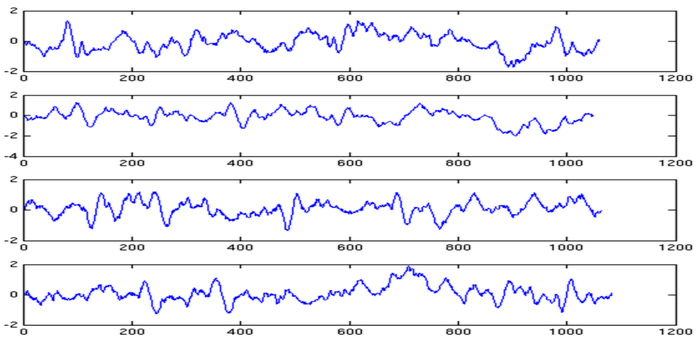


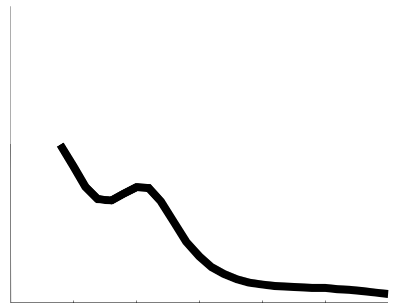
Specify model
Maximise the model evidence (~-*F*)
Extract Data Features
Experimental data
Reveal hidden brain architectures
Get estimates of hidden parameters

Cross Spectral Density

EEG - MEG – LFP Time Series

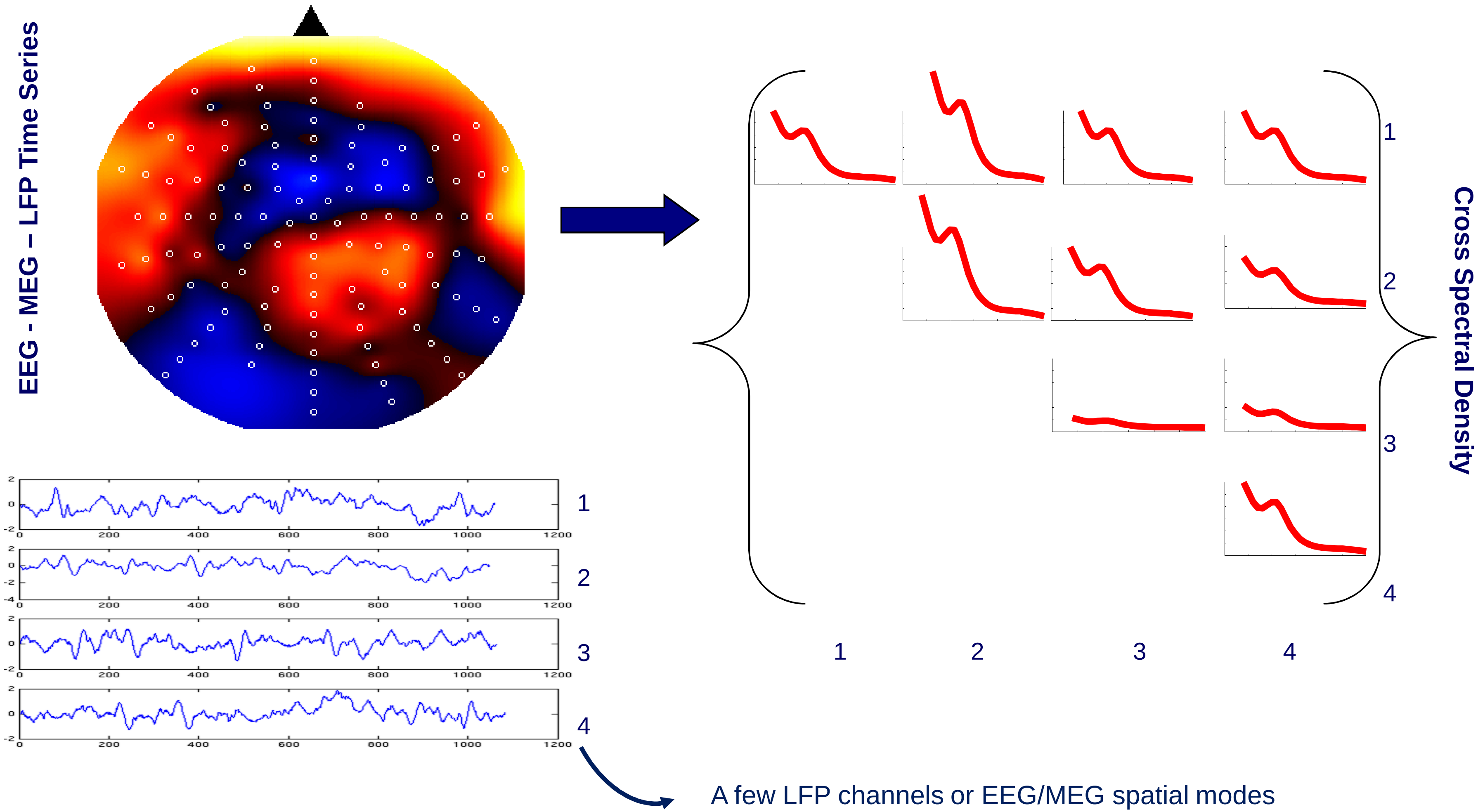






- Summarizes brain response in terms of power at each frequency

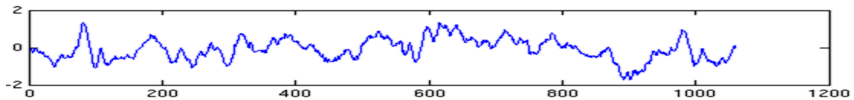
From Time Series to Cross Spectral Densities



From Time Series to Cross Spectral Densities

Vector Auto-regression *p*-order model:

Linear prediction formulas that attempt to predict an output *y[n]* of a system based on the previous outputs



$$y_n = \alpha_1 y_{n-1} + \alpha_2 y_{n-2} \ldots + \alpha_p y_{n-p} + e_n$$

Resulting in *a* matrices for *c* Channels

$$\{\alpha_{1\ldots.p} \in A(p) : \{c \times c\}$$

Cross Spectral Density for channels *i,j* at frequencies

$\omega = 2\pi f$

$$g(\omega)_{ij} = f(A(p))$$

$$H_{ij}(\omega) = \frac{1}{\alpha_1^{ij} e^{iw} + \alpha_2^{ij} e^{iw2} + \ldots\ldots + \alpha_p^{ij} e^{iwp}}$$

$$\begin{Bmatrix} g(\omega)_{11} & g(\omega)_{12} & .. \\ g(\omega)_{12} & .. & \\ & & \end{Bmatrix}$$

$$g(\omega)_{ij} = H_{ij}(\omega) \prod_{ij} H(\omega)_{ij}^{*}$$

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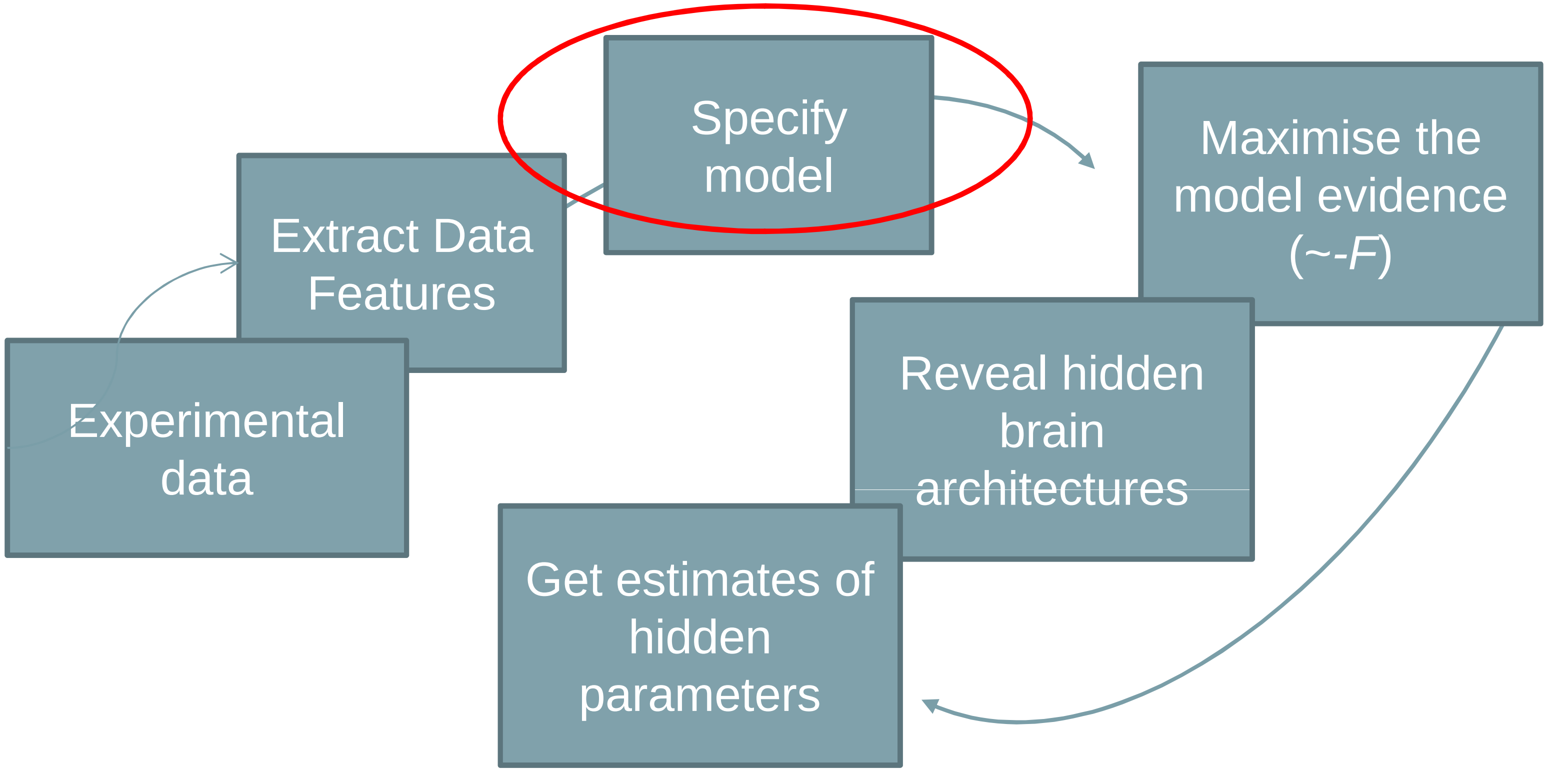
Specify model
Maximise the model evidence (~-F)
Extract Data Features
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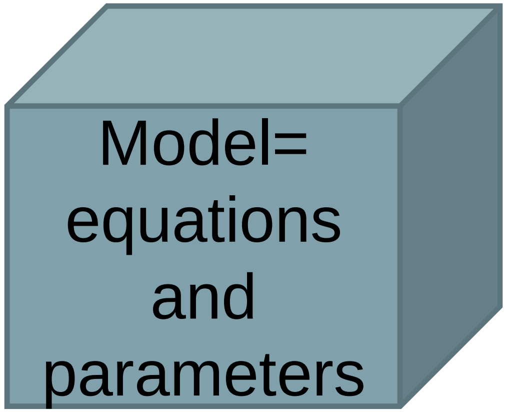
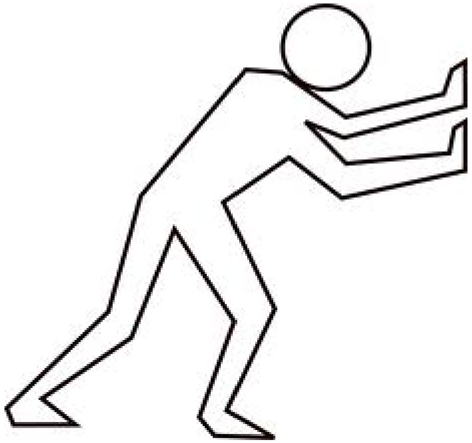
A Brain Region as an Input - Output System

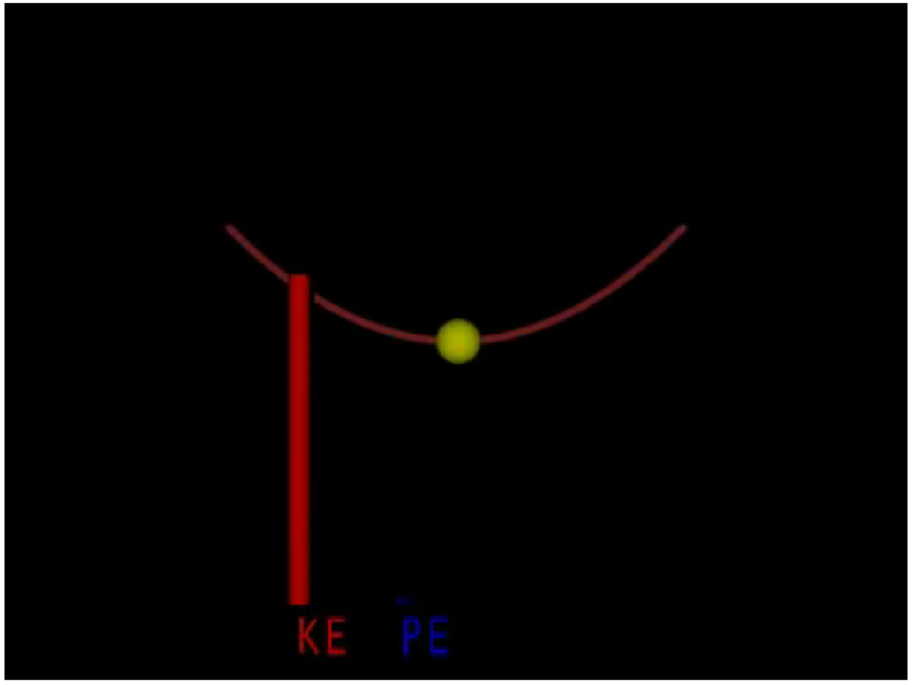


- equations: determine the dynamics (eg. limit cycles, transients, steady –state)
- parameters: fine tune the dynamics (e.g. faster, shorter)

$$\theta = \{H_e, H_i, \kappa_e, \kappa_i, \kappa_a, \gamma_1, \gamma_2, \gamma_3, \gamma_4, \gamma_5, g, A_F, A_B, A_L, \lambda\}$$

Maximum PSP, time constants, intrinsic and extrinsic connectivity etc





Pink line =
Container (bowl)

If there is no external perturbation, the ball will stay at the centre

If there is, the container will be tilted and the ball will oscillate around the centre as shown

Now, imagine that the ball has a bell inside. If there is an external perturbation the bell will start ringing.

CAUSE of CONTAINER TILTING ⟷ NEURAL NOISE

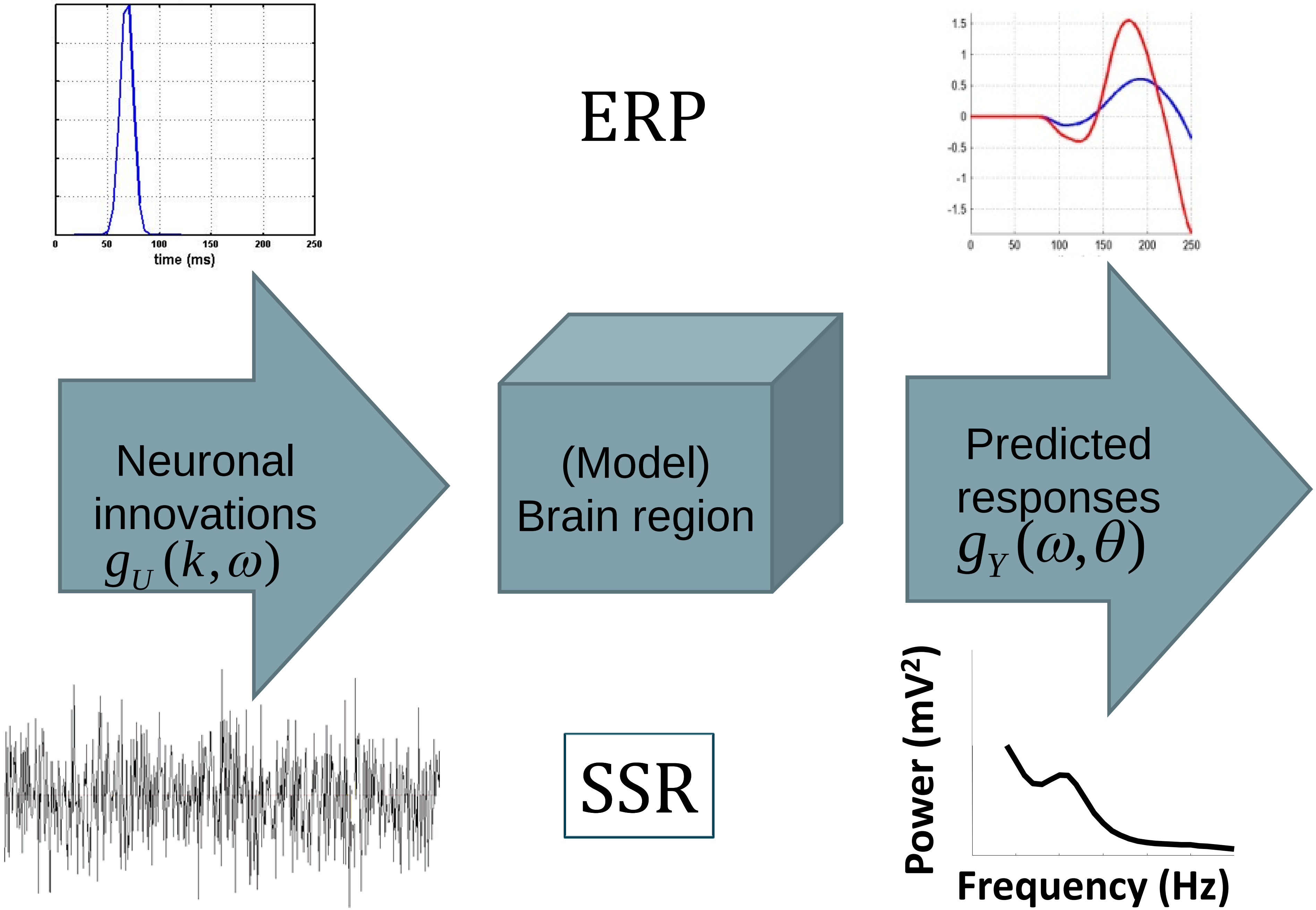
BALL ⟷ BRAIN REGION

RINGINGS ⟷ RESPONSES (cross spectral densities)

CONTAINER ⟷ MODEL (equations, parameters cf. shape/friction)

STEADY STATE PERTURBATIONS means that

“ the ball always stays very close to the centre” (while the bowl is tilted)


0
50
100
150
200
250
time (ms)
ERP
1.5
1
0.5
0
-0.5
-1
-1.5
0
50
100
150
200
250
Neuronal innovations
$g_U(k,\omega)$
(Model) Brain region
Predicted responses
$g_Y(\omega,\theta)$
SSR
Power (mV^2)
Frequency (Hz)

Frequency Domain Generative Model
(Perturbations about a fixed point)

Time Differential Equations

$$\dot{x} = f(x) + Bu$$
$$y = l(x)$$

→

State Space Characterisation

$$\dot{x} = Ax + Bu$$
$$y = Cx$$

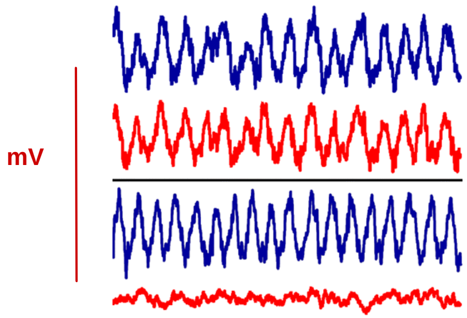
→

Transfer Function

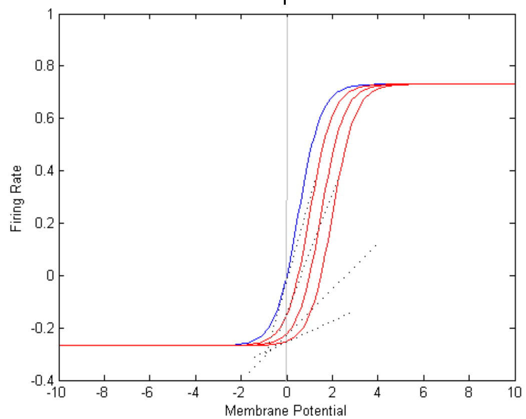
Frequency Domain

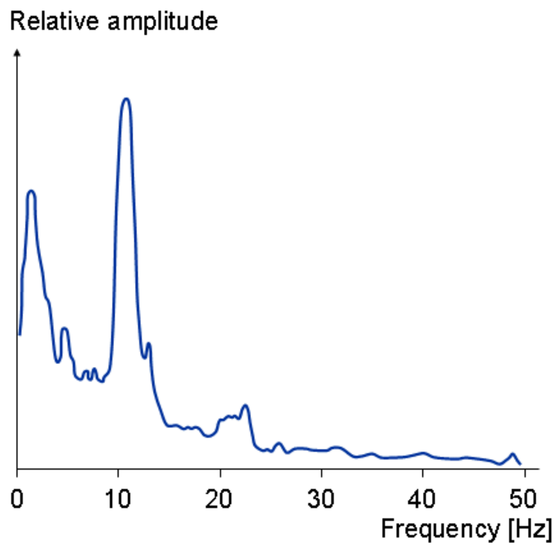
$$H(s) = C(sI - A)B$$

Stationary Time Series



Linearise





Frequency Domain Generative Model
(Perturbations about a fixed point)

Transfer Function Frequency Domain

$$H1(\omega) = f(\theta : H_{e/i}, \tau_{e,i} ..)$$

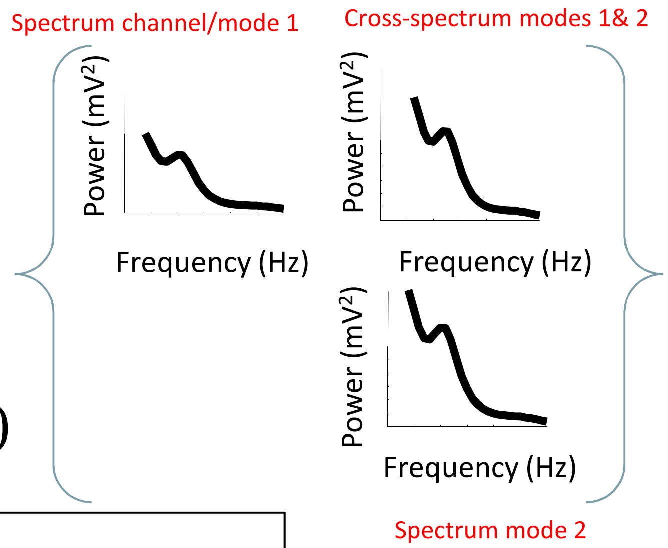
Cross-Transfer Function Frequency Domain

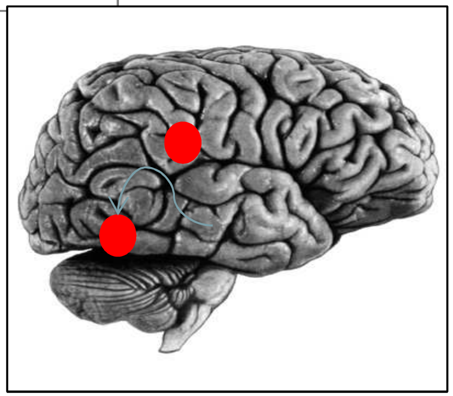
$$H12(\omega) = f(\theta : H_{e/i}, \tau_{e,i} ..)$$

Transfer Function Frequency Domain

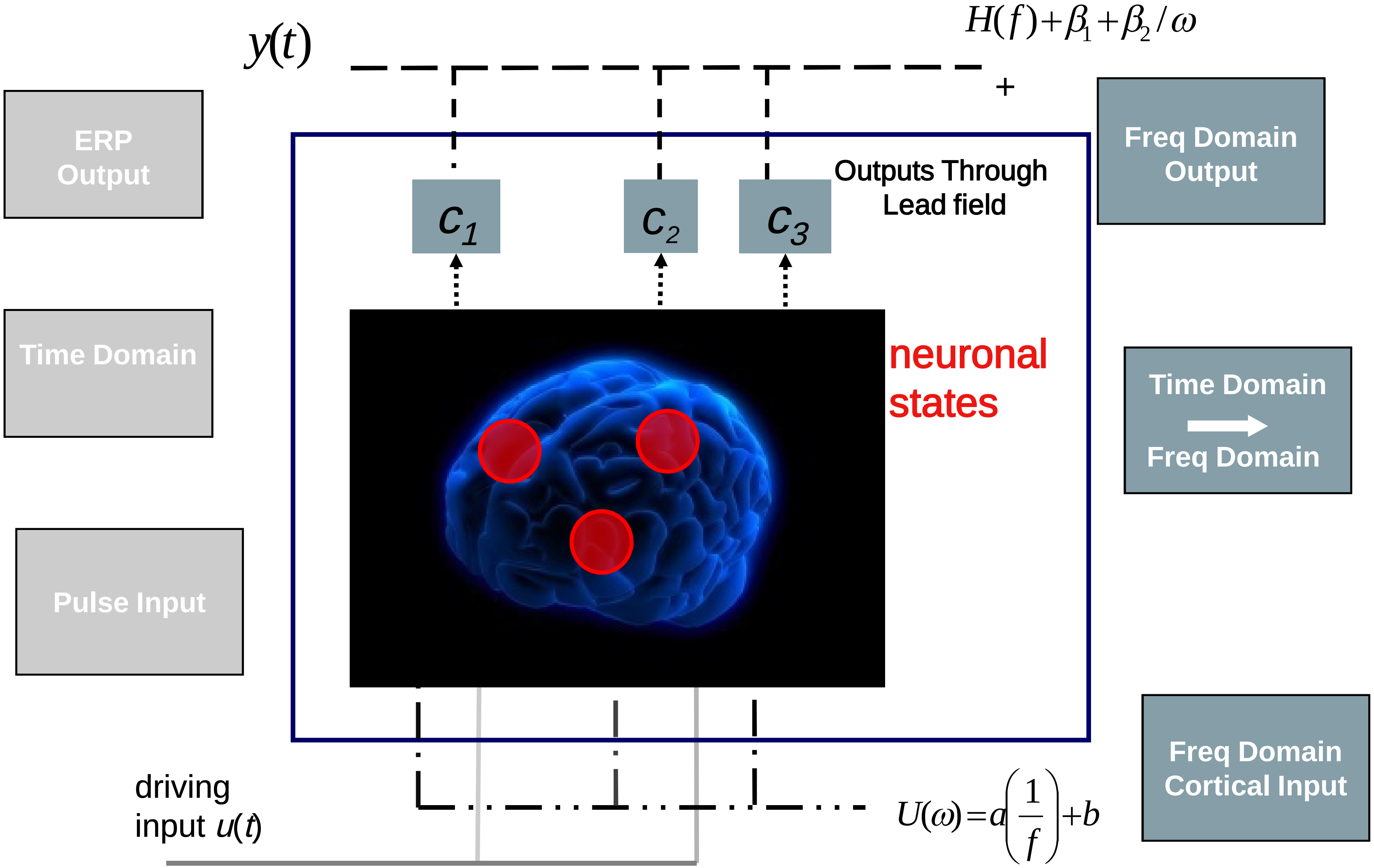
$$H2(\omega) = f(\theta : H_{e/i}, \tau_{e,i} ..)$$

Peaks ⟷ Parameters

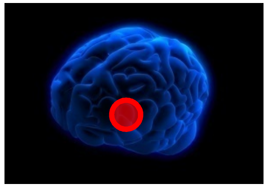




ERP vs Steady State Responses

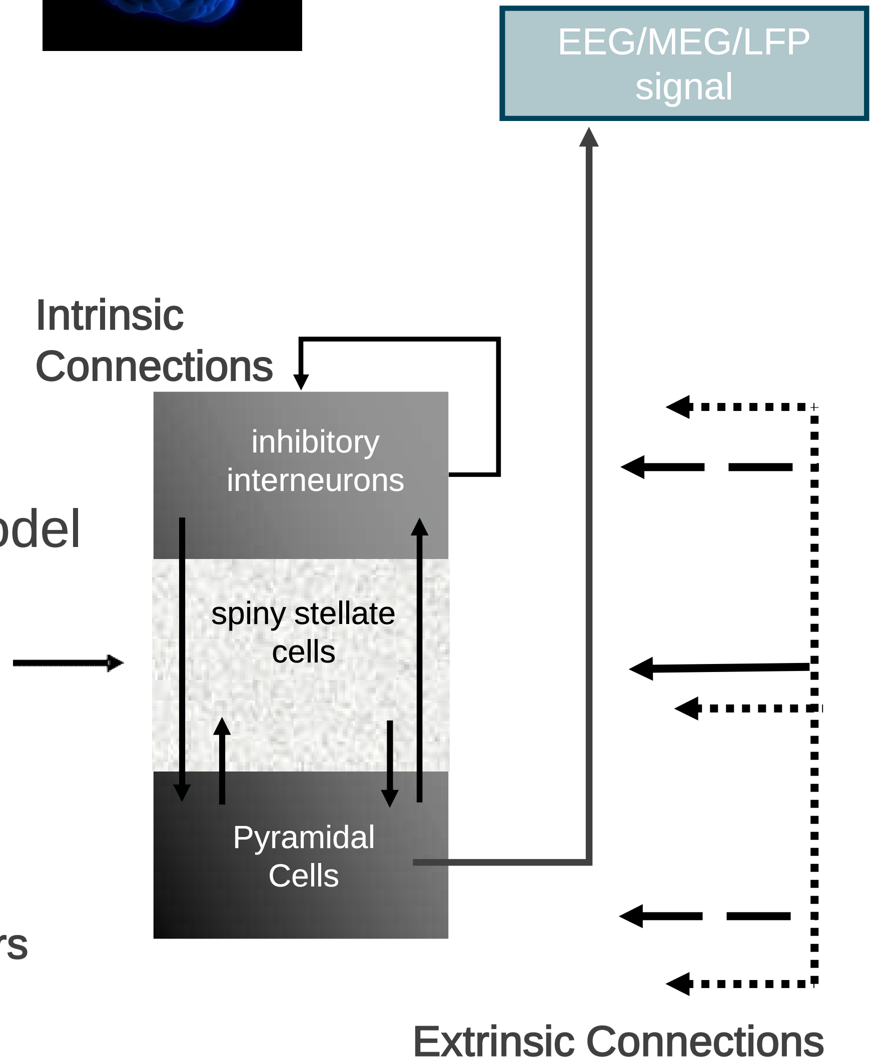


Neural Mass Model



Tens of thousands of neurons approximated by their average response. Neural mass models describe the interaction of these averages between populations and sources

neuronal (source) model

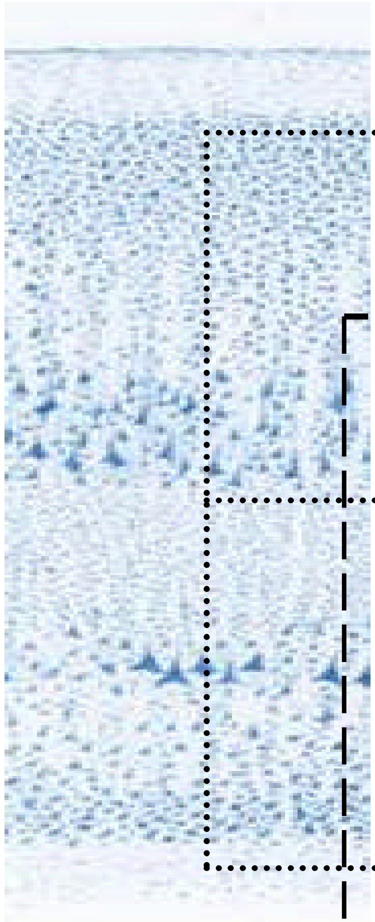


Internal Parameters

$\dot{x} = F(x, u, \theta)$ State equations

Neural Mass Model: Equations for Voltage and Current

Intrinsic connections



γ_5

Inhibitory cells in agranular layers

$$\dot{x}_7 = x_8$$
$$\dot{x}_8 = \kappa_e H_e (A^B + A^L + \gamma_3) S(x_9) - 2\kappa_e x_8 - \kappa_e^2 x_7$$
$$\dot{x}_{10} = x_{11}$$
$$\dot{x}_{11} = \kappa_i H_i \gamma_5 S(x_{12}) - 2\kappa_i x_{11} - \kappa_i^2 x_{10}$$
$$\dot{x}_{12} = x_8 - x_{11}$$

γ_4 γ_3

Excitatory spiny cells in granular layers

$$\dot{x}_1 = x_4$$
$$\dot{x}_4 = \kappa_e H_e ((A^F + A^L + \gamma_1) S(x_9) + Cu) - 2\kappa_e x_4 - \kappa_e^2 x_1$$

γ_1 γ_2

Excitatory pyramidal cells in agranular layers

$$\dot{x}_2 = x_5$$
$$\dot{x}_5 = \kappa_e H_e ((A^B + A^L) S(x_9) + \gamma_2 S(x_1)) - 2\kappa_e x_5 - \kappa_e^2 x_2$$
$$\dot{\mu}_3 = x_6$$
$$\dot{x}_6 = \kappa_i H_i \gamma_4 S(x_{12}) - 2\kappa_i x_6 - \kappa_i^2 x_3$$
$$\dot{x}_9 = x_5 - x_6$$

Extrinsic Connections:

Forward

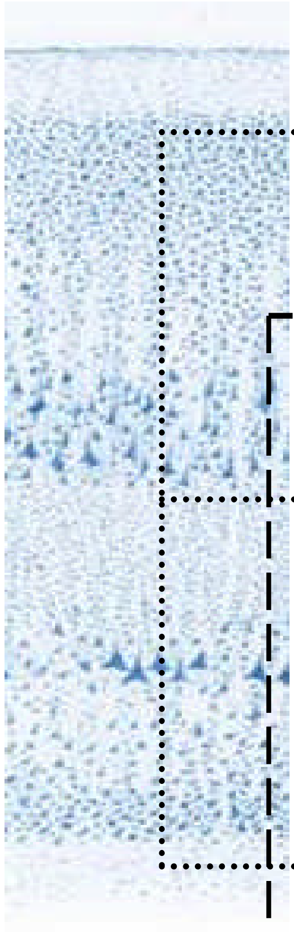
Backward

Lateral

$$\dot{x} = f(x, u)$$

Neural Mass Model: Two Transformations

Intrinsic connections



γ_5

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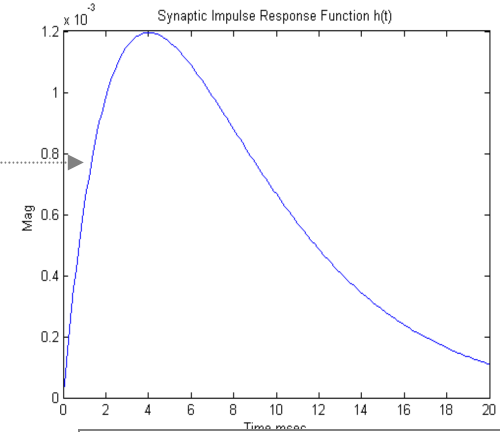
Extrinsic Connections:

Forward

Backward

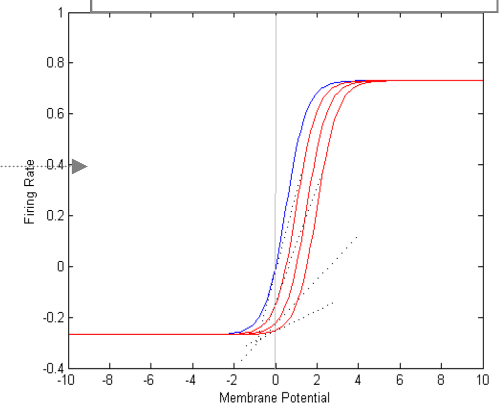
Lateral

$$\dot{x} = f(x,u)$$



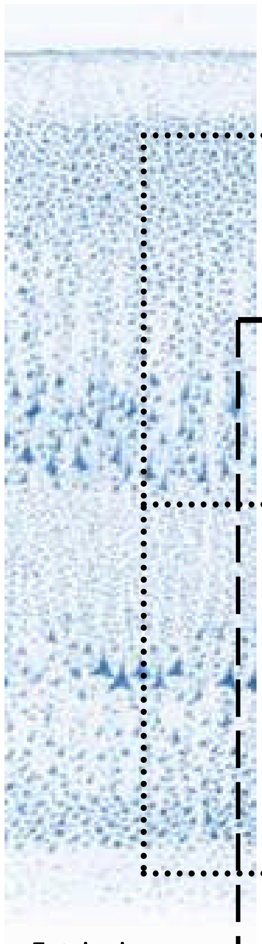
Synaptic 'alpha' kernel

Sigmoid function



Neural Mass Model: 1st Transformation

Intrinsic connections



γ_5

Inhibitory cells in agranular layers

$$\dot{x}_7 = x_8$$
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$$\dot{x}_9 = x_5 - x_6$$

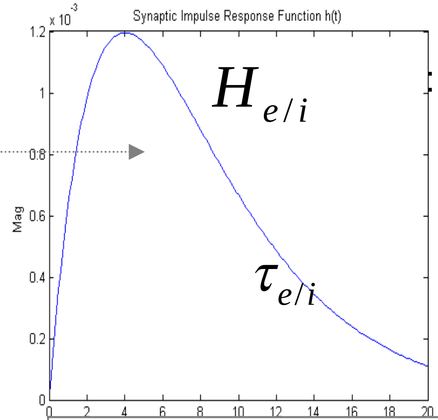
Extrinsic Connections:

Forward →

Backward - - →

Lateral ····→

$$\dot{x} = f(x, u)$$



$H_{e/i}$: Receptor Density

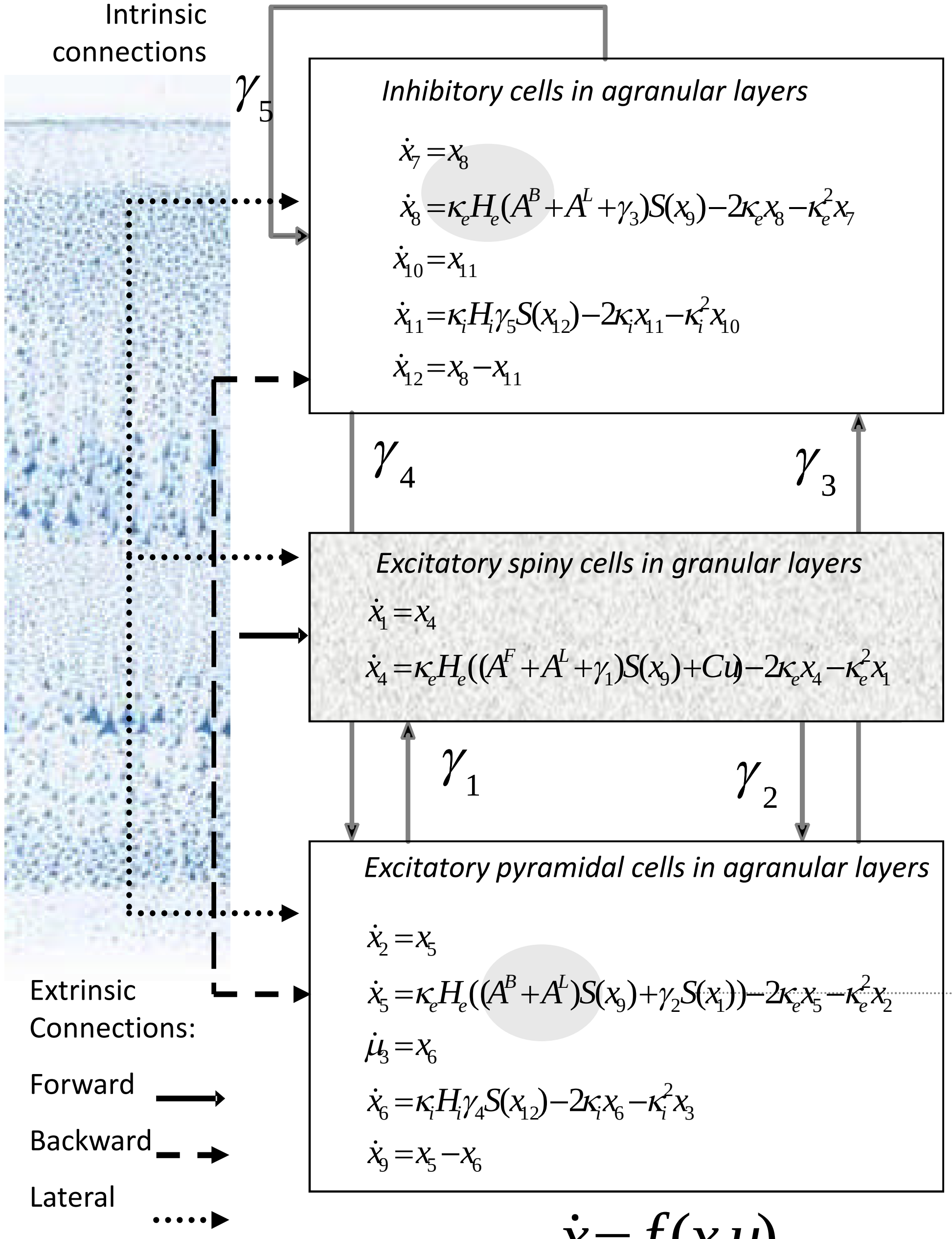
$\tau_{e/i}$

Synaptic 'alpha' kernel

$$v = r \otimes h$$

- Input: presynaptic rate
- Output: Average synaptic depolarization

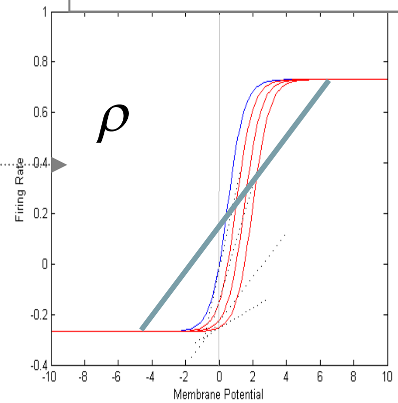
Neural Mass Model: 2nd Transformation



$$\dot{x} = f(x,u)$$

- Input: Avg synaptic depolirization
- Output: firing rate

Sigmoid function



: Firing Rate

$$r = S(v)$$

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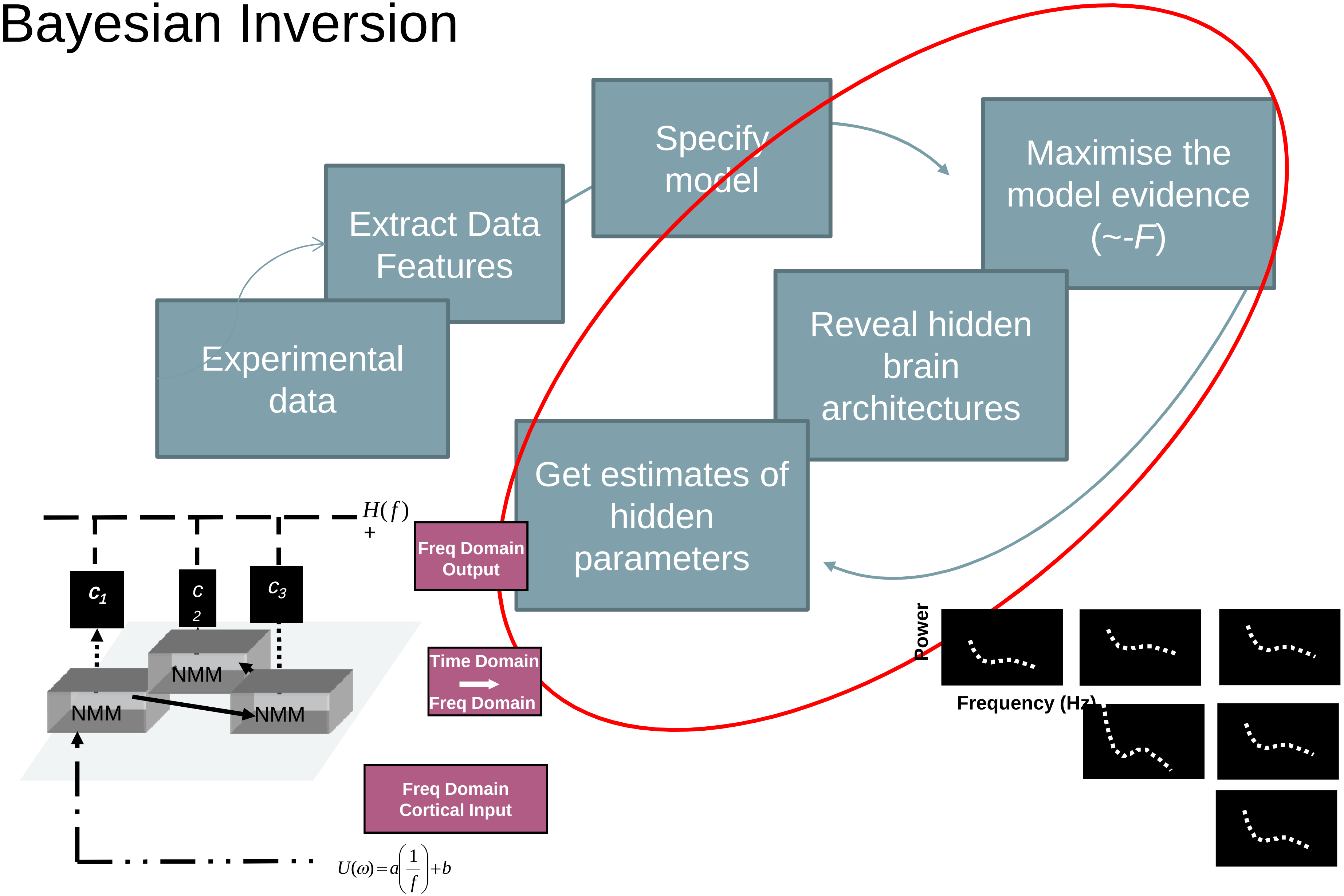
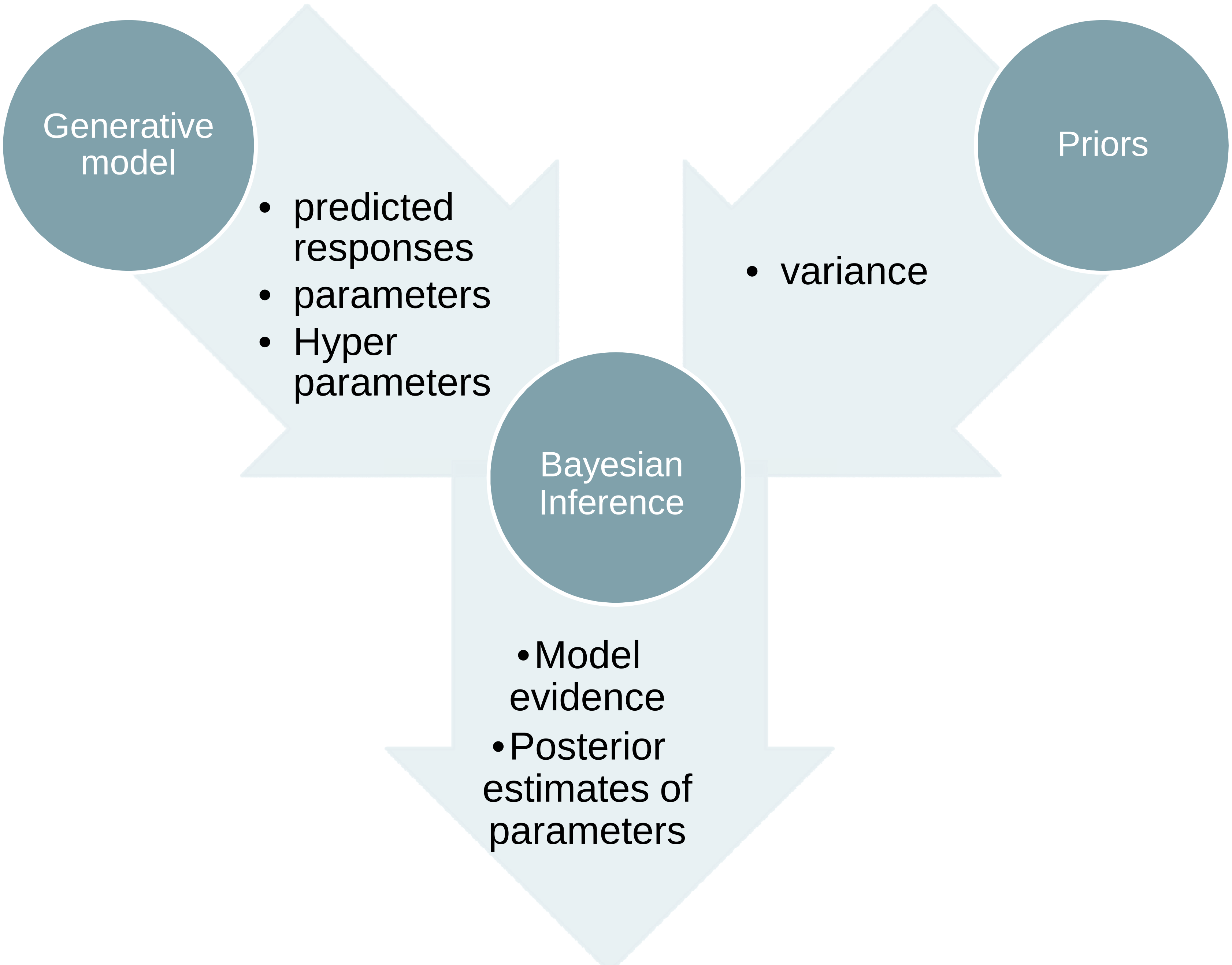
Bayesian Inversion



Roadmap


Generative model
predicted responses
parameters
Hyper parameters
Priors
variance
Bayesian Inference
Model evidence
Posterior estimates of parameters

Model Fit and Comparison

DATA

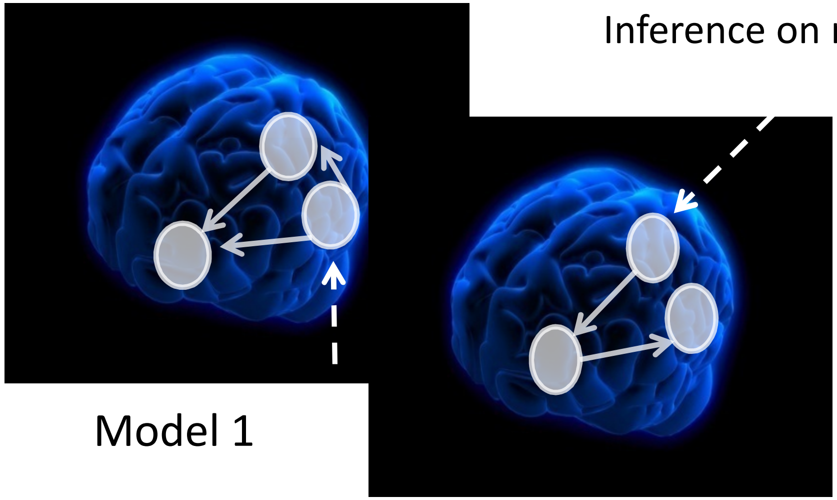
PREDICTIONS

Variational Bayes Algorithm

- Iterative procedure
- Minimize free energy and optimize parameters
- Maximum accuracy over complexity constraints

Bayesian Inversion

Inference on models



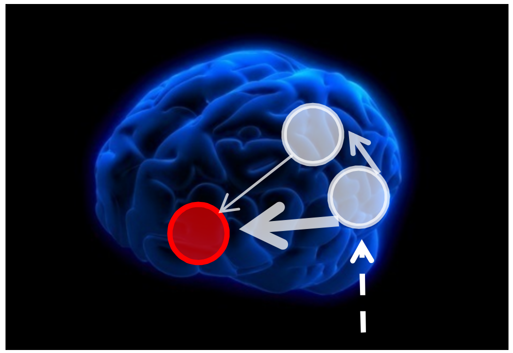
Model 1

Model 2

Model comparison via Bayes factor:

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

Inference on parameters



Model 1

$$q(\theta) \approx p(\theta \mid y, m)$$

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Pharmacological Manipulation of Glutamate and GABA

Questions of Study:

- ❑ Are our estimates of excitation and inhibition veridical, e.g. H_e, H_i?
- ❑ Can we obtain hierarchical relationships between brain regions?

AIM:

NOT to explain mechanisms of isoflurane BUT

to exploit isoflurane to induce known changes in synaptic transmission and THEN

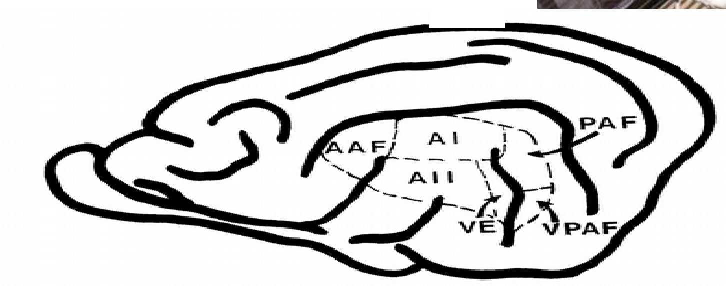
Use LFP recordings and DCM for SSR to infer changes

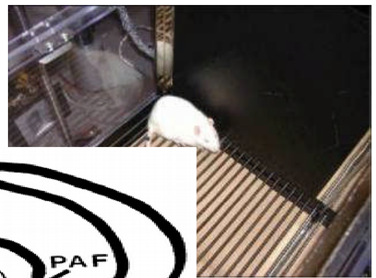
Moran, Tetsuya, Jung, Endepols, Graf, Dolan, Friston, Stephan, Tittgemeyer, *PLoSONE, 2011*

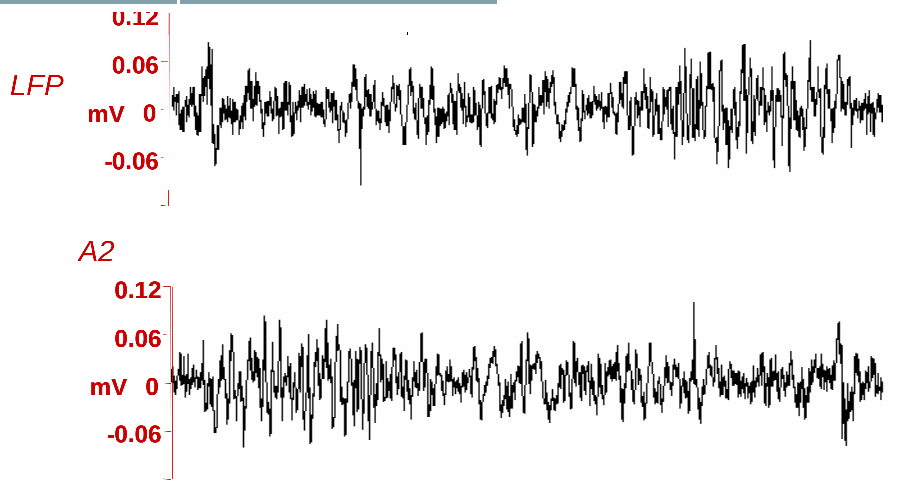
Pharmacological Manipulation of Glutamate and GABA

- Use animal LFP recordings from primary auditory cortex (A1) & posterior auditory field (PAF)
- Manipulate neurotransmitter processing via anaesthetic agent Isoflurane
- 4 levels of anaesthesia: each successively decreasing glutamate and increasing GABA (Larsen *et al* Brain Research 1994; Lingamaneni *et al* Anesthesiology 2001; Caraiscos *et al* J Neurosci 2004 ; de Sousa *et al* Anesthesiology 2000
- White noise stimulus & Silence

1.4 % Isoflurane	1.8 % Isoflurane	2.4 % Isoflurane	2.8 % Isoflurane

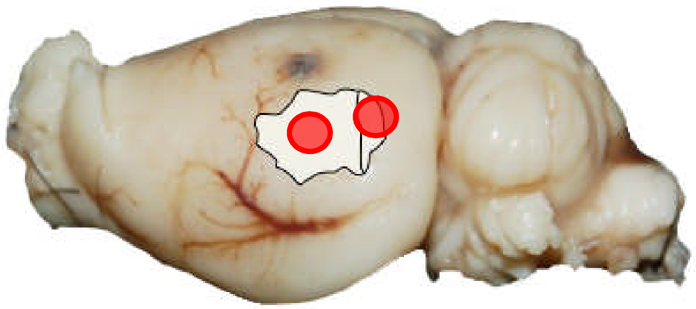




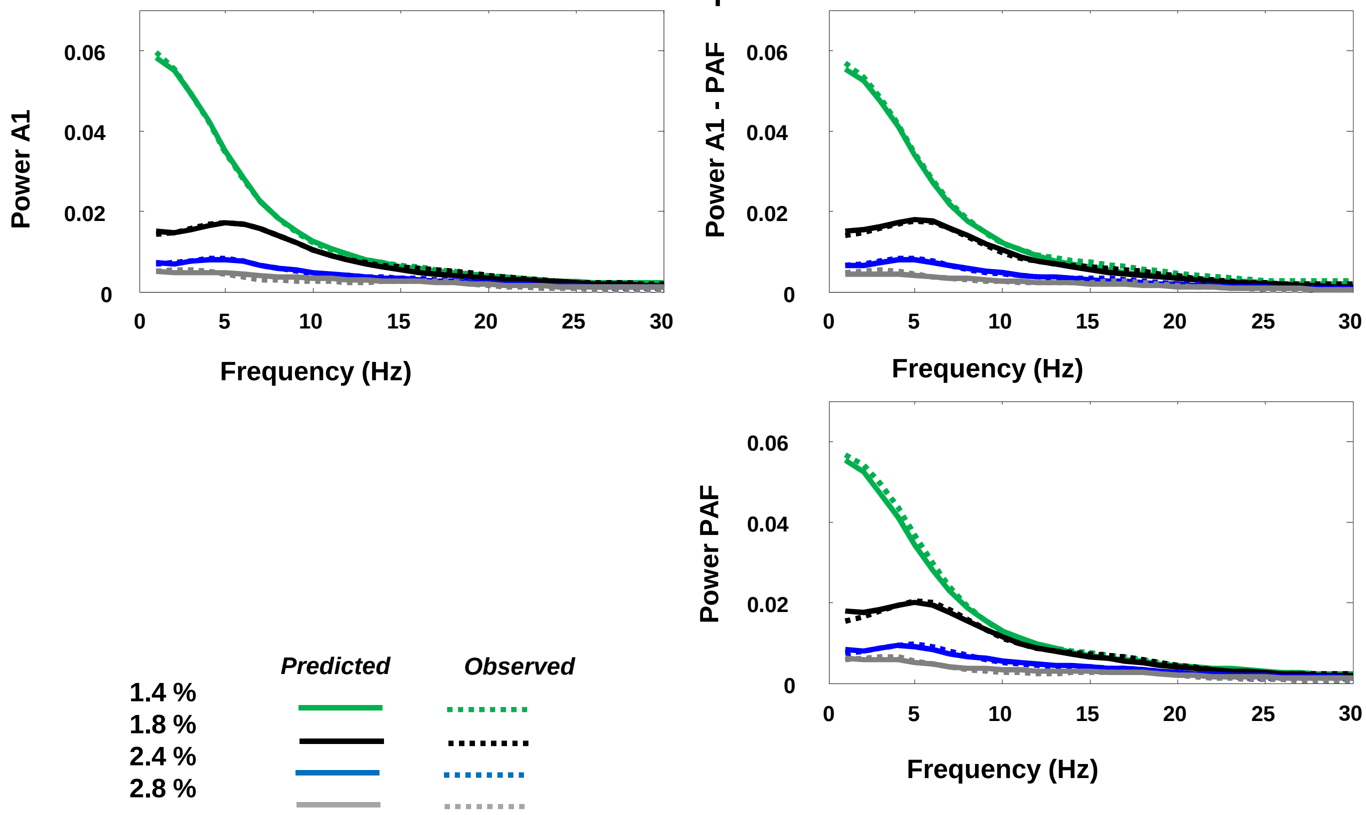


Pharmacological Manipulation of Glutamate and GABA

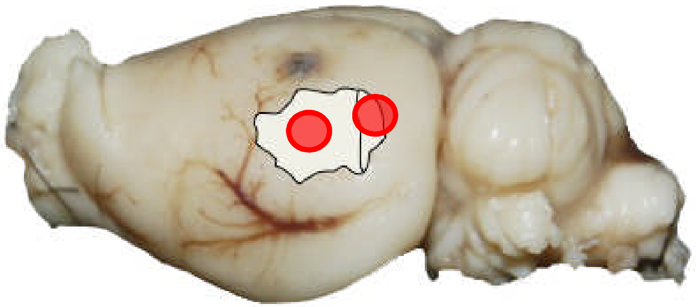
Data

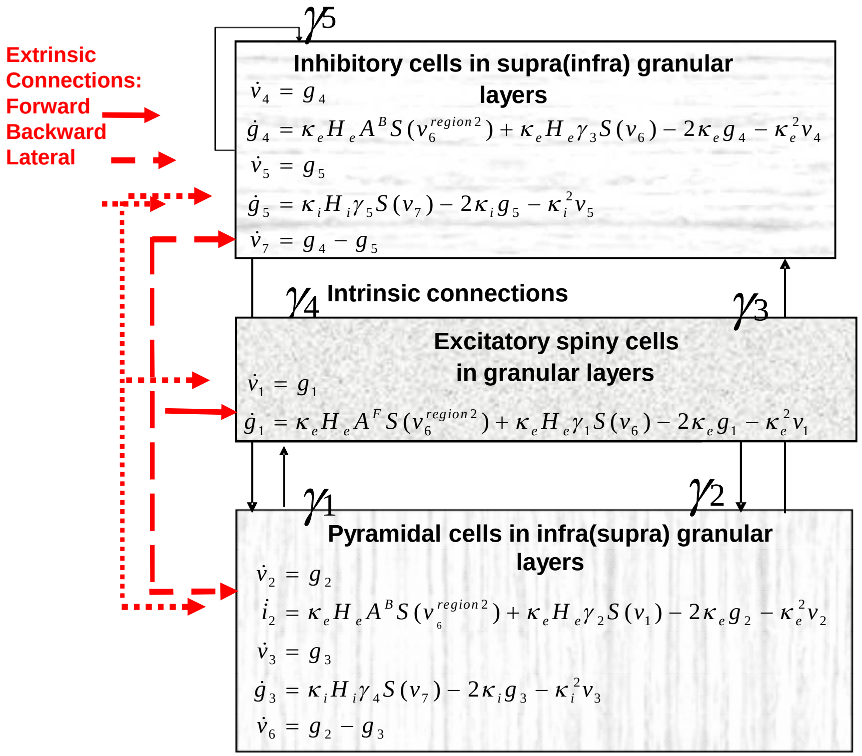


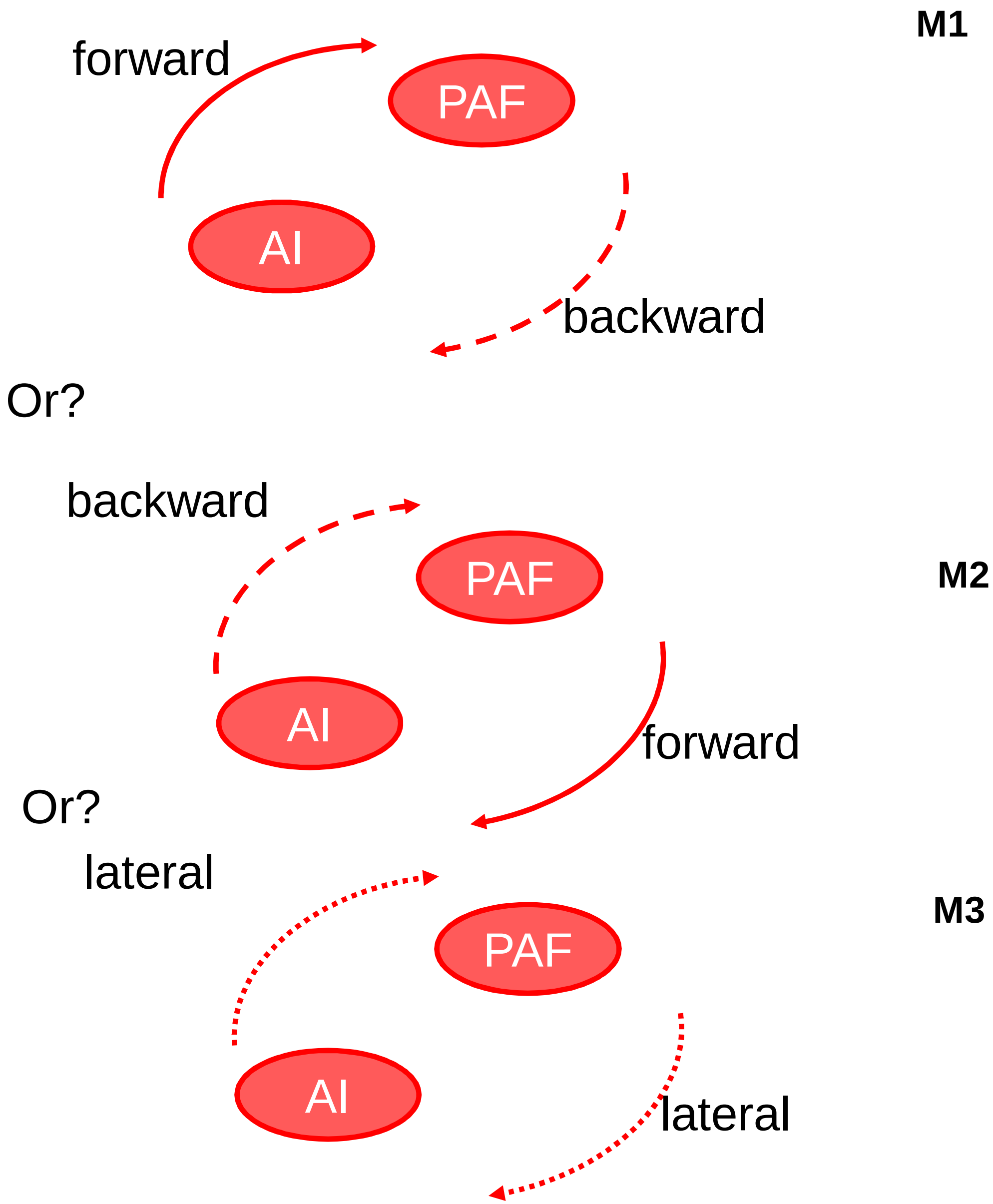
Cross-spectra white noise



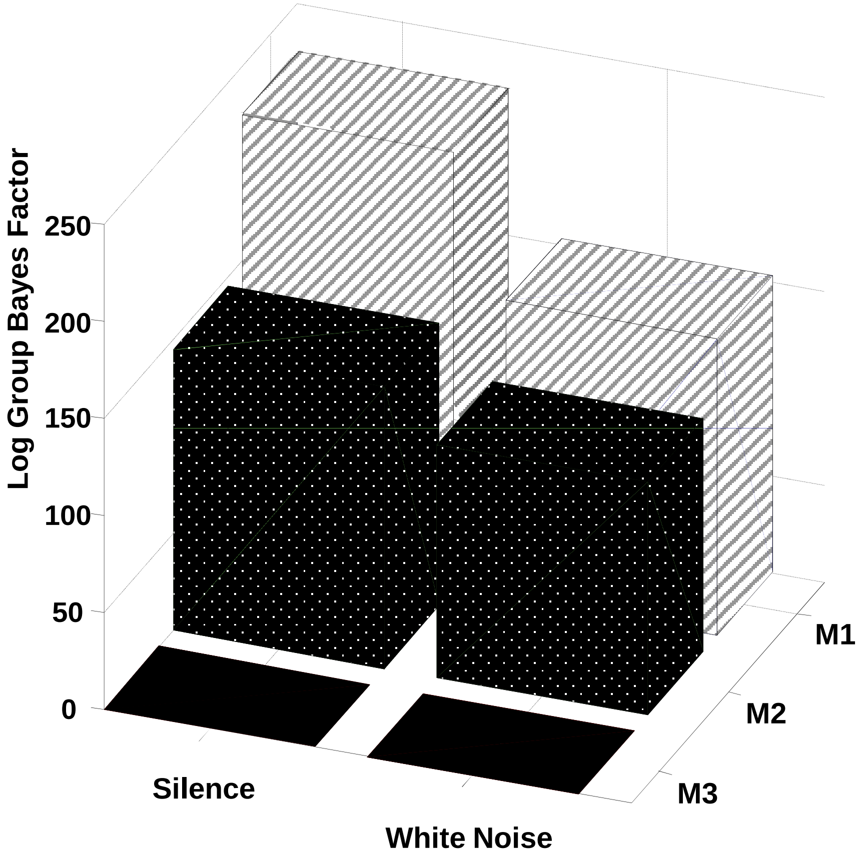
Model

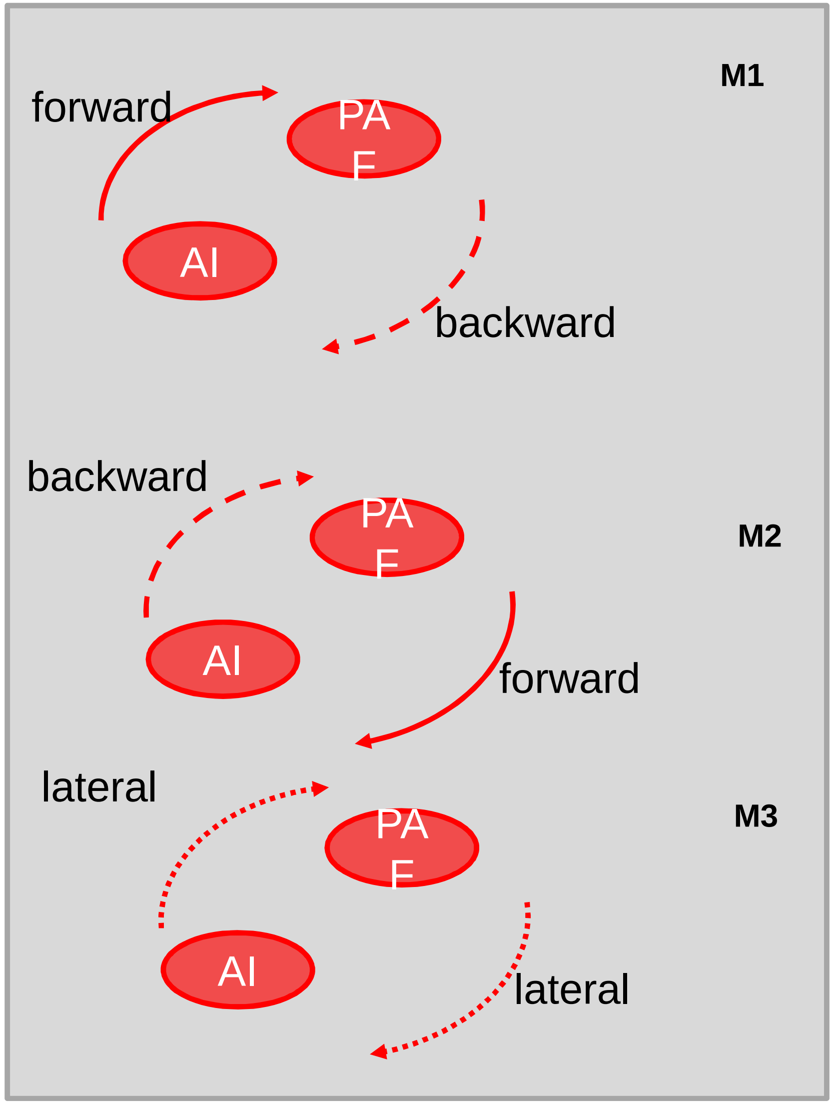



Extrinsic Connections:
Forward
Backward
Lateral
γ_5
Inhibitory cells in supra(infra) granular layers
$\dot{v}_4 = g_4$
$\dot{g}_4 = \kappa_e H_e A^B S(v_6^{region2}) + \kappa_e H_e \gamma_3 S(v_6) - 2\kappa_e g_4 - \kappa_e^2 v_4$
$\dot{v}_5 = g_5$
$\dot{g}_5 = \kappa_i H_i \gamma_5 S(v_7) - 2\kappa_i g_5 - \kappa_i^2 v_5$
$\dot{v}_7 = g_4 - g_5$
γ_4 Intrinsic connections
γ_3
Excitatory spiny cells in granular layers
$\dot{v}_1 = g_1$
$\dot{g}_1 = \kappa_e H_e A^F S(v_6^{region2}) + \kappa_e H_e \gamma_1 S(v_6) - 2\kappa_e g_1 - \kappa_e^2 v_1$
γ_1
γ_2
Pyramidal cells in infra(supra) granular layers
$\dot{v}_2 = g_2$
$\dot{i}_2 = \kappa_e H_e A^B S(v_6^{region2}) + \kappa_e H_e \gamma_2 S(v_1) - 2\kappa_e g_2 - \kappa_e^2 v_2$
$\dot{v}_3 = g_3$
$\dot{g}_3 = \kappa_i H_i \gamma_4 S(v_7) - 2\kappa_i g_3 - \kappa_i^2 v_3$
$\dot{v}_6 = g_2 - g_3$


M1
forward
PAF
AI
backward
Or?
backward
PAF
M2
AI
forward
Or?
lateral
PAF
M3
AI
lateral

Model





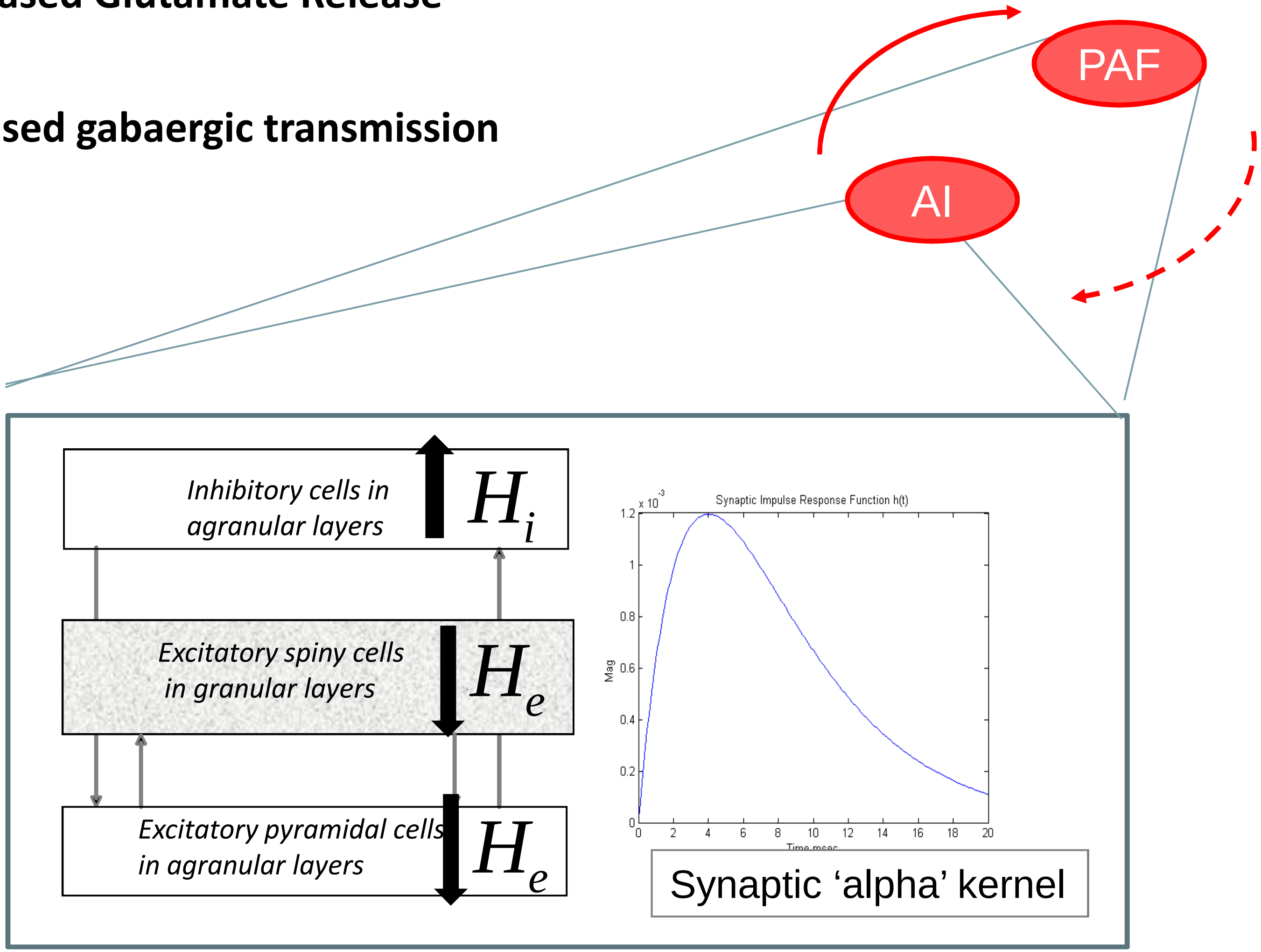
❑DCM recovers known neuronatomy

Physiological Parameters

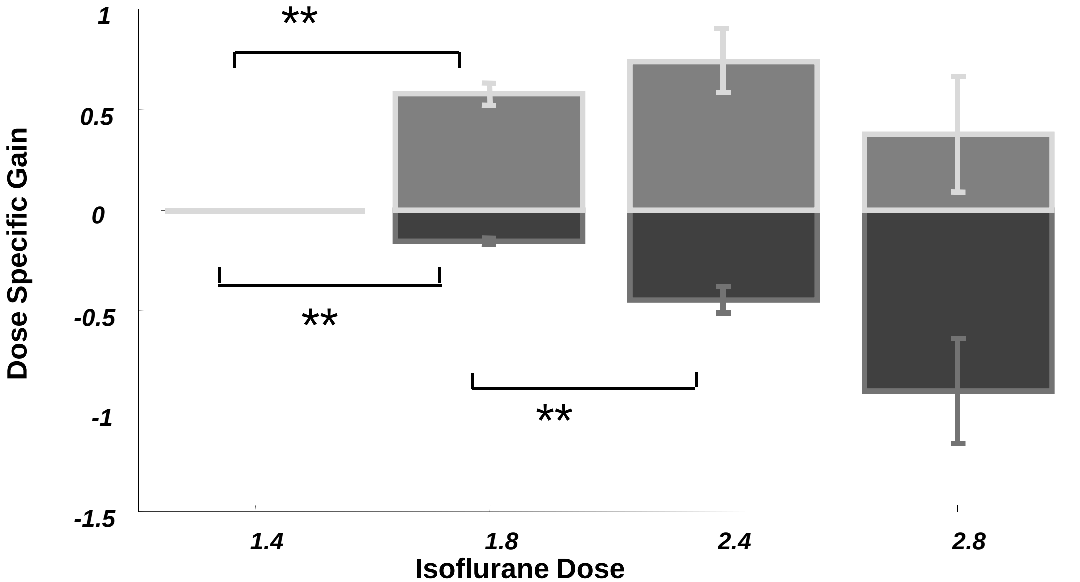
1.4 % Isoflurane	1.8 % Isoflurane	2.4 % Isoflurane	2.8 % Isoflurane

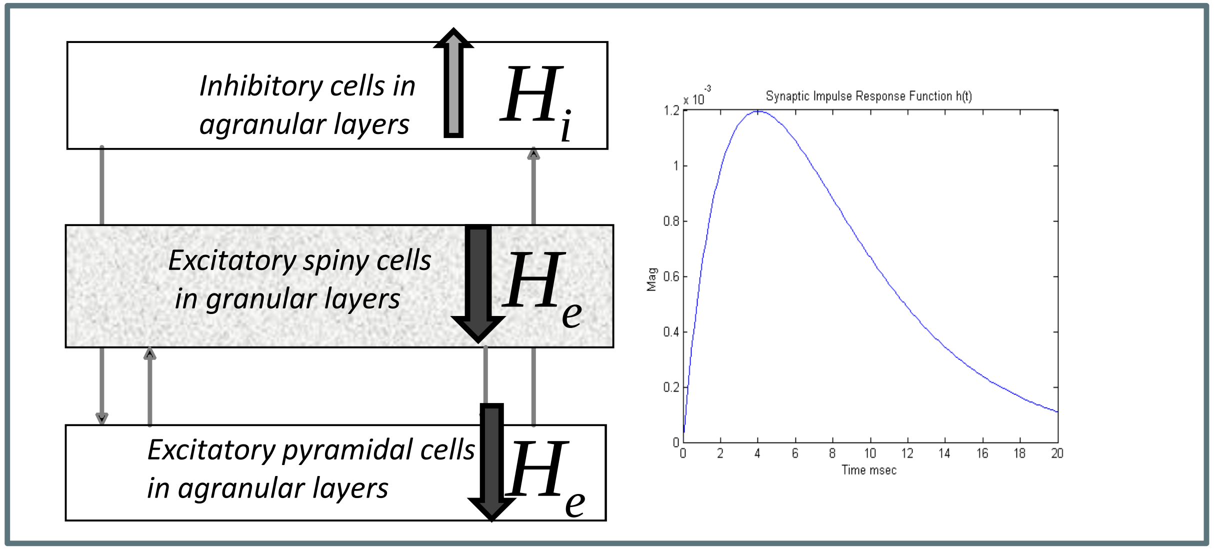
Decreased Glutamate Release

Increased gabaergic transmission



Parametric Effect of Isoflurane

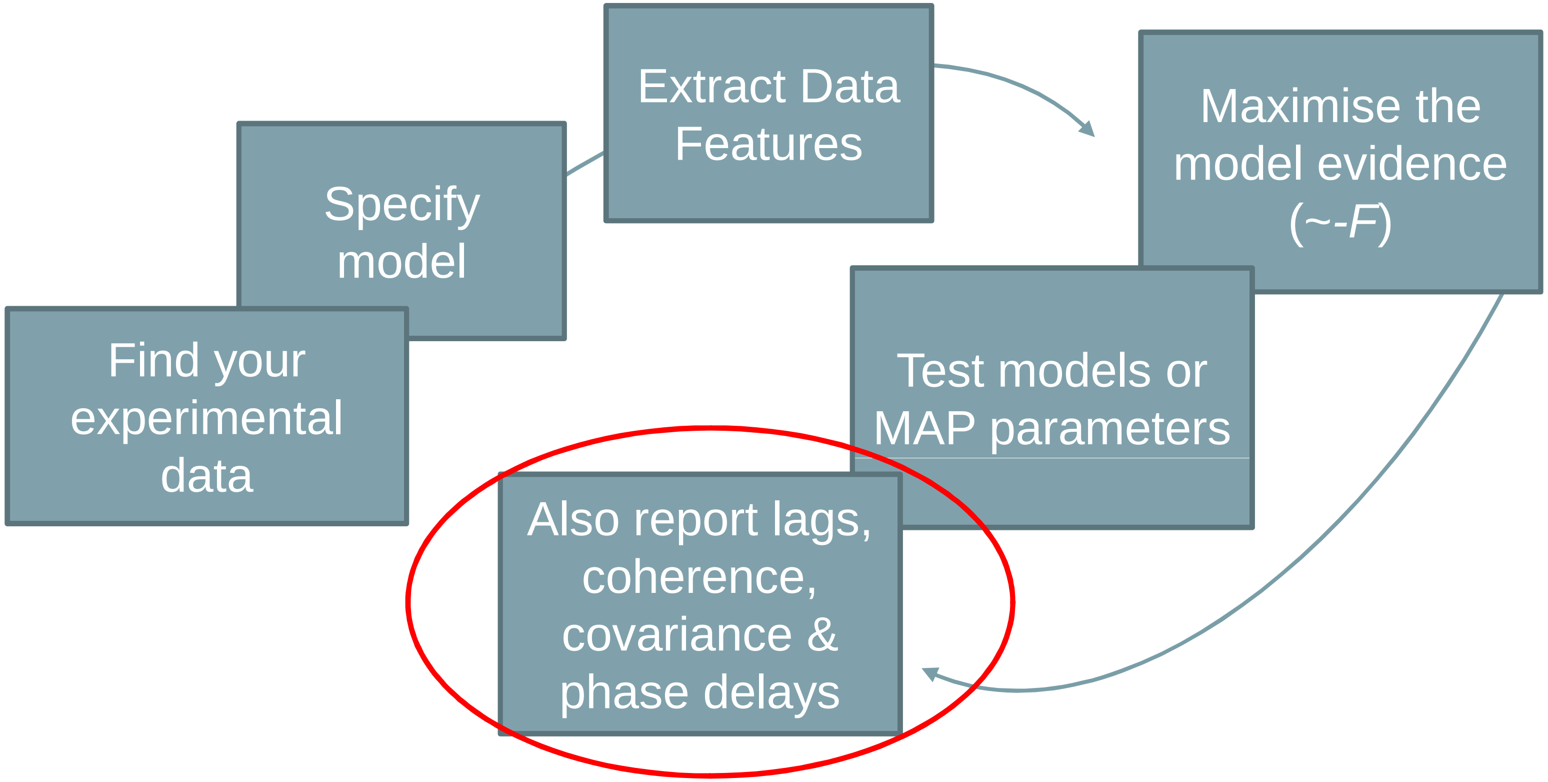




- ❑ DCM recovers known drug-induced changes

Overview

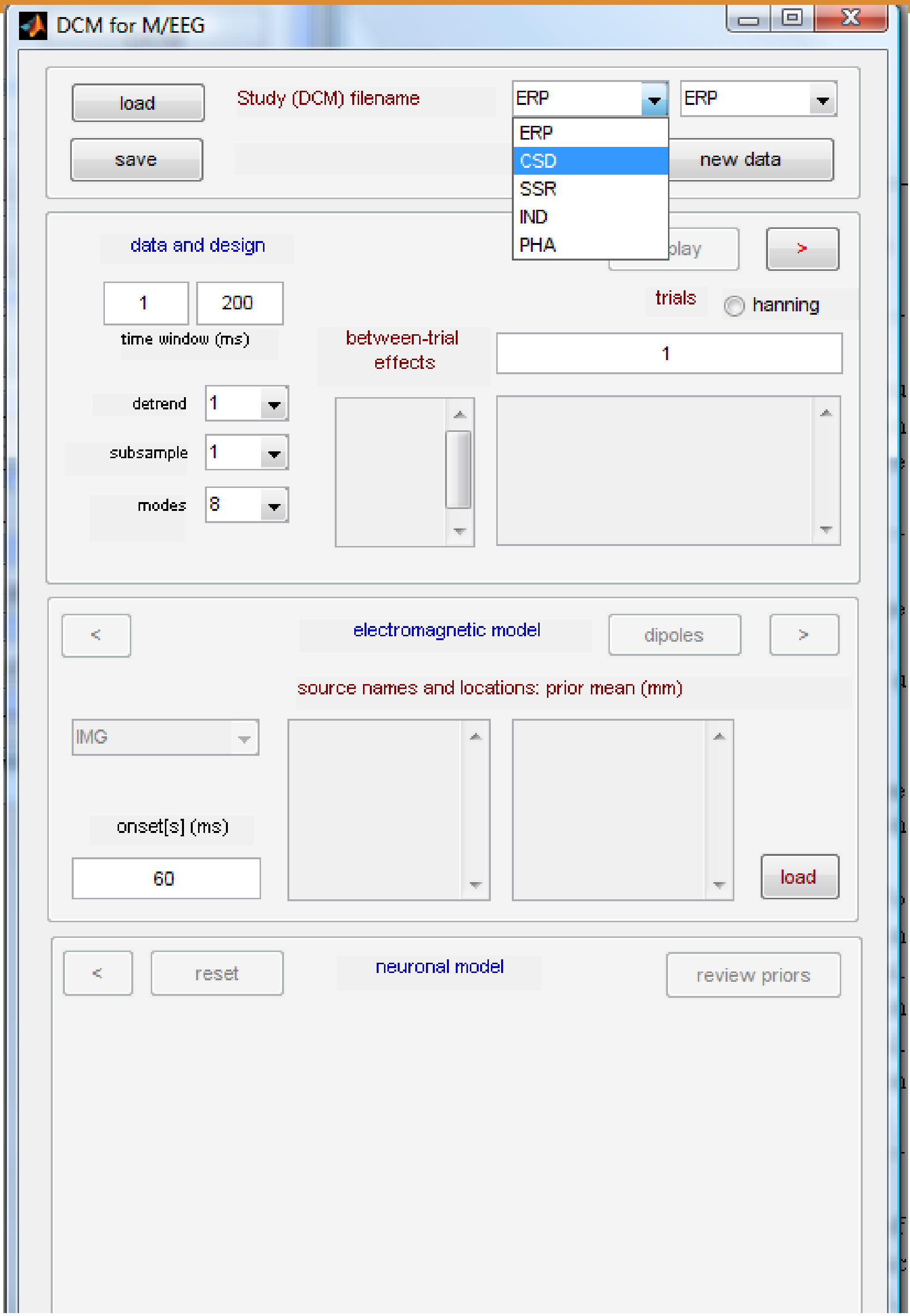
1. Data Features
2. Generative Model
3. Bayesian Inversion: Parameter Estimates and Model Comparison
4. Example: Glutamate and GABA in Rodent Auditory Cortex
5. DCM for Cross Spectral Density
6. DCM for Neural Fields

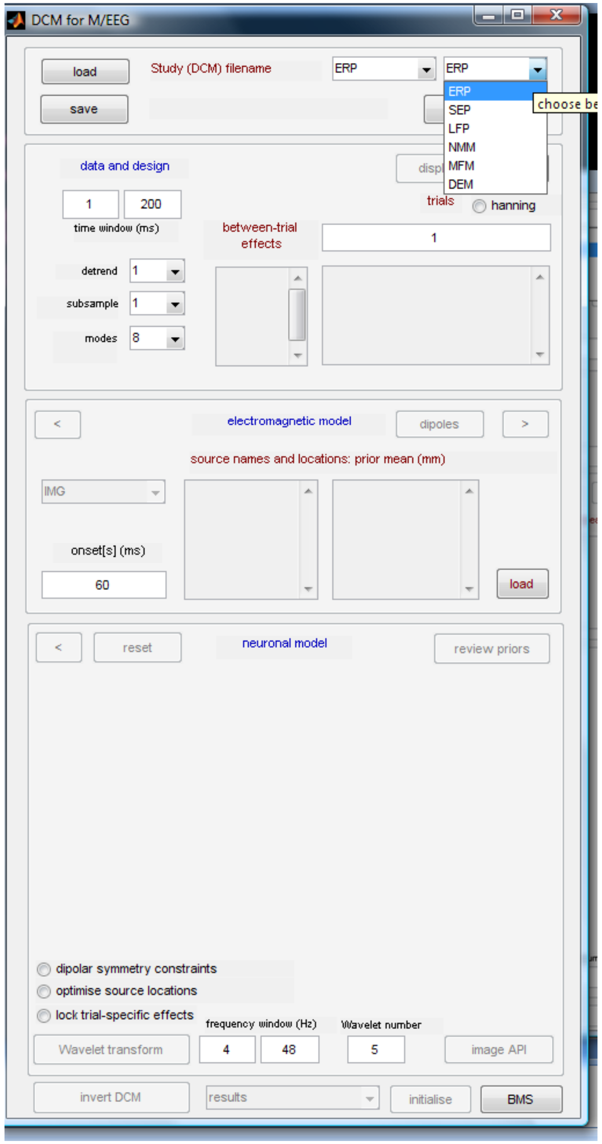


1. Interface Additions
2. New CSD routines, similar to SSR
3. SPM_NLSI_GN accommodates imaginary numbers, slopes, curvatures
4. A host of new results features, in channel and source space!

Friston, Bastos, Litvak, Stephan, Fries, Moran, *Neuroimage, 2012*

Interface Additions





Frequency Domain Generative Model
(Perturbations about a fixed point)

Complex Number

Transfer Function

Time Differential Equations

$$\dot{x} = f(x) + Bu$$
$$y = l(x)$$

→

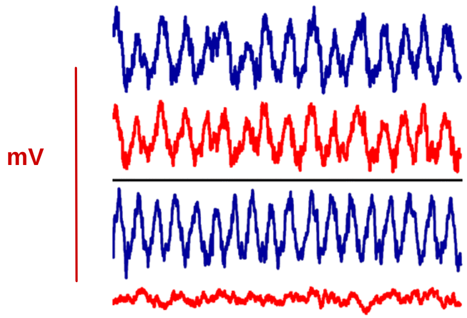
State Space Characterisation

$$\dot{x} = Ax + Bu$$
$$y = Cx$$

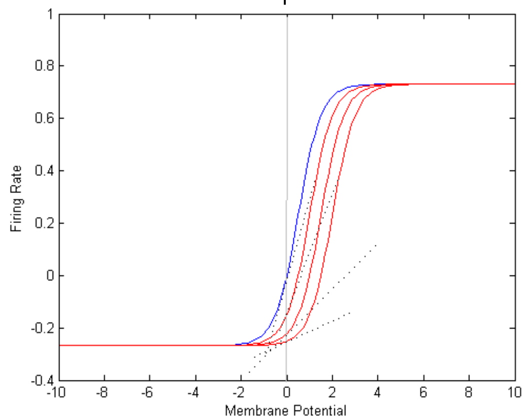
→

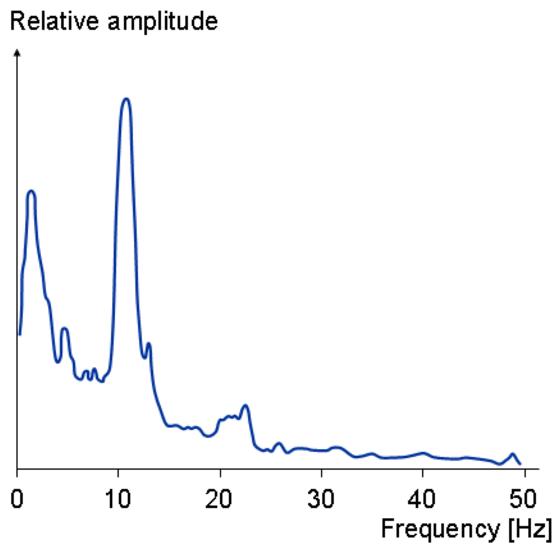
Frequency Domain

$$H(s) = C(sI - A)B$$

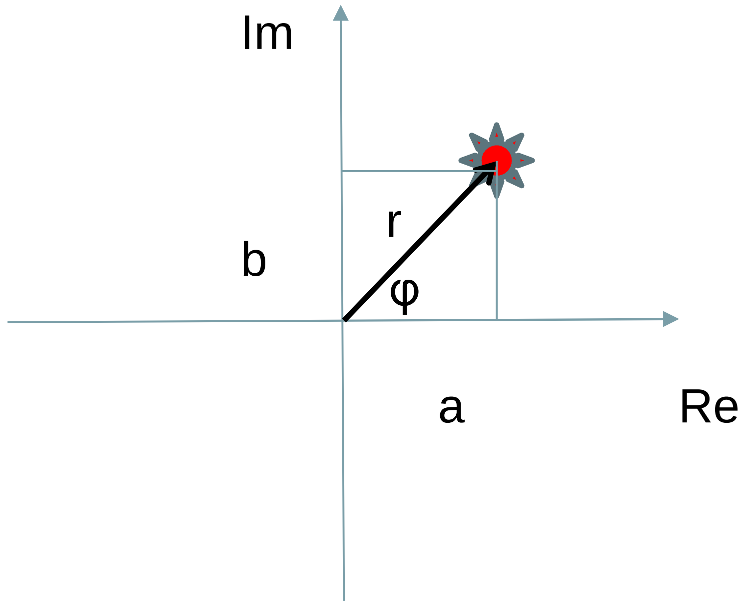


Linearise





What is a complex number ?



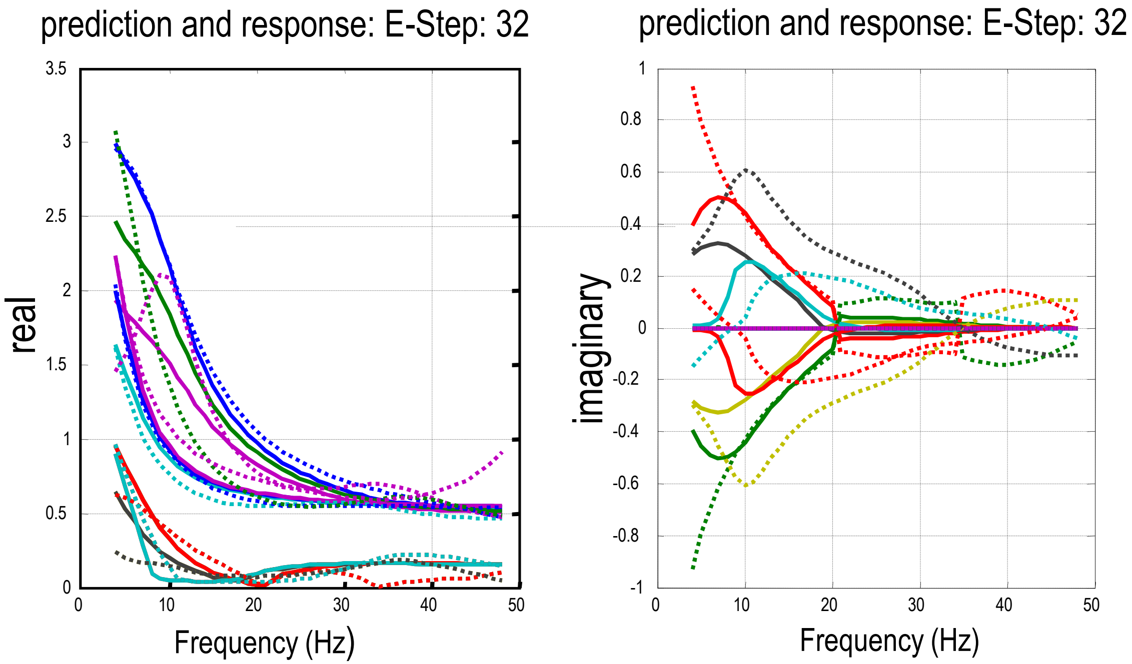
$$z = a + ib = r(\cos\varphi + i\sin\varphi) = re^{i\varphi}$$

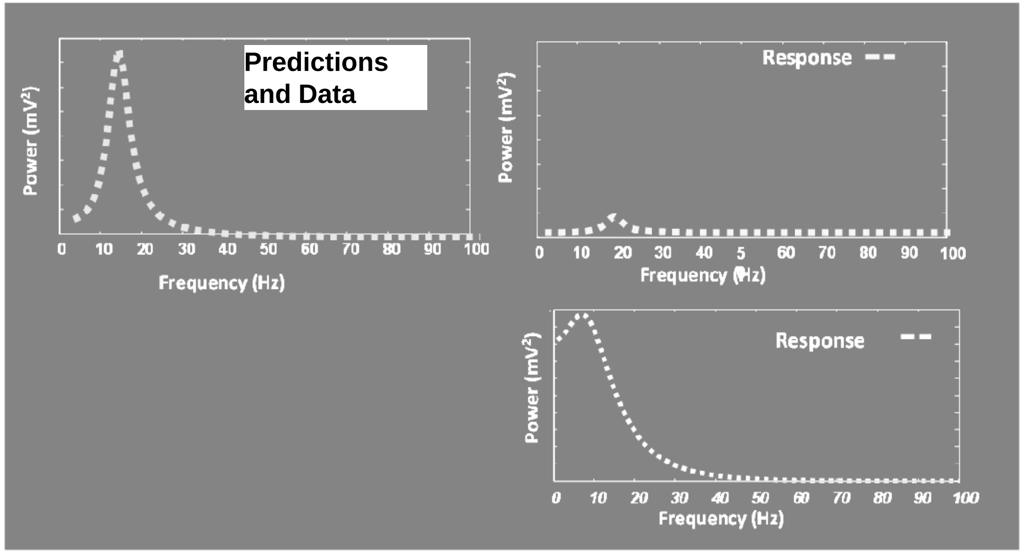
$$i^2 = -1$$

$$H(\omega) = \mathcal{F}(f) = \int_{-\infty}^{\infty} f(t)\, e^{-2\pi\omega i t}\, dt$$

The Fourier transform of a signal is a continuous complex function

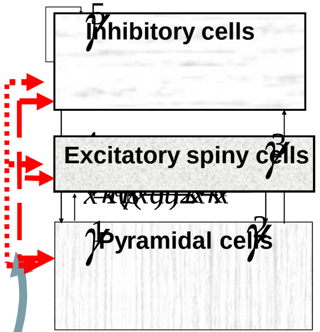
❑DCM for CSD: data fits have **two** parts: real and imaginary



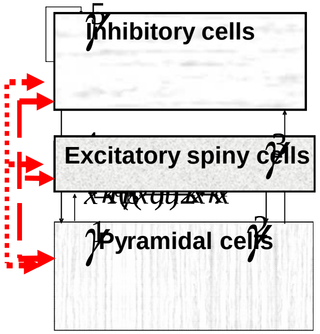


Model Inversion using absolute value (modulus)

$| H_2(\omega) \,.\, H_2^*(\omega) |$



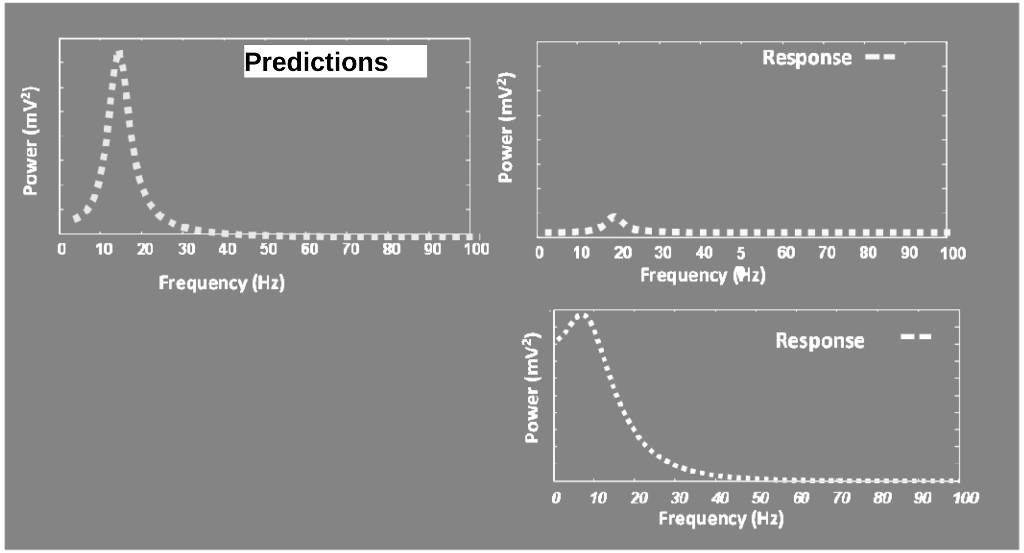
$| H_1(\omega) \,.\, H_1^*(\omega) |$



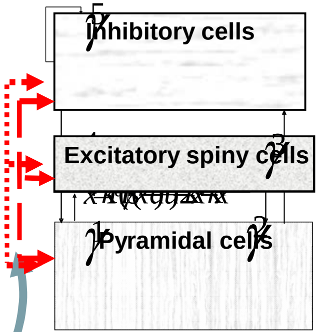
$| H_1(\omega) \,.\, H_2^*(\omega) |$

DCM for Cross Spectral Density

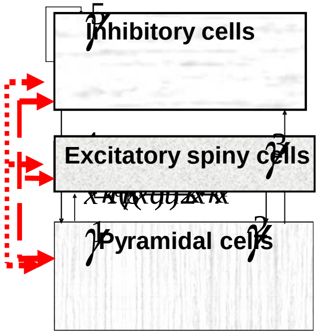
Generative Model



$H_2(\omega) \,.\, H_2^*(\omega)$

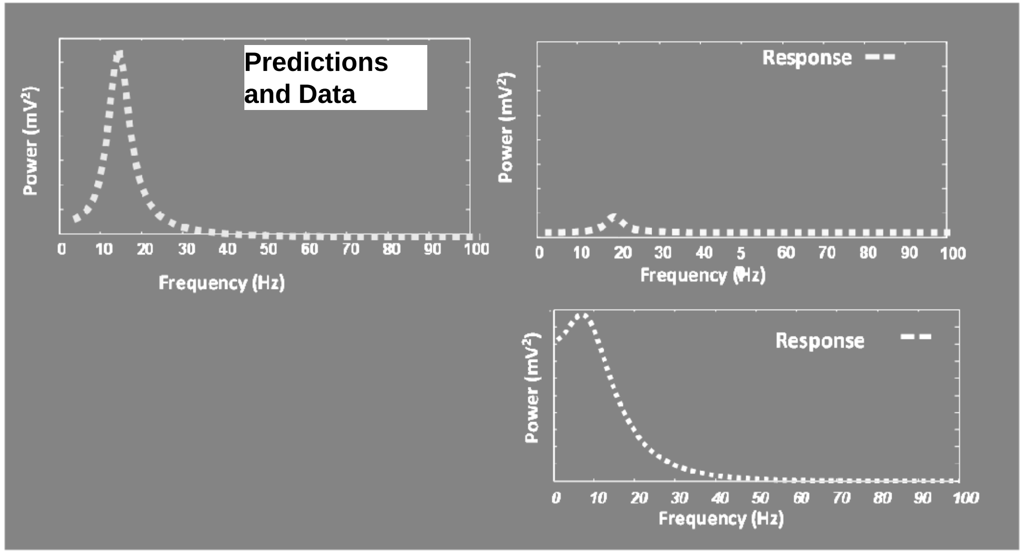


$H_1(\omega) \,.\, H_1^*(\omega)$



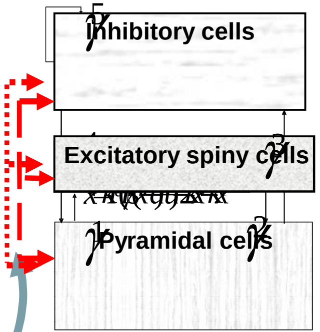
$H_1(\omega) \,.\, H_2^*(\omega)$

DCM for Cross Spectral Density

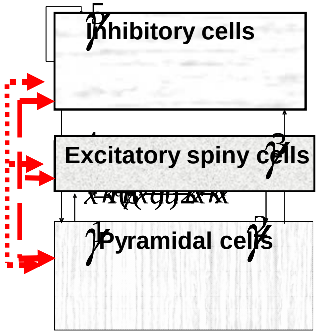


Model Inversion using full complex signal

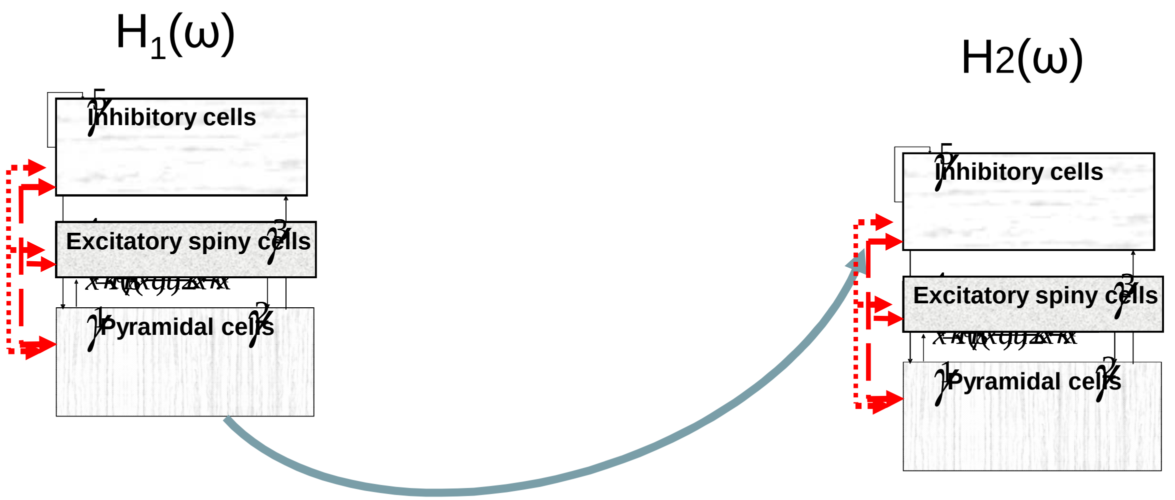
$H_2(\omega) . H_2^*(\omega)$



$H_1(\omega) . H_1^*(\omega)$



$H_1(\omega) . H_2^*(\omega)$



Spectra $\mathrm{Abs}(H_1(\omega) \,.\, H_1^*(\omega))$, $\mathrm{Abs}(H_1(\omega) \,.\, H_2^*(\omega))$ …

Coherence $|(H_1(\omega).H_2^*(\omega))|^2 \,/\, \{ (H_1(\omega).H_1^*(\omega)) + (H_2(\omega).H_2^*(\omega))\}$

Delay at particular frequencies $\arg(H_1(\omega).H_2^*(\omega))/2\pi f$

Covariance (lags over time, collapsed across frequencies) $\mathrm{Real}(\,\mathcal{F}^{-1}(H_1(\omega).H_2^*(\omega)))$

- ❑Can also optimize complex-valued quantities
- ❑ Understand how biophysical parameters affect conventional linear systems measures

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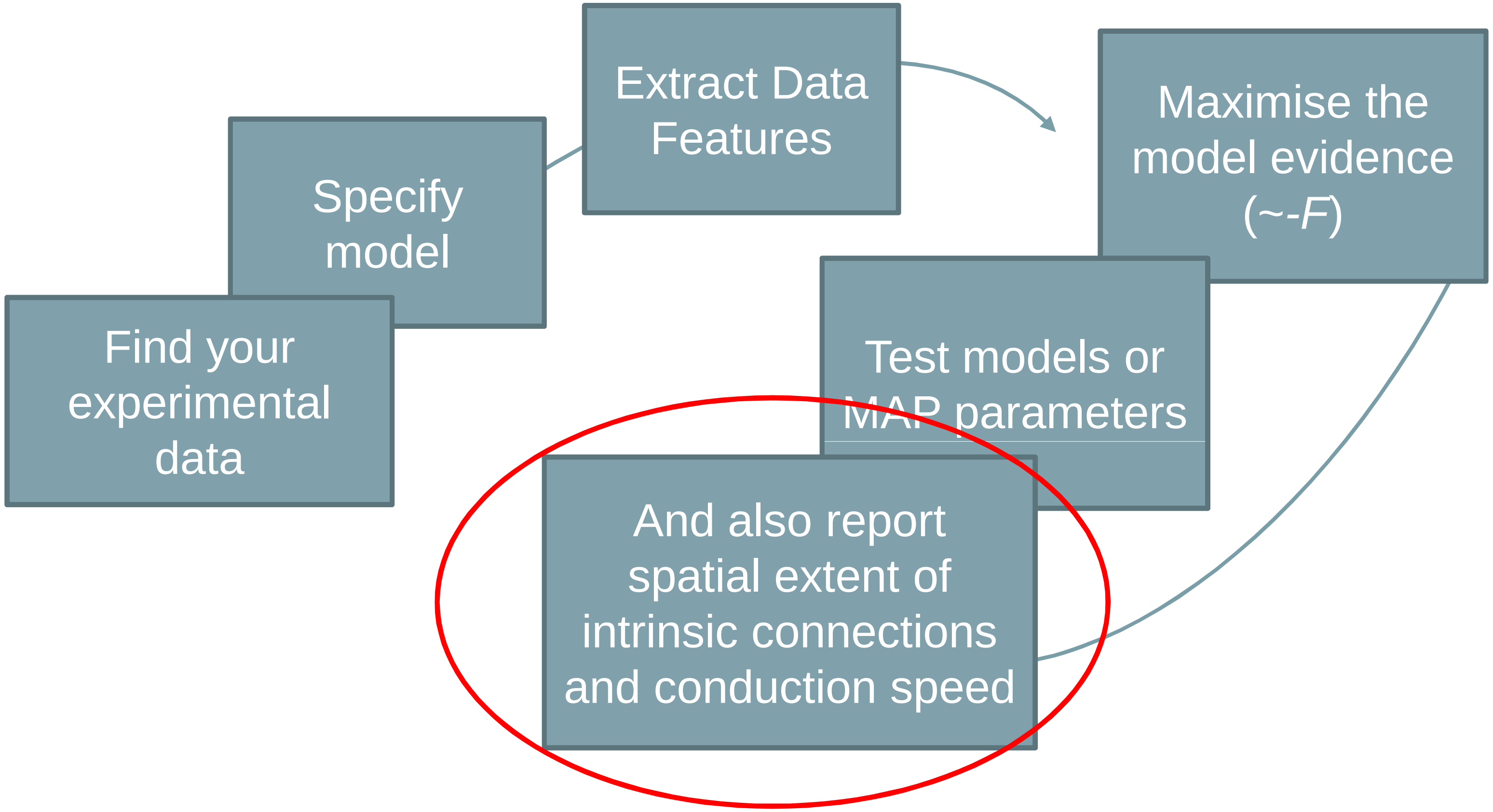
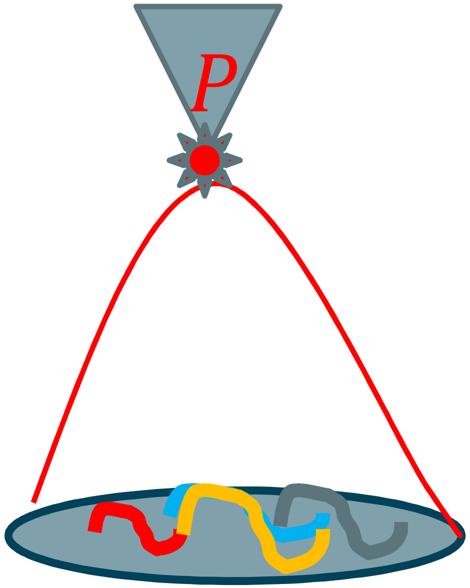


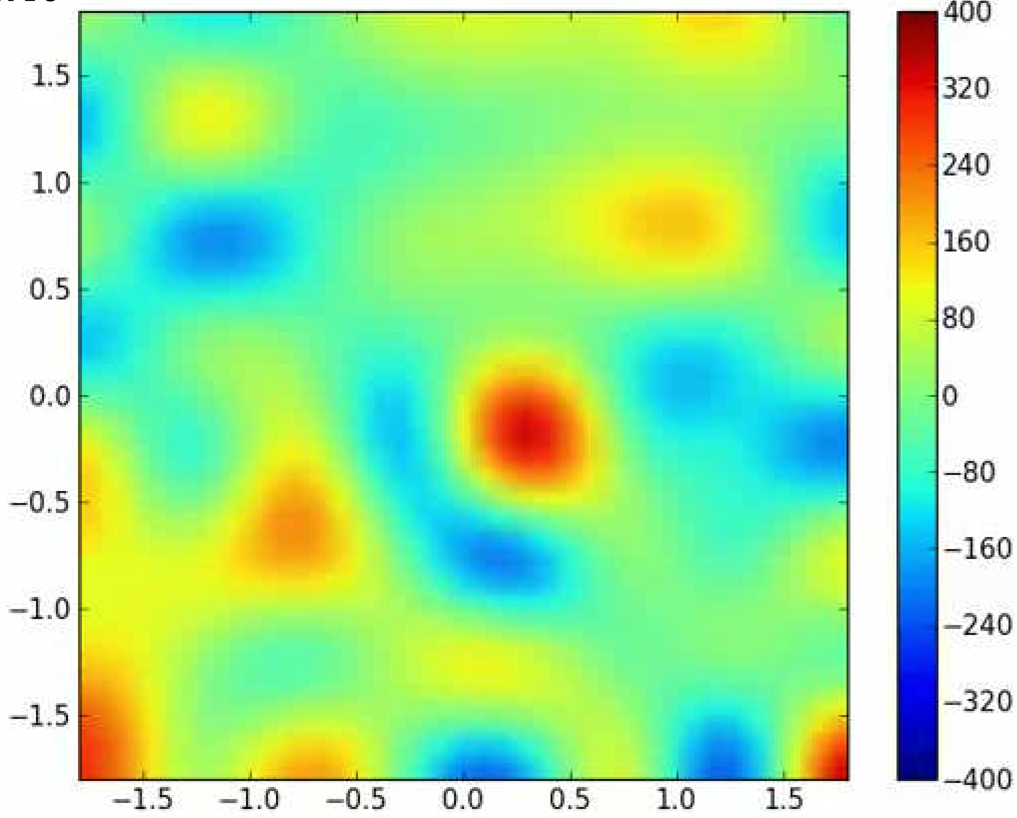
New NFM routines

Pinotsis, Moran, Friston, *Neuroimage 2012*

- Main difference with previous models:
Brain activity is deployed on a cortical *patch* as opposed to being centred around a *point*

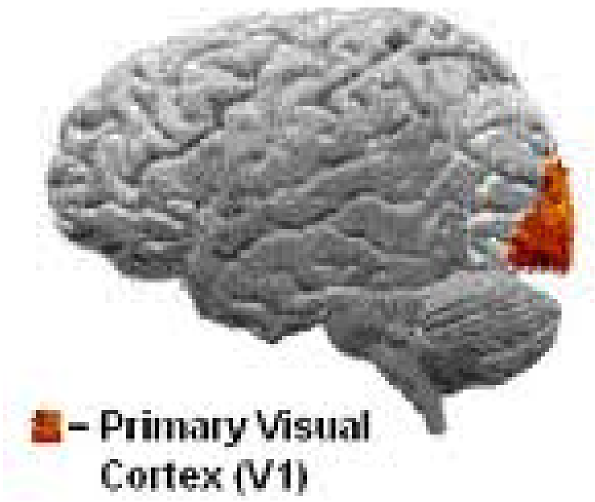


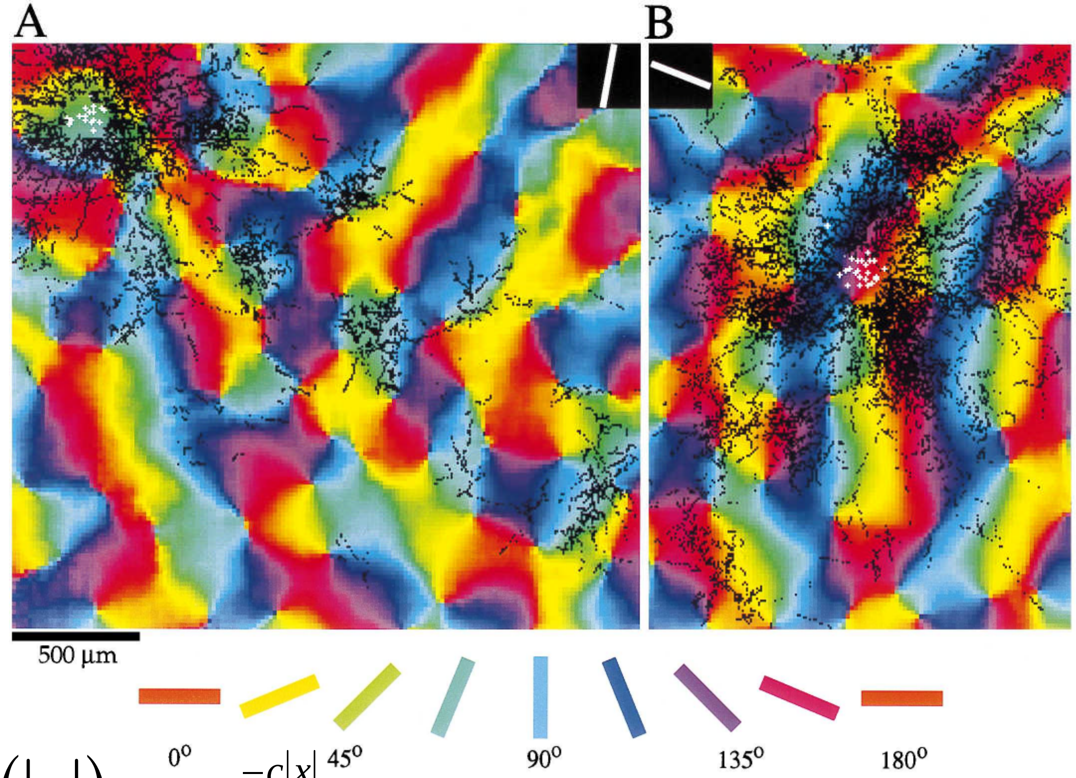
$$L(x,\varphi) = \varphi_1 \exp\left(-\frac{x^2}{\varphi_2}\right)$$

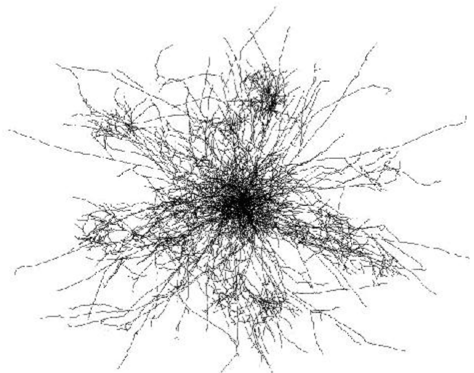


- Lead field modified to enable a mapping of spatially distributed activity (coloured waves) to a time series at *P*

Novelty of DCM for NFs: Can get estimates of parameters relating to spatial properties of sources when there is NO SPATIAL INFO in the data







$$K(|x|) = ae^{-c|x|}$$

a intrinsic connection strength

c spatial decay rate $\leftrightarrow$ connection extent

- Augment old equations with wave equations describing propagation of afferent spike rate between points on the cortex

Inhibitory cells in supragranular layers

$$\ddot{v}_2 + 2\kappa_i \dot{v}_2 + \kappa_i^2 v_2 = \kappa_i m_i \mu_2$$

$$\ddot{\mu}_2 + 2sc_{23}\dot{\mu}_2 - s^2(\mu_{2xx} - c_{23}^2\mu_2) = \alpha_{23}(s^2 c_{23}\sigma(v_3) + s\dot{\sigma}(v_3))$$

a_{32}, c_{32} a_{23}, c_{23}

$U(t)$ →

Excitatory spiny cells in granular layers

$$\ddot{v}_1 + 2\kappa_e \dot{v}_1 + \kappa_e^2 v_1 = \kappa_e m_e(\mu_1 + U)$$

$$\ddot{\mu}_1 + 2sc_{13}\dot{\mu}_1 - s^2(\mu_{1xx} - c_{13}^2\mu_1) = \alpha_{13}(s^2 c_{13}\sigma(v_3) + s\dot{\sigma}(v_3))$$

a_{13}, c_{13} a_{31}, c_{31}

Excitatory pyramidal cells in supra- and infragranular layers

$$\ddot{v}_3 + 2\kappa_e \dot{v}_3 + \kappa_e^2 v_3 = \kappa_e m_e \mu_3$$

$$\ddot{\mu}_3 + 2sc_{31}\dot{\mu}_3 - s^2(\mu_{3xx} - c_{31}^2\mu_3) = \alpha_{31}\left(s^2 c_{31}(\sigma(v_1) - \sigma(v_2)) + s\left(\dot{\sigma}(v_1) - \dot{\sigma}(v_2)\right)\right)$$

Endogenous output $v_3(t)$

Exogenous input $U(t)$

Hidden states

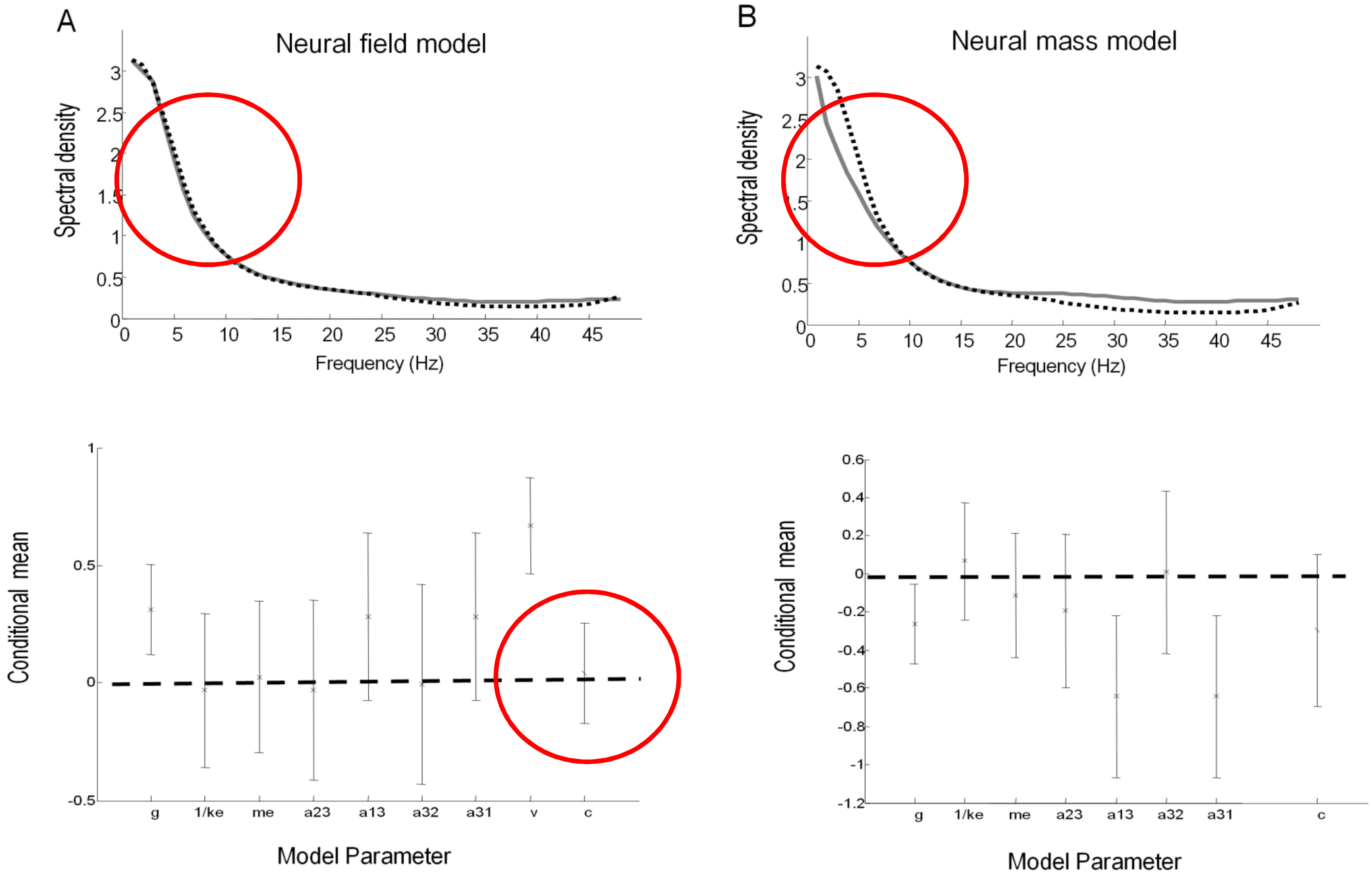
$$\dot{V}(x,t) = f(V,U)$$

Observed signals

$$y(t) = \int L(x,\varphi)\cdot V(x,t)\,dx$$

s conduction velocity

Inference on Model Parameters



Summary

- DCM is a generic framework for asking mechanistic questions based on neuroimaging data (e.g. drug-induced changes in balance of synaptic transmission)

- Neural mass models parameterise intrinsic and extrinsic ensemble connections and synaptic measures (time constants, effective connectivity,…)

- DCM for SSR and CSD provide a compact characterisation of multi- channel LFP or EEG data in the frequency domain

- Bayesian inversion provides parameter estimates and allows model comparison for competing hypothesised architectures

- Neural field models incorporate propagation of activity on a cortical patch, so one can distinguish between spatial effects and other factors such as cortico-thalamic interactions or intrinsic cell properties

- Neural field models yield estimates of parameters related to topographic properties of the sources such as spatial decay rate of synaptic connections and intrinsic conduction speed, even when using spatially unresolved data

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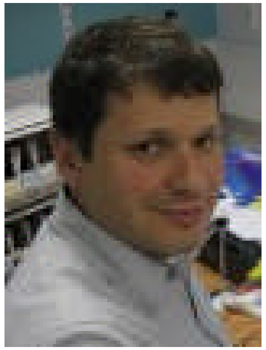
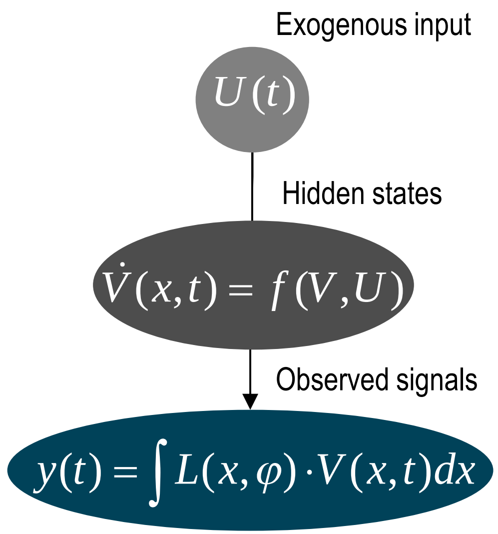
Jeremie Mattout
Rosalyn Moran
Ashwini Oswal
Will Penny
Ged Ridway
Klaas Stephan
Yen Yu



...and thank you !

$$L(x,\varphi) = \varphi_1 \exp\left(-\frac{x^2}{\varphi_2}\right)$$

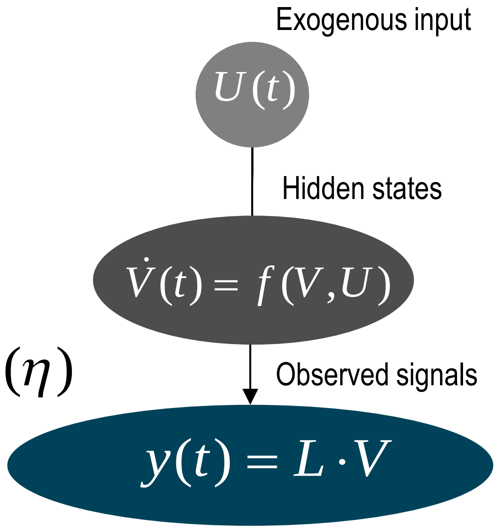


$$\mathbf{g}_Y(\omega) = g_Y(\omega,\theta) + g_N(\omega,\theta) + \varepsilon(\omega)$$

$$g_Y(\omega,\theta) \approx \frac{\pi}{\ell} \sum_j L\left(\frac{j\pi}{\ell}\right) T_m\left(\frac{j\pi}{\ell},\omega\right) g_U\left(\frac{j\pi}{\ell},\omega\right) T_{m'}\left(\frac{j\pi}{\ell},\omega\right)^* L\left(\frac{j\pi}{\ell}\right)^*$$

$$g_N(\omega,\theta) = \alpha_N + \frac{\beta_N}{\omega} \qquad g_U(k,\omega) = \alpha_U + \frac{\beta_U}{\omega}$$

$$\mathrm{Re}(\varepsilon) \sim \mathcal{N}(0,\Sigma(\omega,\lambda)) \qquad \mathrm{Im}(\varepsilon) \sim \mathcal{N}(0,\Sigma(\omega,\lambda))$$



$$\mathbf{g}_Y(\omega) = g_Y(\omega, \theta) + g_N(\omega, \theta) + \varepsilon(\omega)$$

$$L(x, \varphi) = L(\eta)$$

$$g_Y(\omega, \theta) \approx \sum_k L(\eta) T_m^k(\omega, \theta) g_U(\omega) T_{m'}(\omega, \theta)^* L(\eta)^*$$

$$g_N(\omega, \theta) = \alpha_N + \frac{\beta_N}{\omega}$$

$$g_U(k, \omega) = \alpha_U + \frac{\beta_U}{\omega}$$

$$T_m^k(\omega, \theta) = \int \kappa_m^k(t, \theta) e^{-j\omega t} dt$$

$$\kappa_m^k(t, \theta) = \frac{\partial g}{\partial x} e^{\Im \tau} \Im^{-1} \frac{\partial f}{\partial u_k}$$

$$\mathrm{Re}(\varepsilon) \sim \mathcal{N}(0, \Sigma(\omega, \lambda)) \qquad \mathrm{Im}(\varepsilon) \sim \mathcal{N}(0, \Sigma(\omega, \lambda))$$

Maximum postsynaptic depolarization
8, 32 (mV)

Postsynaptic time constants
1/4, 1/28 (ms^{-1})

Amplitude of intrinsic connectivity kernels
2000, 8000, 2000, 1000

Intrinsic connectivity decay constant
0.32 (mm^{-1})

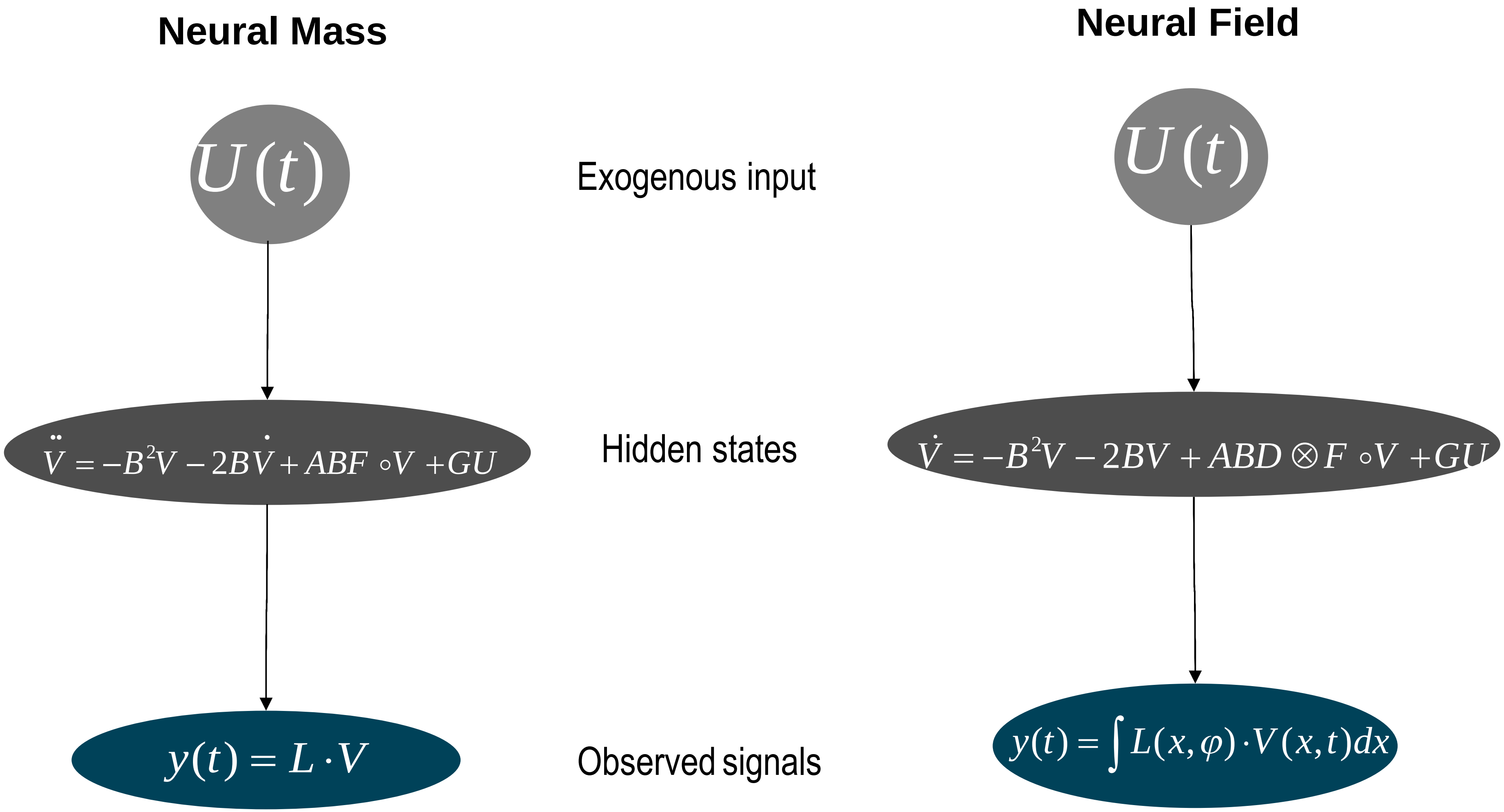
Sigmoid parameters(post synaptic firing rate function)
0.54, 0,0.135

Conduction velocity
3 m/s

Radius of cortical source
50 (mm)

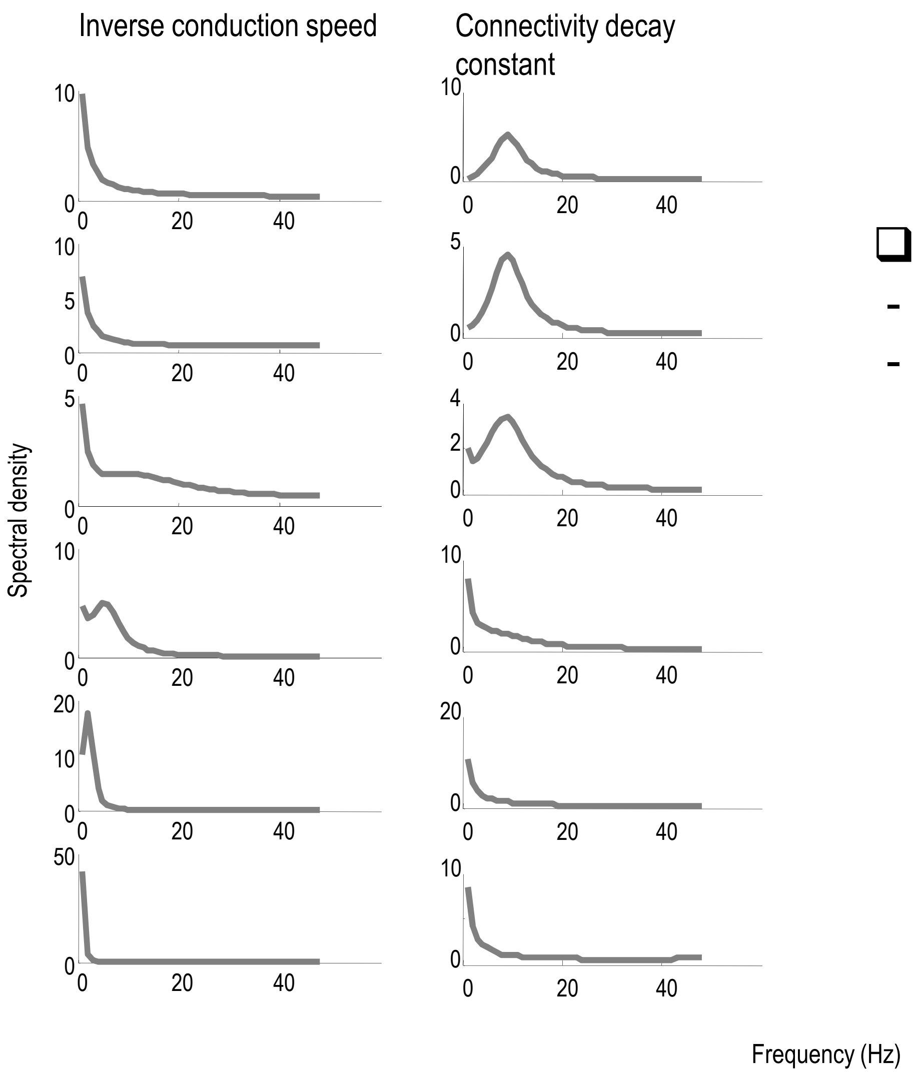
Neural Mass vs Neural Field

Difference in predicted spectra $g_Y(\omega, \theta)$ because of difference in underlying model:



$$D \otimes Q = \iint D(x - x', t - t') \cdot Q(x', t')dx'dt'$$

Changing model parameters



- New peaks appear:
  - as intrinsic speed decreases
  - as connectivity extent increases

Pinotsis, Moran, Friston, *Neuroimage 2012*

Roadmap

UCL

- Generative model
  - predicted responses
  - parameters
  - hyperparameters
- Priors
  - variance
- Bayesian Inference
  - Model Evidence
  - Posteriors

$$\mathbf{g}_Y(\omega) = g_Y(\omega,\theta) + g_N(\omega,\theta) + \varepsilon(\omega)$$

$$g_N(\omega,\theta) = \alpha_N + \frac{\beta_N}{\omega}$$

$$\mathrm{Re}(\varepsilon) \sim \mathcal{N}(0,\Sigma(\omega,\lambda)) \qquad \mathrm{Im}(\varepsilon) \sim \mathcal{N}(0,\Sigma(\omega,\lambda))$$

+

$$p(\theta,m) = N(\mu_\theta,\Sigma_\theta)$$

=

$$p(G\,|\,\theta,m) = N(\mathbf{g}_Y(\omega),\Sigma(\omega,\lambda))$$

$$p(G\,|\,m) = \int p(G\,|\,\theta,m)\,p(\theta)\,d\theta$$

$$p(\theta\,|\,G,m) = \frac{p(G\,|\,\theta,m)\,p(\theta,m)}{p(G\,|\,m)}$$

Measured data

Specify generative forward model (with prior distributions of parameters)

Variational Laplace Algorithm

Maximize a free energy bound to model evidence :

$$F = \log p(y|m) - D(q(\theta) \| p(\theta|y,m))$$

$$= < \log p(y|\theta,m) >_q - D(q(\theta) \| p(\theta|m))$$

<u>Iterative procedure:</u>

1. Compute model response using current set of parameters and hyperparameters
2. Compare model response with data
3. Improve parameters and hyperparameters

Model comparison via Bayes factor:

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

$$q(\theta) \approx p(\theta|y,m)$$

Maximum accuracy over complexity constraints