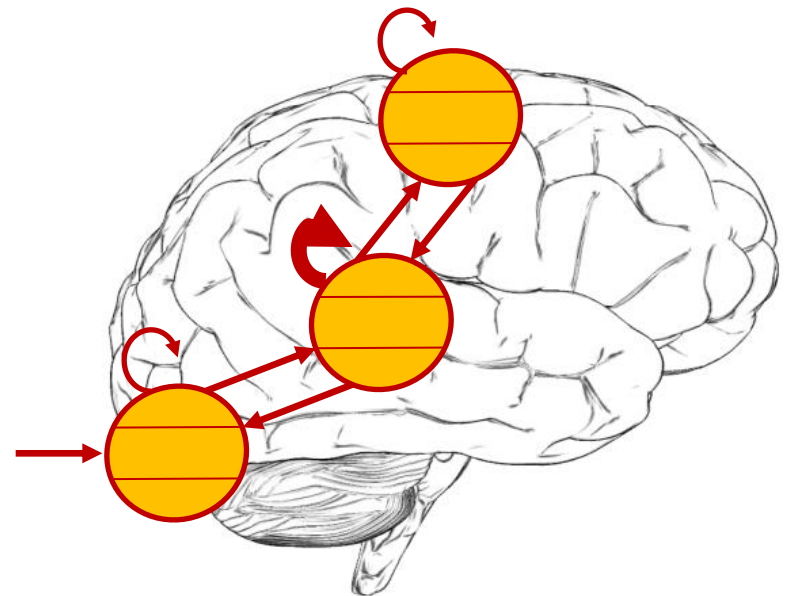


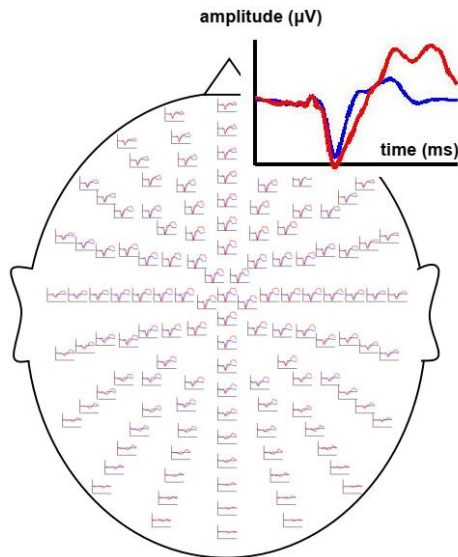
DCM for evoked responses

Ryszard Aukštulewicz

SPM for M/EEG course



Modelling aim and approach



*M/EEG are complex data

Analyses / modelling: in time, frequency,
time-frequency and space domains

Modelling Aim

Explain all data with few
parameters

How to ...

Assume data are caused by few
interacting brain sources ...

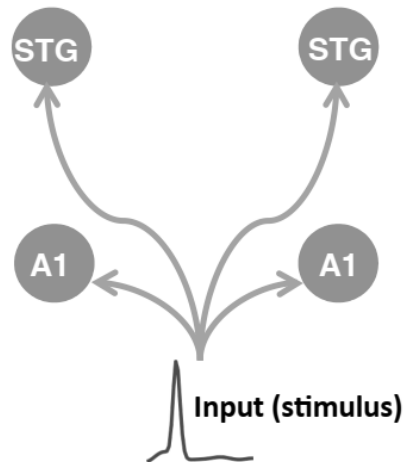
... and / or their respective intrinsic
connectivity / intrinsic parameters

Conventional analyses vs. DCM

“Conventional” Analysis

Which regions are involved in a task?

Sensor space / functional connectivity

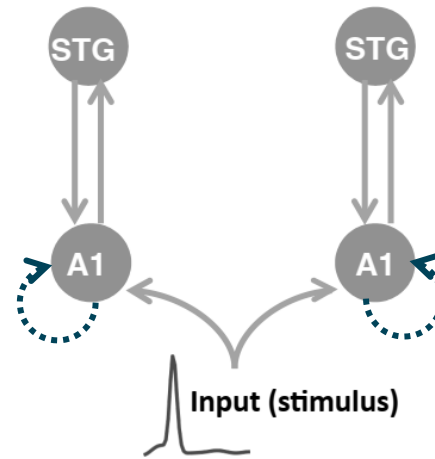


A1 - left and right primary auditory cortex
STG - left and right superior temporal gyrus

DCM Analysis

How do regions communicate?
What role do intrinsic connections have?

Source space / effective connectivity



Does network XYZ explain my data better than network XY?

Which XYZ connectivity structure best explains my data?

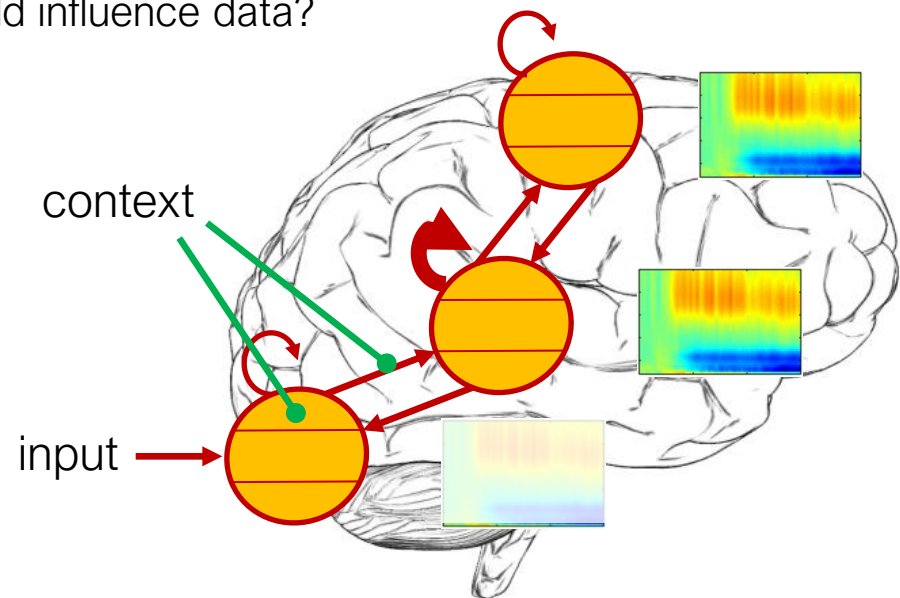
Are X & Y linked in a bottom-up, top-down or recurrent fashion?

Is my effect driven by extrinsic or intrinsic connections?

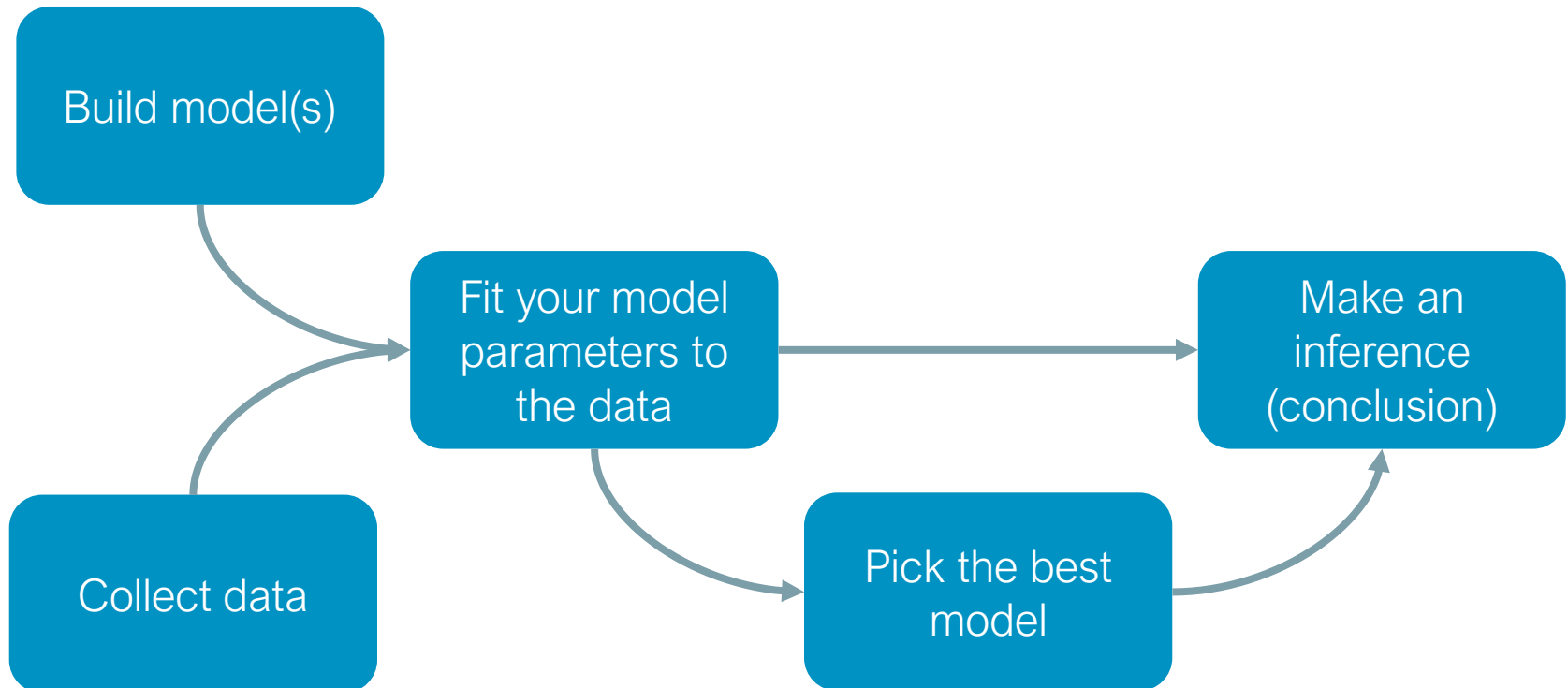
Which neural populations are affected by contextual factors?

Which connections determine observed frequency coupling?

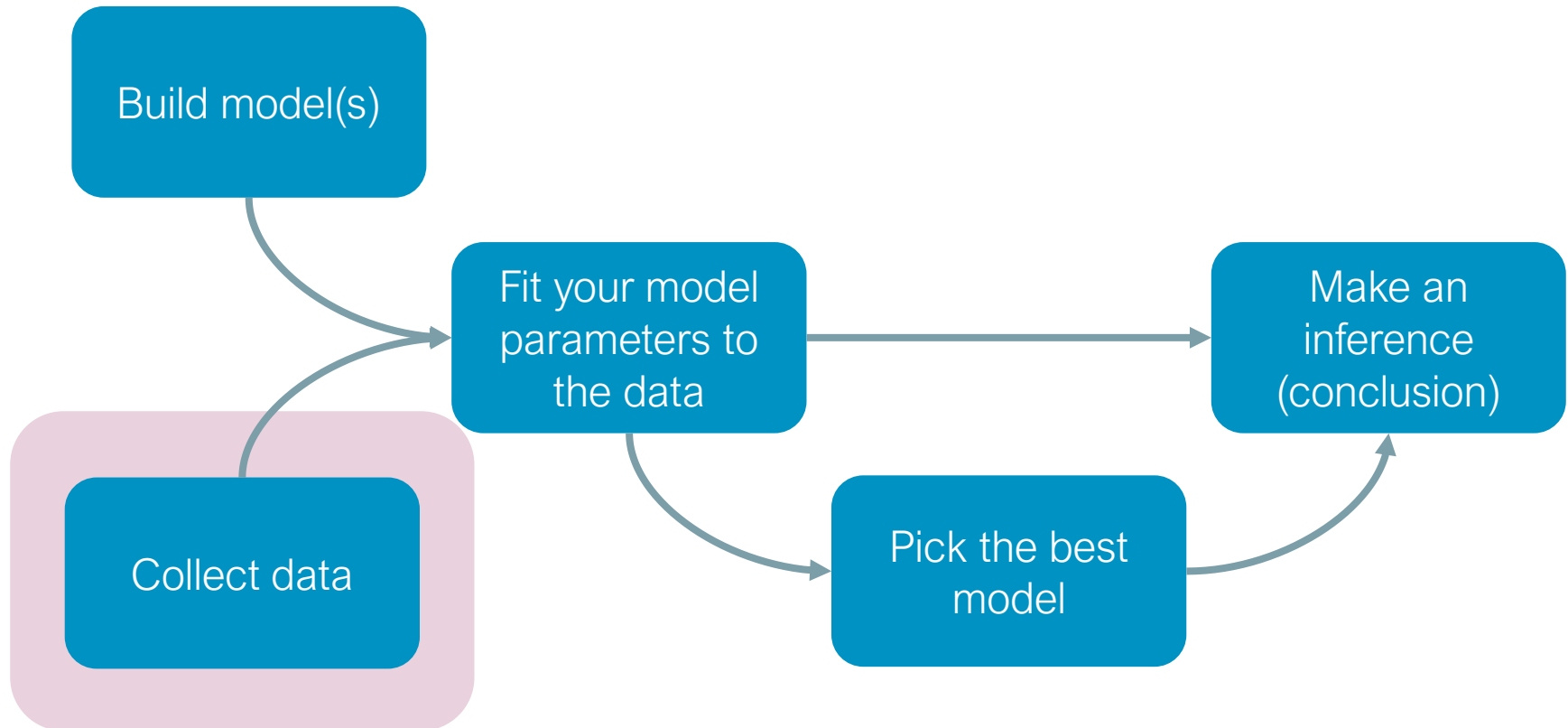
How changing a connection/parameter would influence data?



The DCM analysis pathway

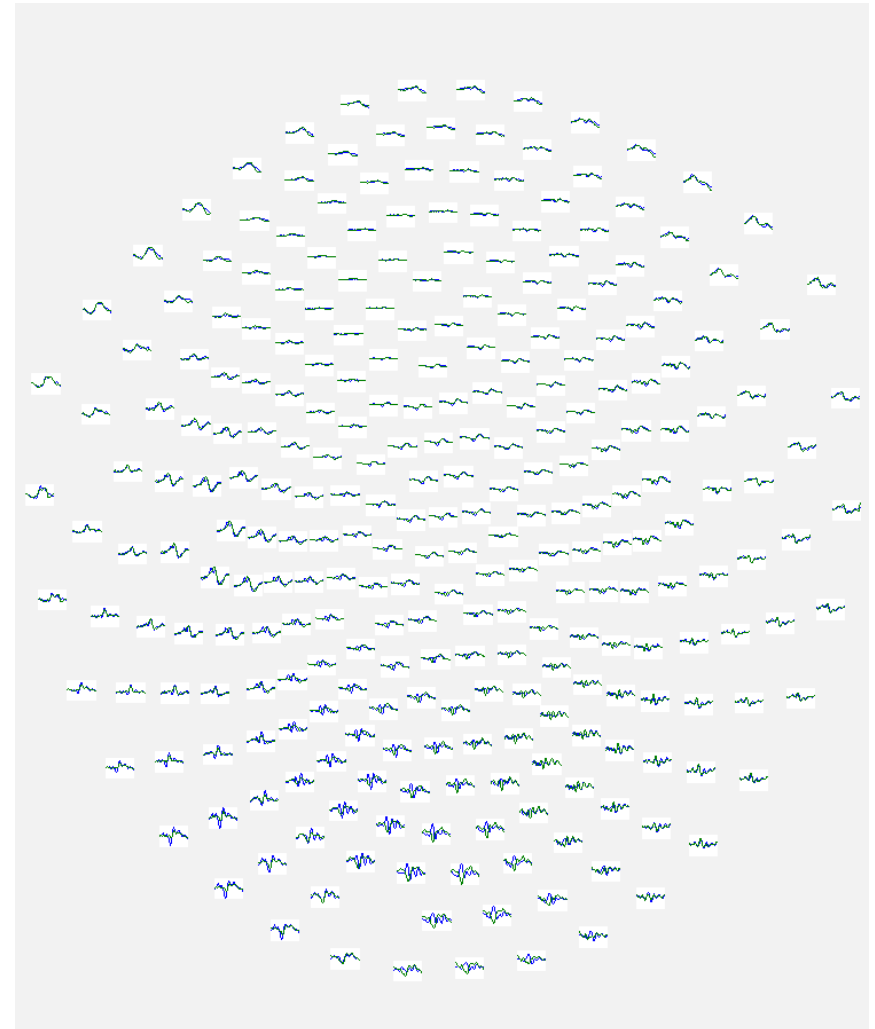


The DCM analysis pathway

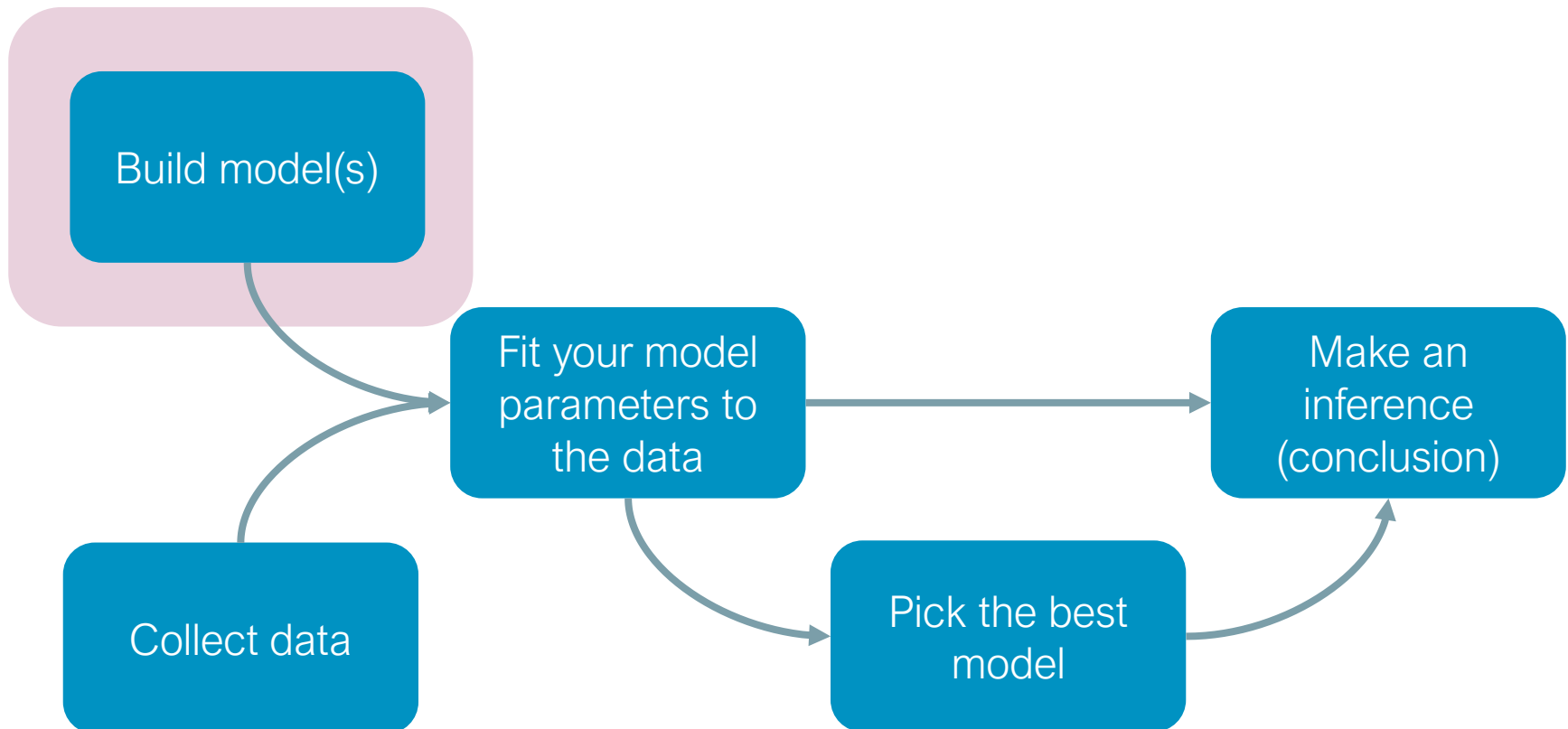


Data for DCM for ERPs / ERFs

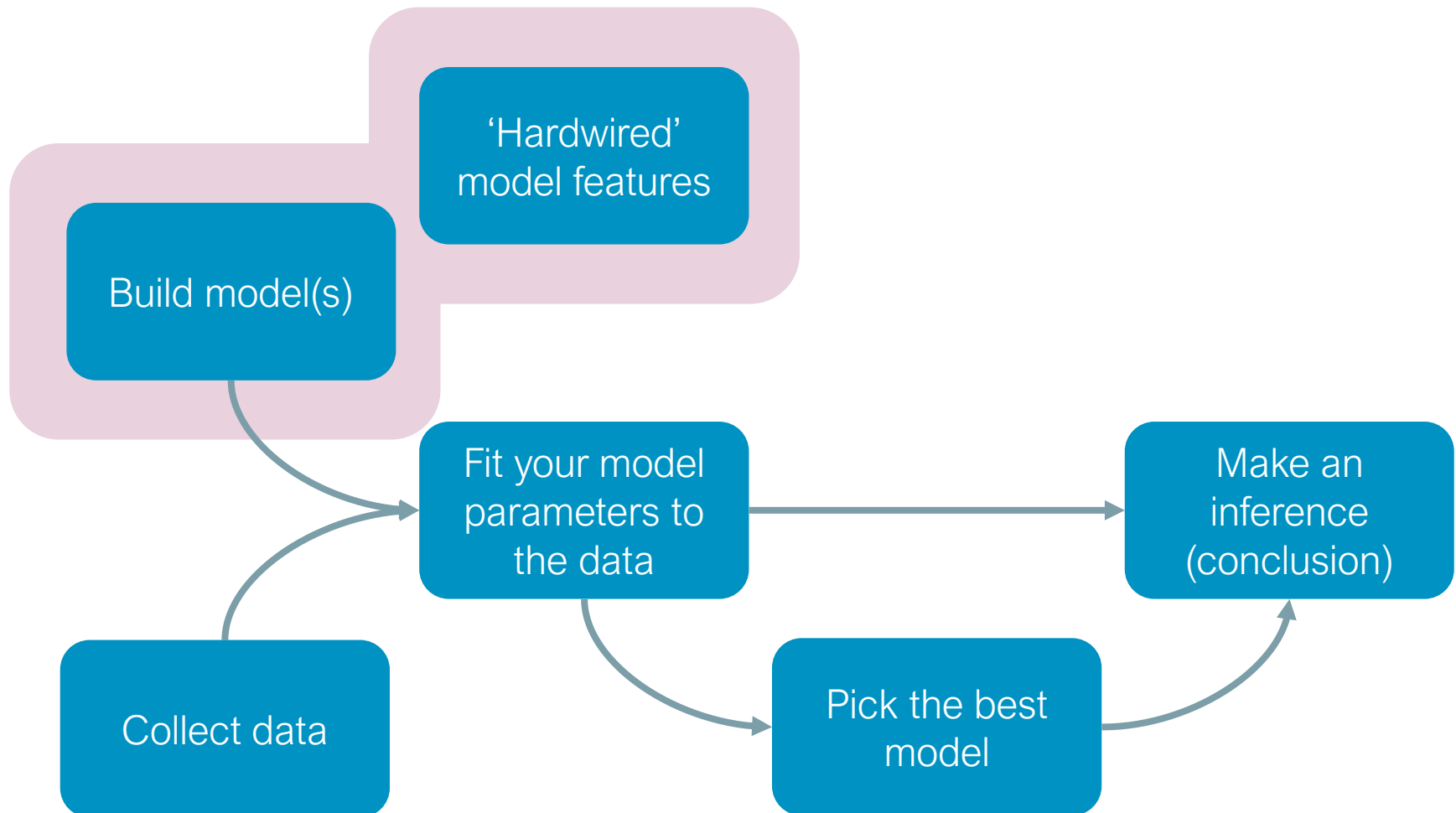
1. Downsample
2. Filter (e.g. 1-40Hz)
3. Epoch
4. Remove artefacts
5. Average
 - Per subject
 - Grand average
6. Plausible sources
 - Literature / a priori
 - Dipole fitting
 - Source reconstruction



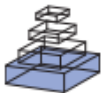
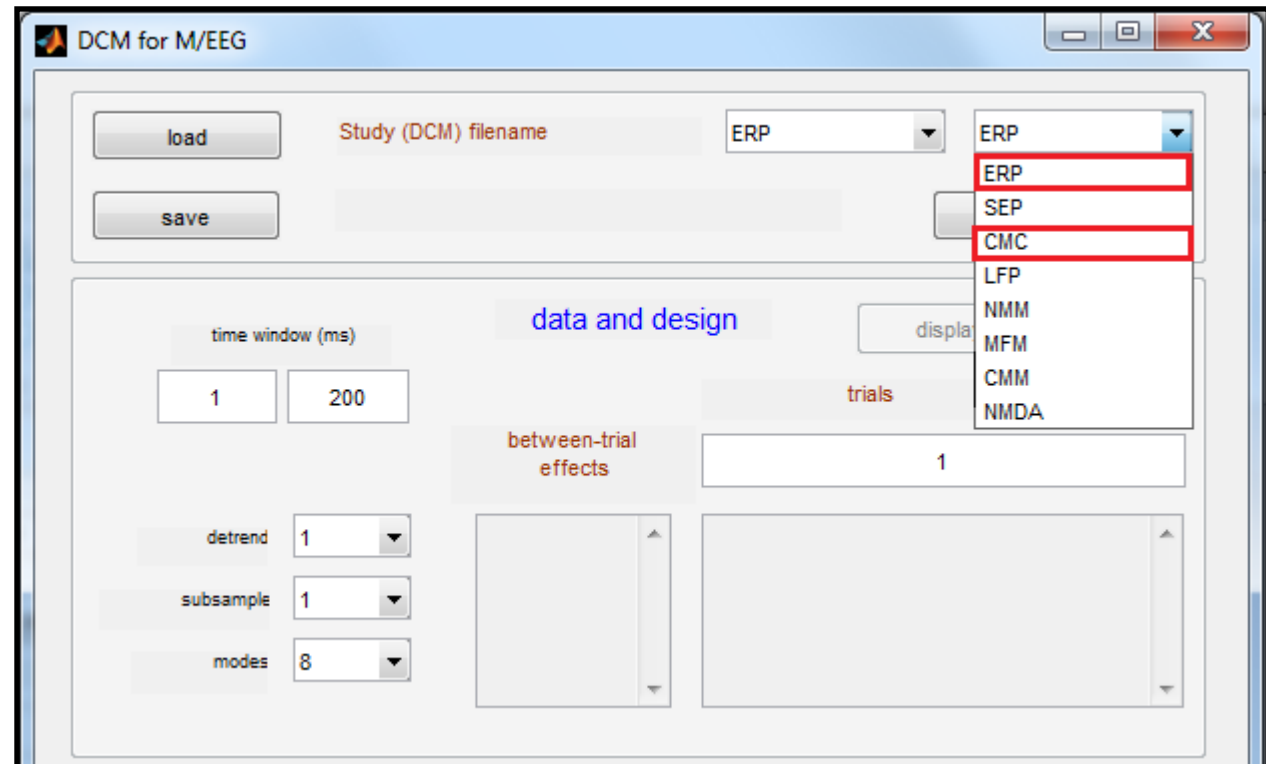
The DCM analysis pathway



The DCM analysis pathway



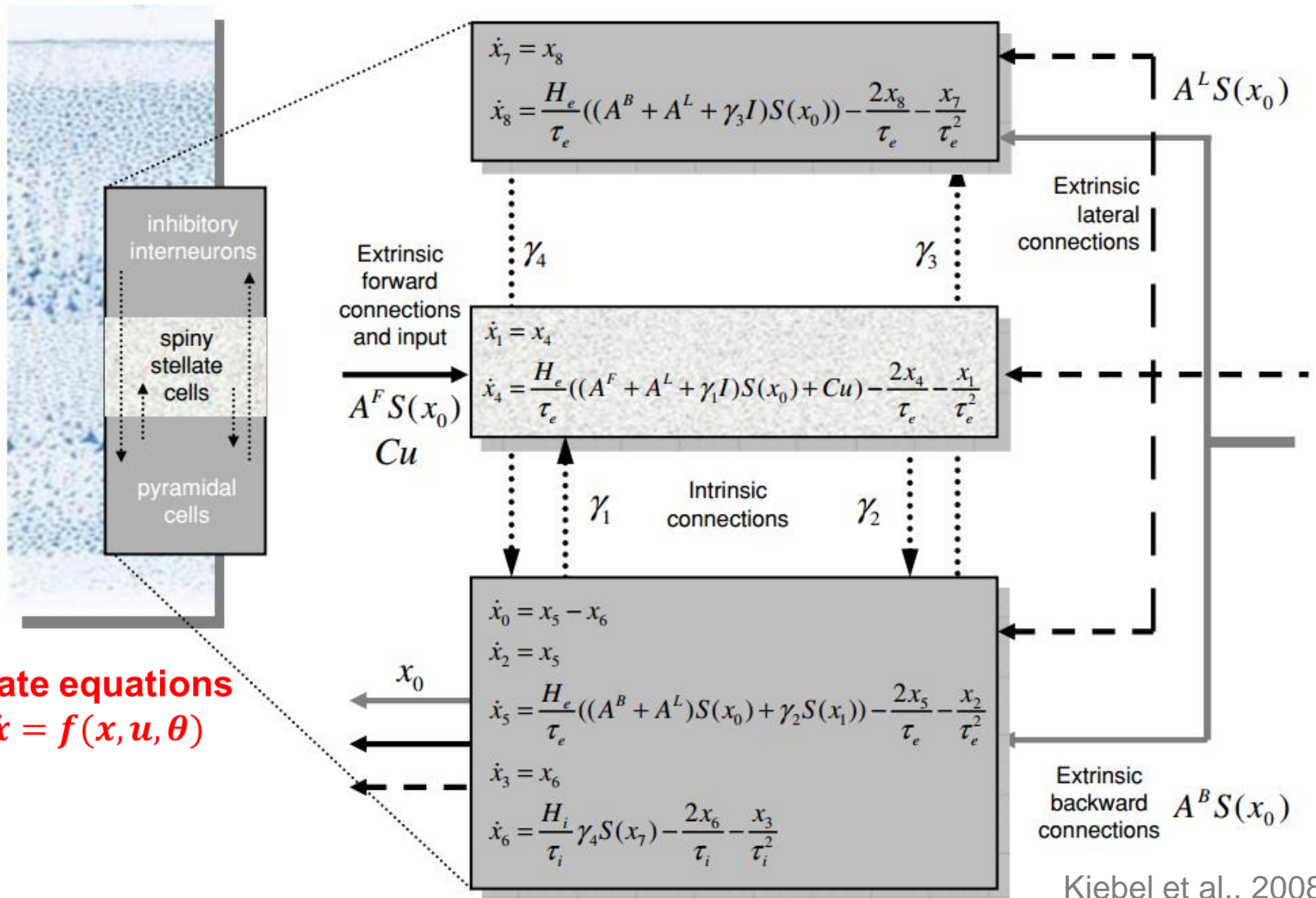
Models

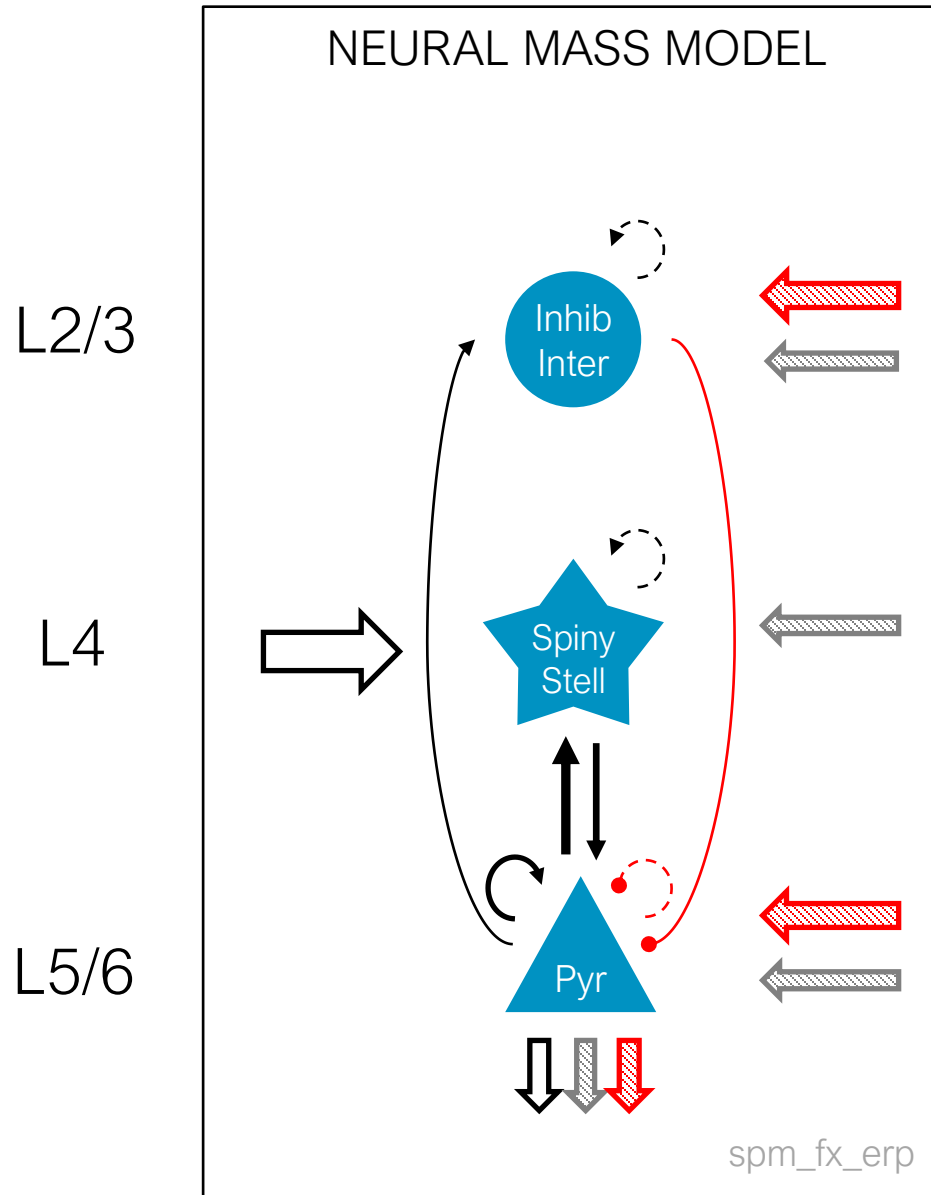


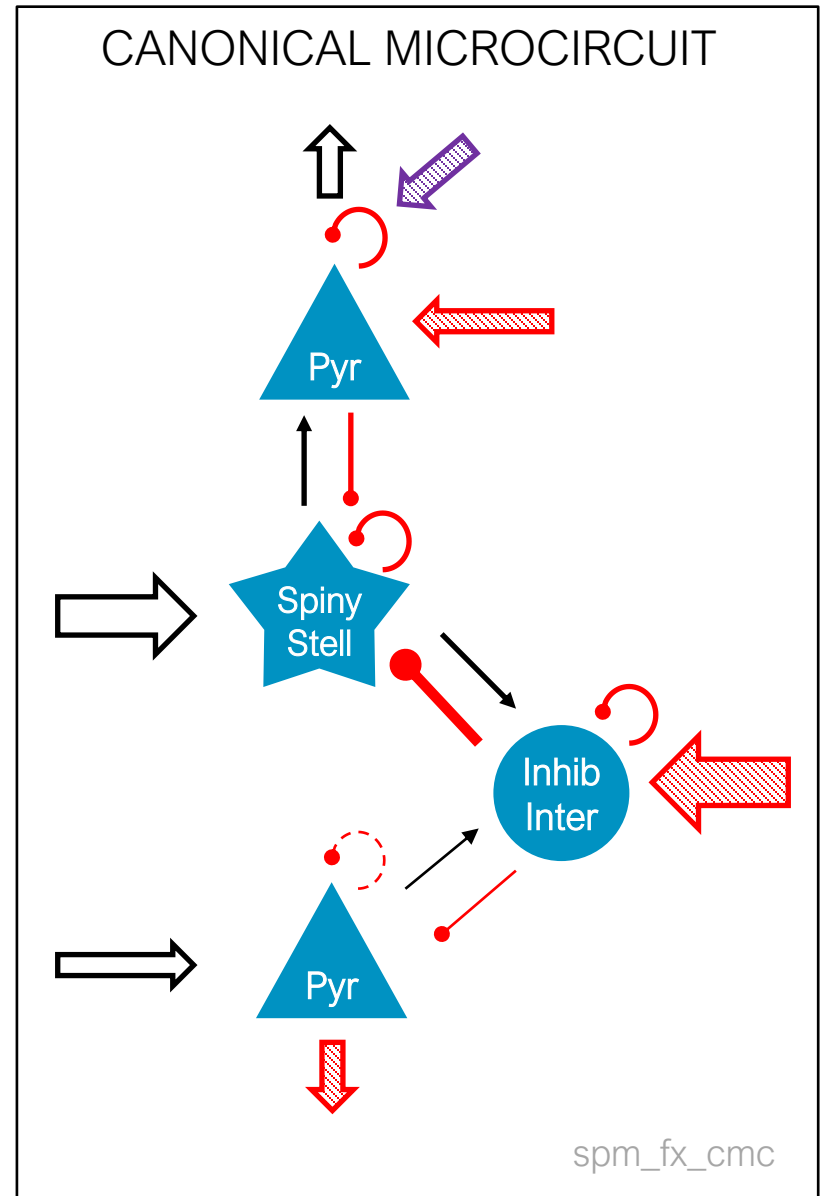
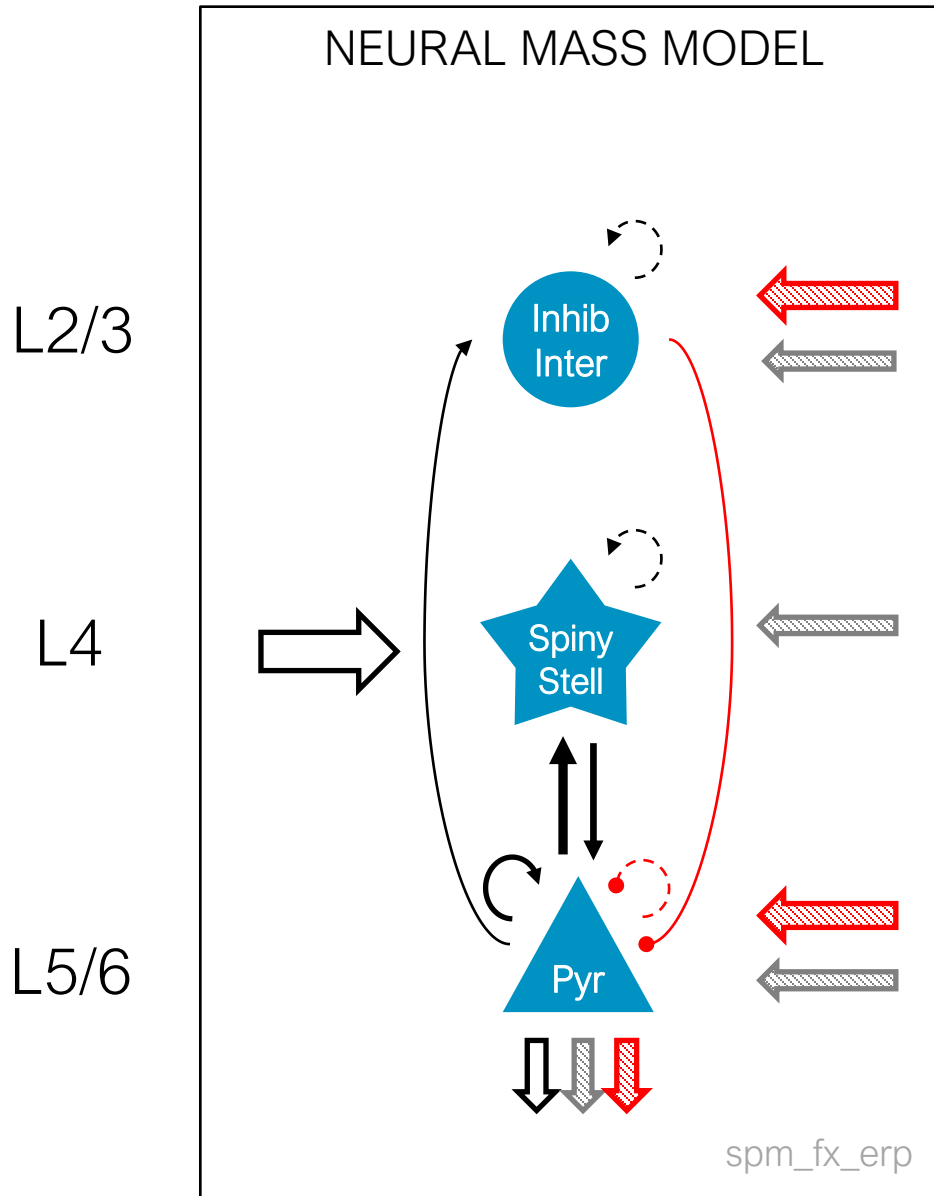
Neural masses and fields in dynamic causal modeling

Rosalyn Moran^{1,2,3*†}, *Dimitris A. Pinotsis*^{1†} and *Karl Friston*¹

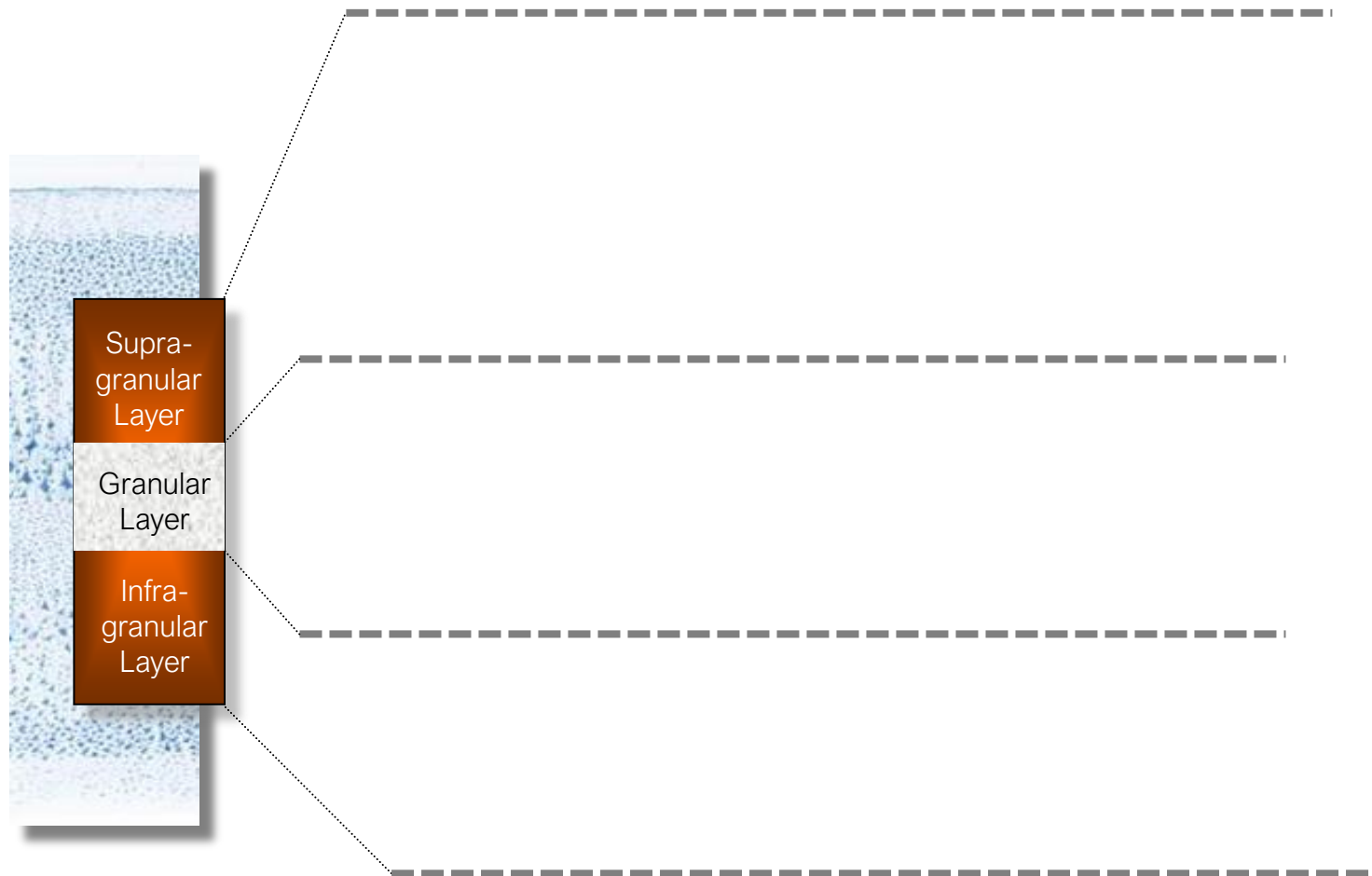
Neuronal (source) model



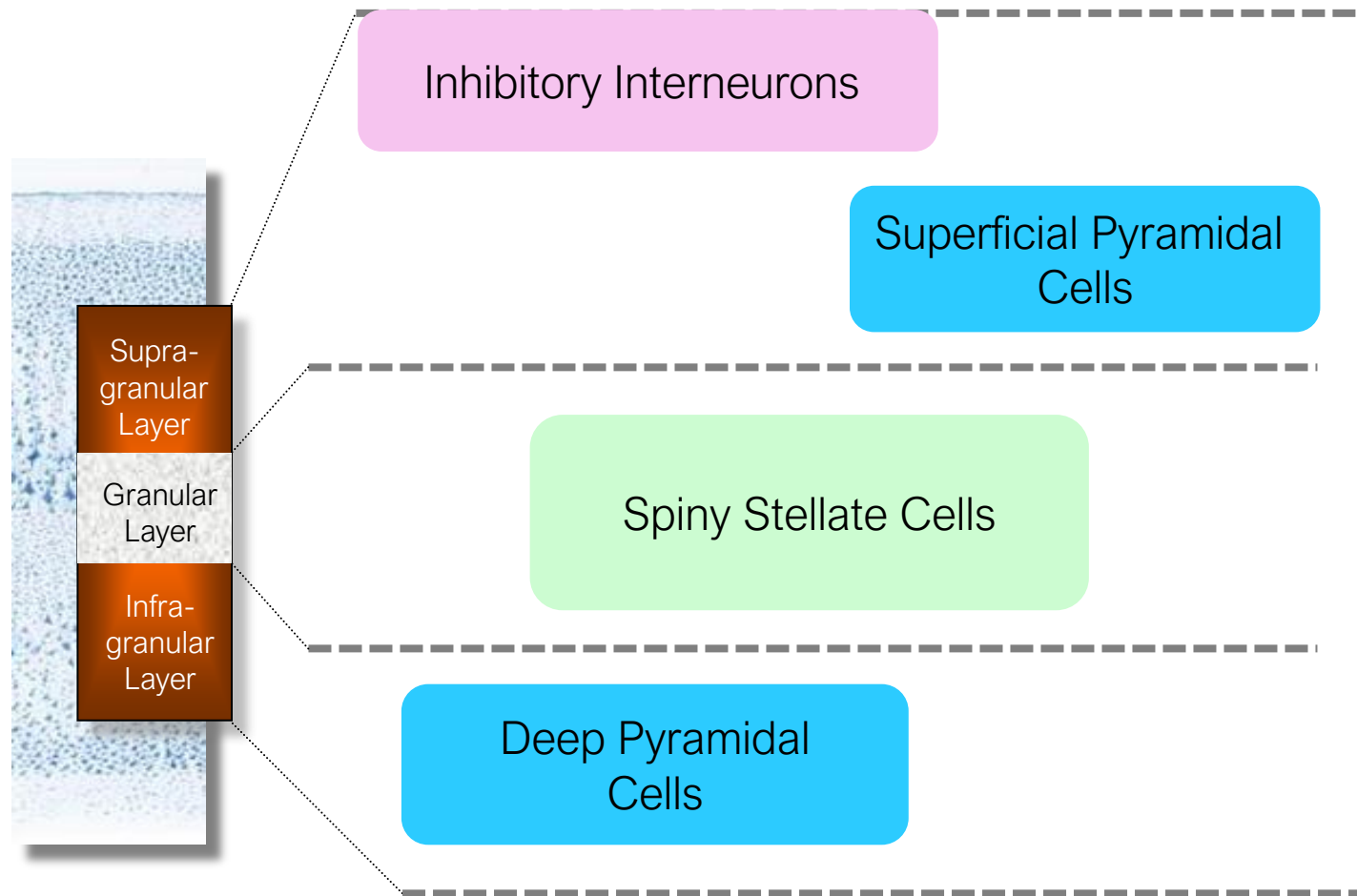




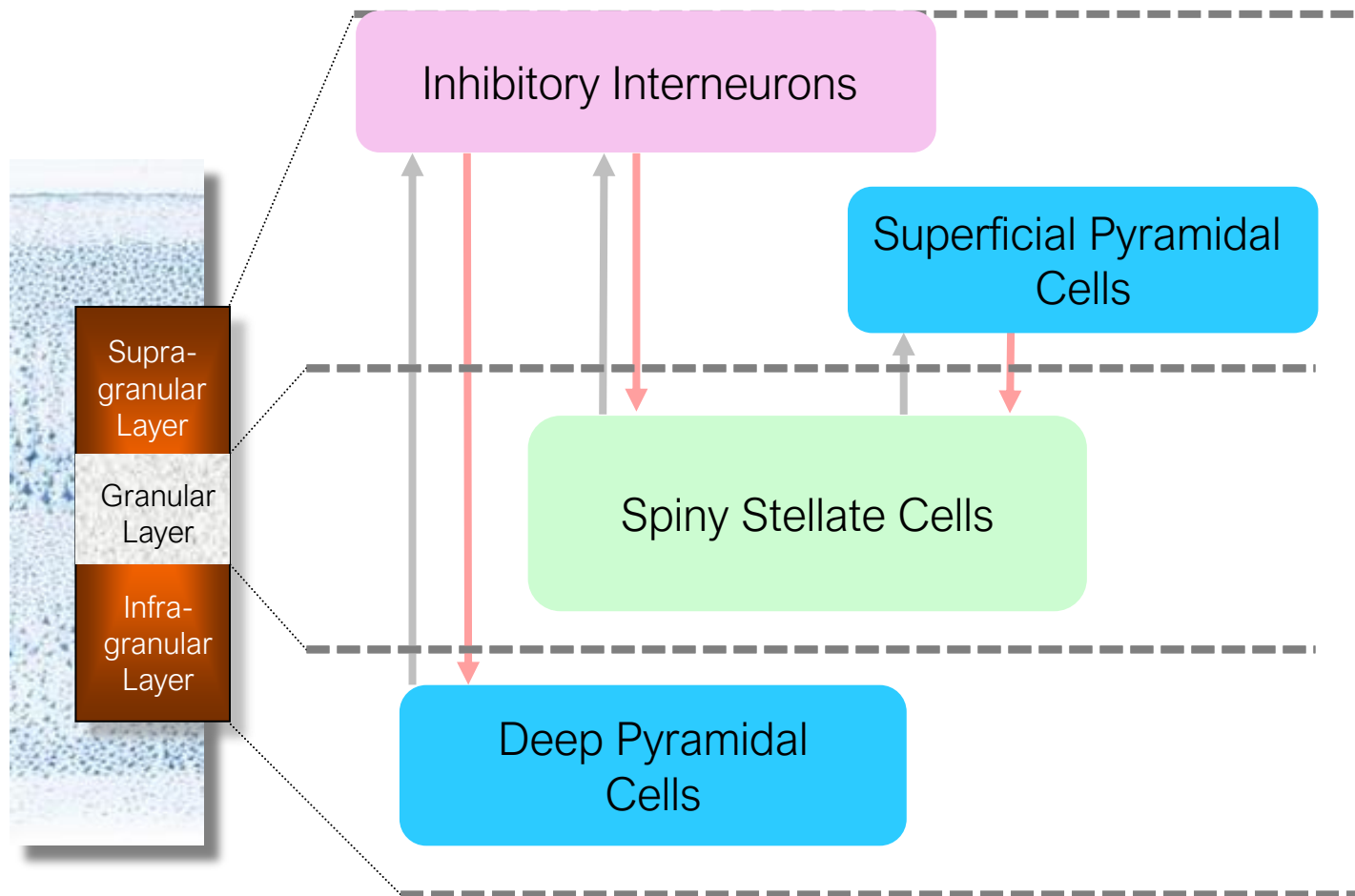
Canonical Microcircuit Model ('CMC')



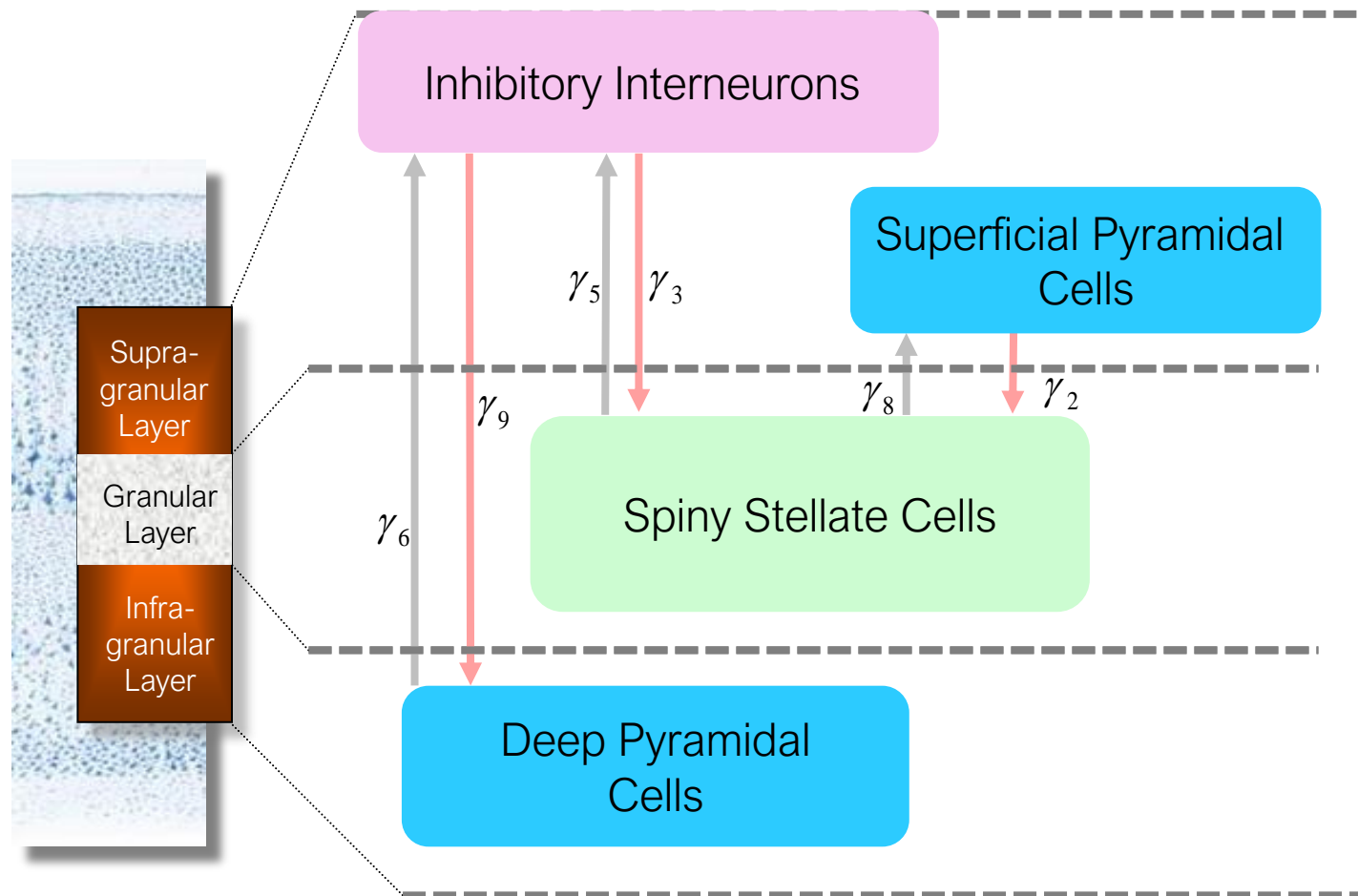
Canonical Microcircuit Model ('CMC')



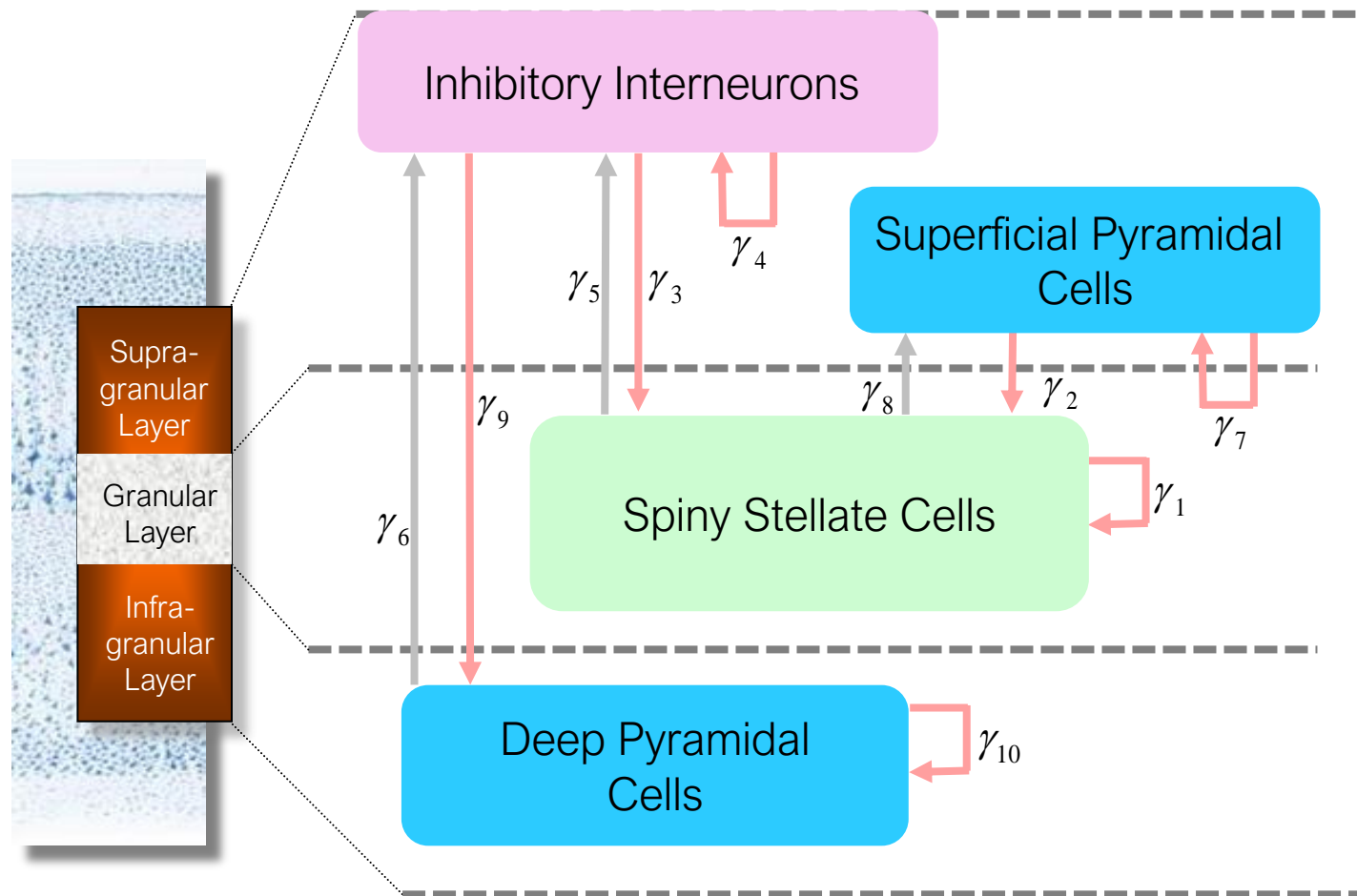
Canonical Microcircuit Model ('CMC')



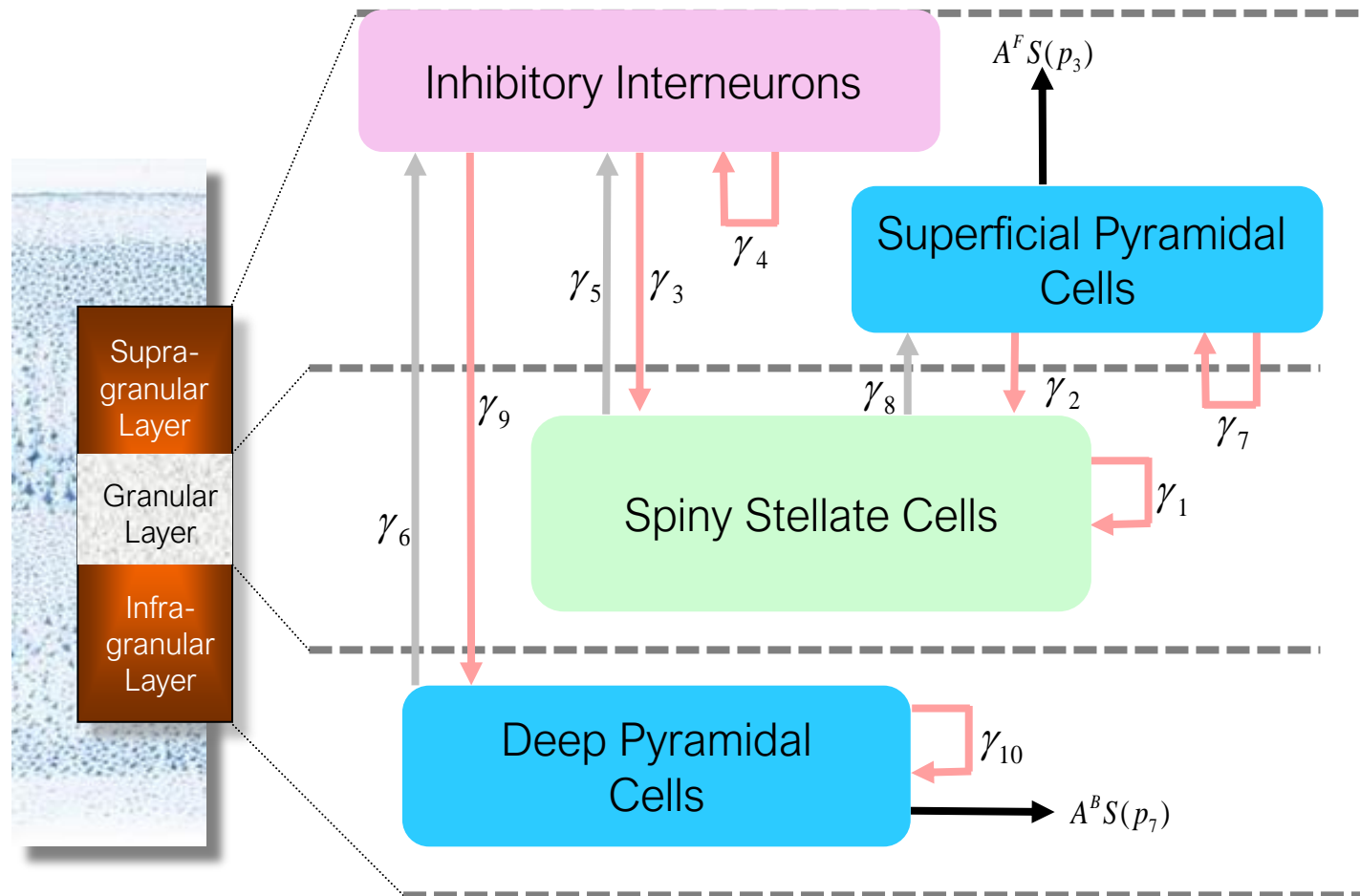
Canonical Microcircuit Model ('CMC')



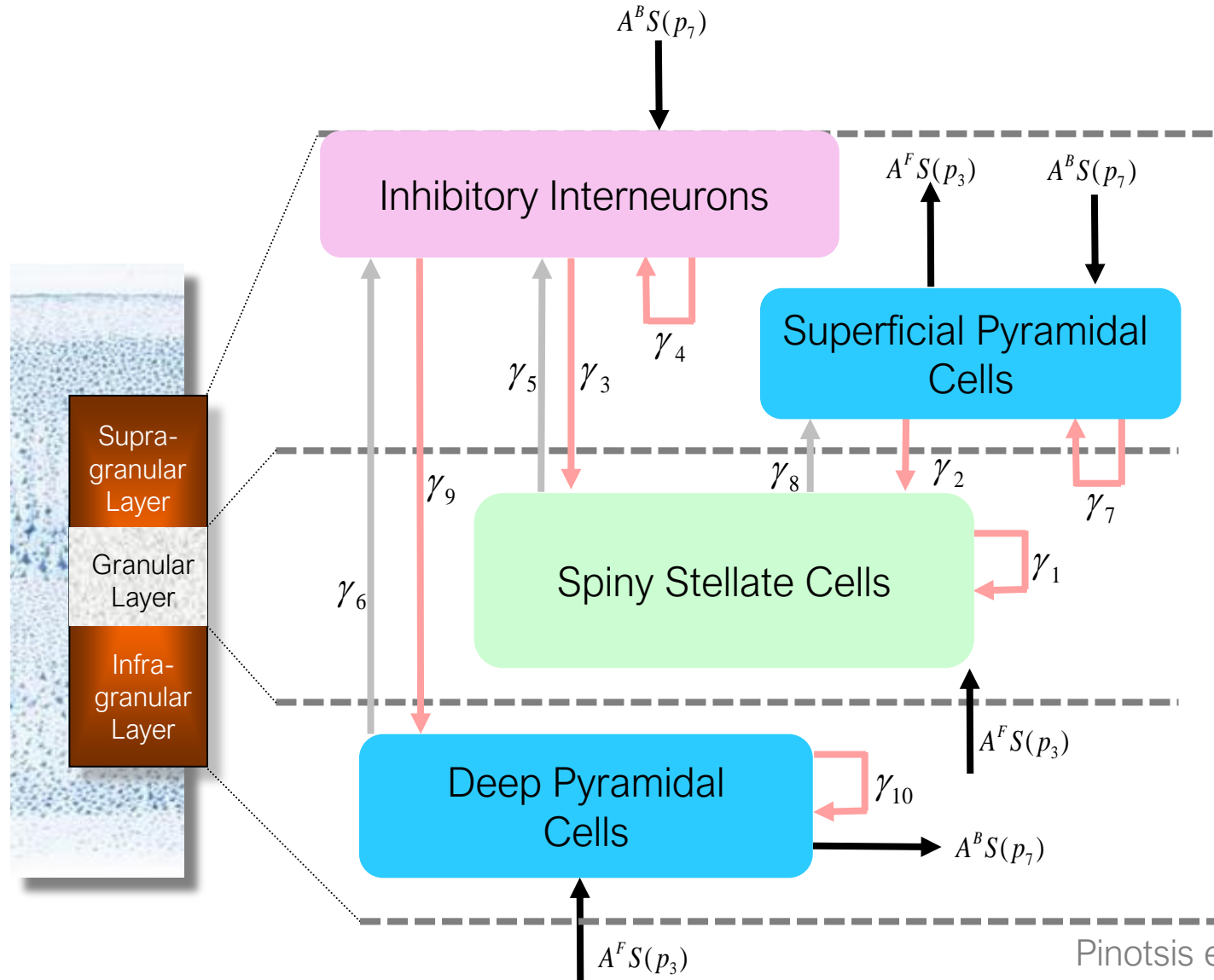
Canonical Microcircuit Model ('CMC')



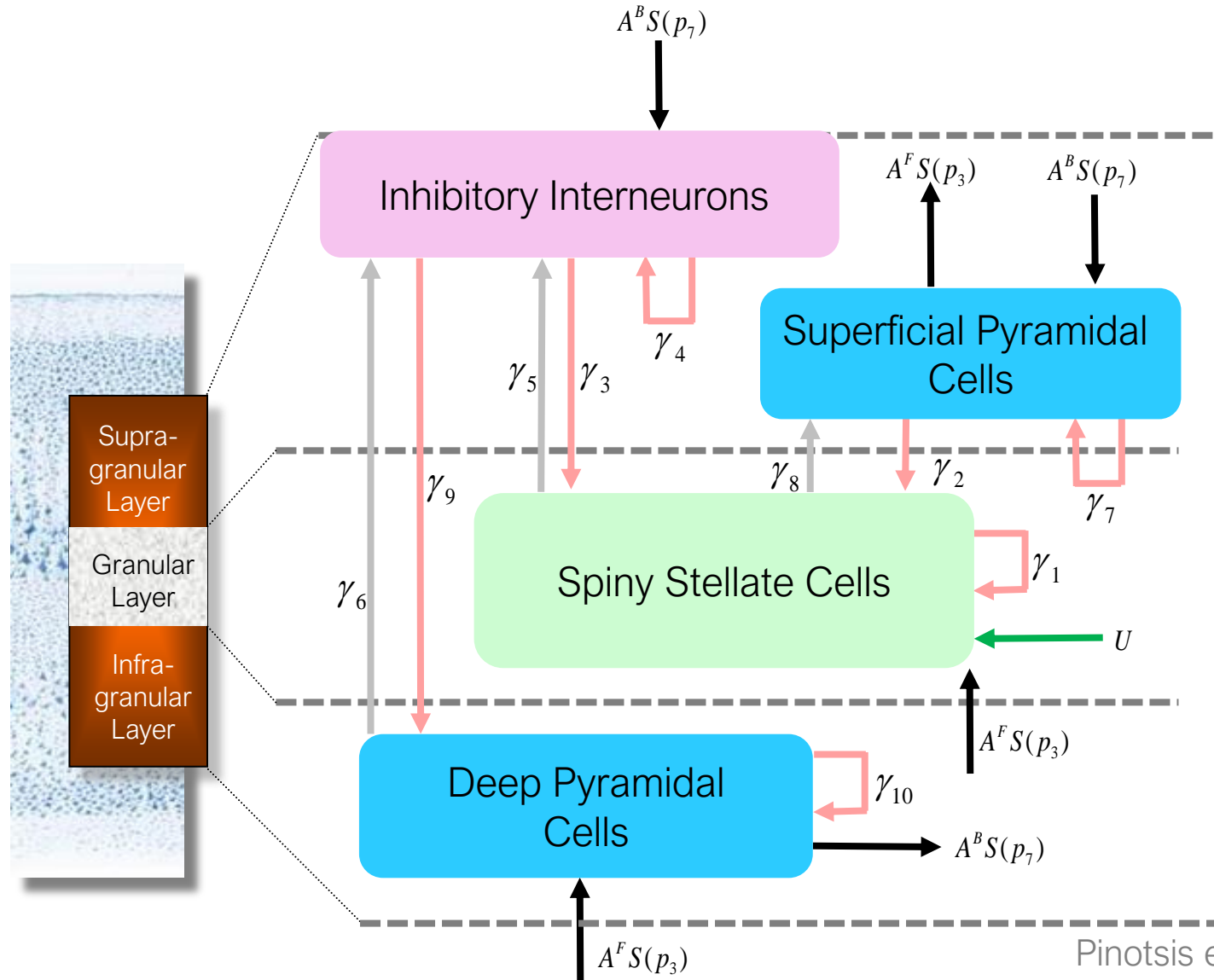
Canonical Microcircuit Model ('CMC')



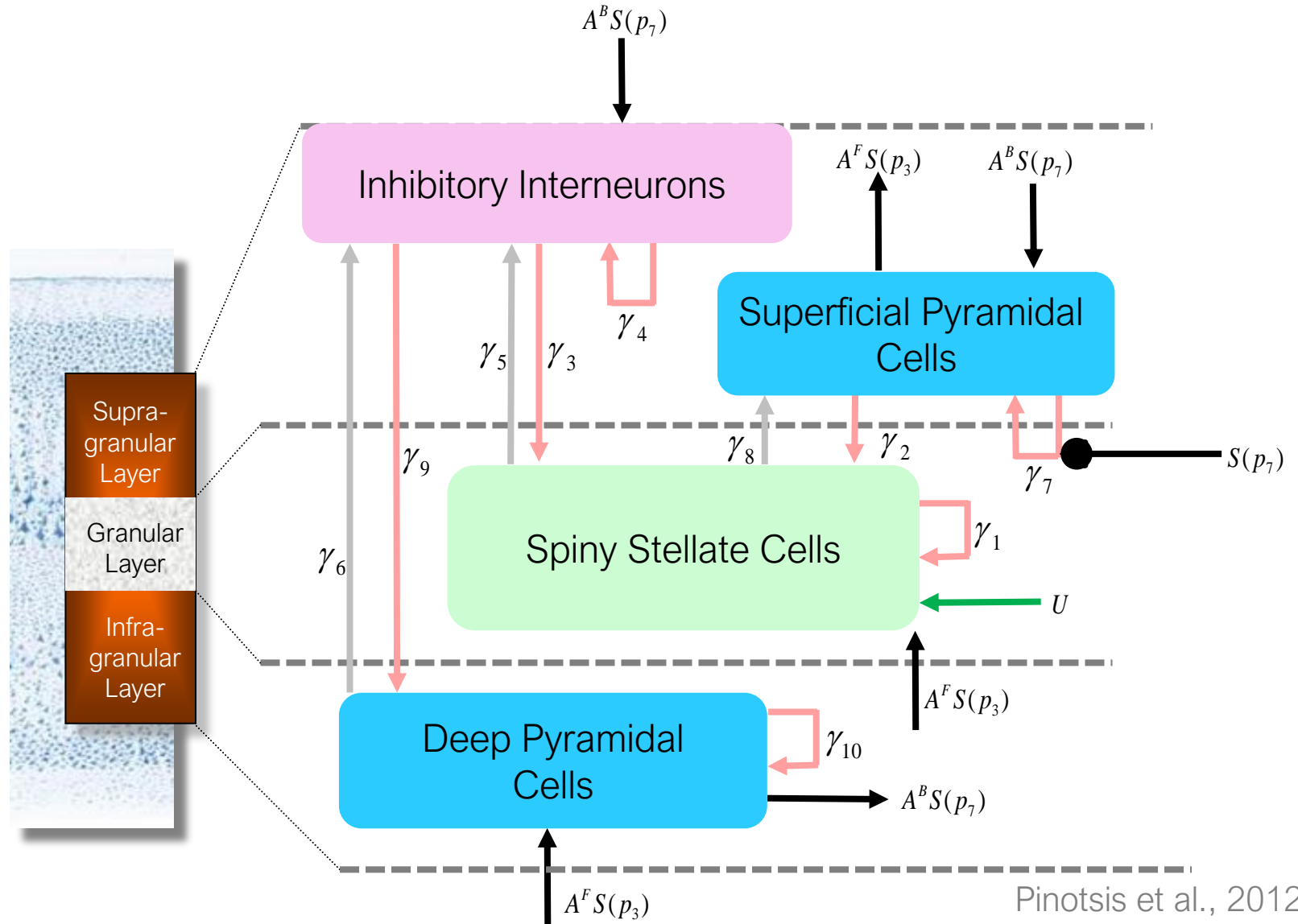
Canonical Microcircuit Model ('CMC')



Canonical Microcircuit Model ('CMC')



Canonical Microcircuit Model ('CMC')



Canonical Microcircuit Model ('CMC')

$$\dot{p}_7 = p_8$$

Voltage change rate: f(current)

Current change rate: f(voltage, current)

$$\dot{p}_8 = \frac{H_4}{\tau_4} (A^F S(p_2) - \gamma_{10} S(p_7) - \gamma_9 S(p_5)) - \frac{2p_8}{\tau_4} - \frac{p_7}{\tau_4^2}$$

Canonical Microcircuit Model ('CMC')

$$\dot{p}_7 = p_8$$

Voltage change rate: f(current)

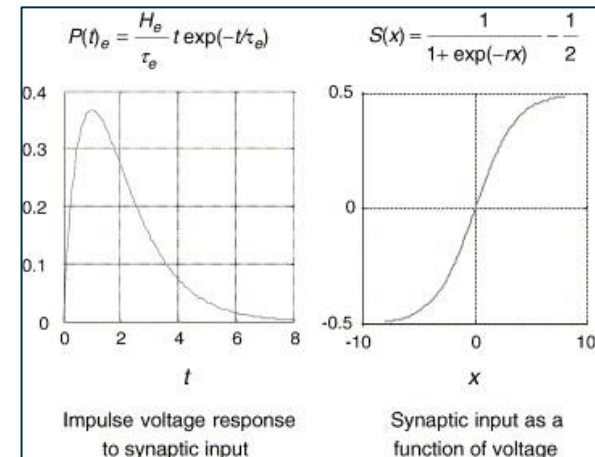
Current change rate: f(voltage, current)

$$\dot{p}_8 = \frac{H_4}{\tau_4} (A^F S(p_2) - \gamma_{10} S(p_7) - \gamma_9 S(p_5)) - \frac{2p_8}{\tau_4} - \frac{p_7}{\tau_4^2}$$

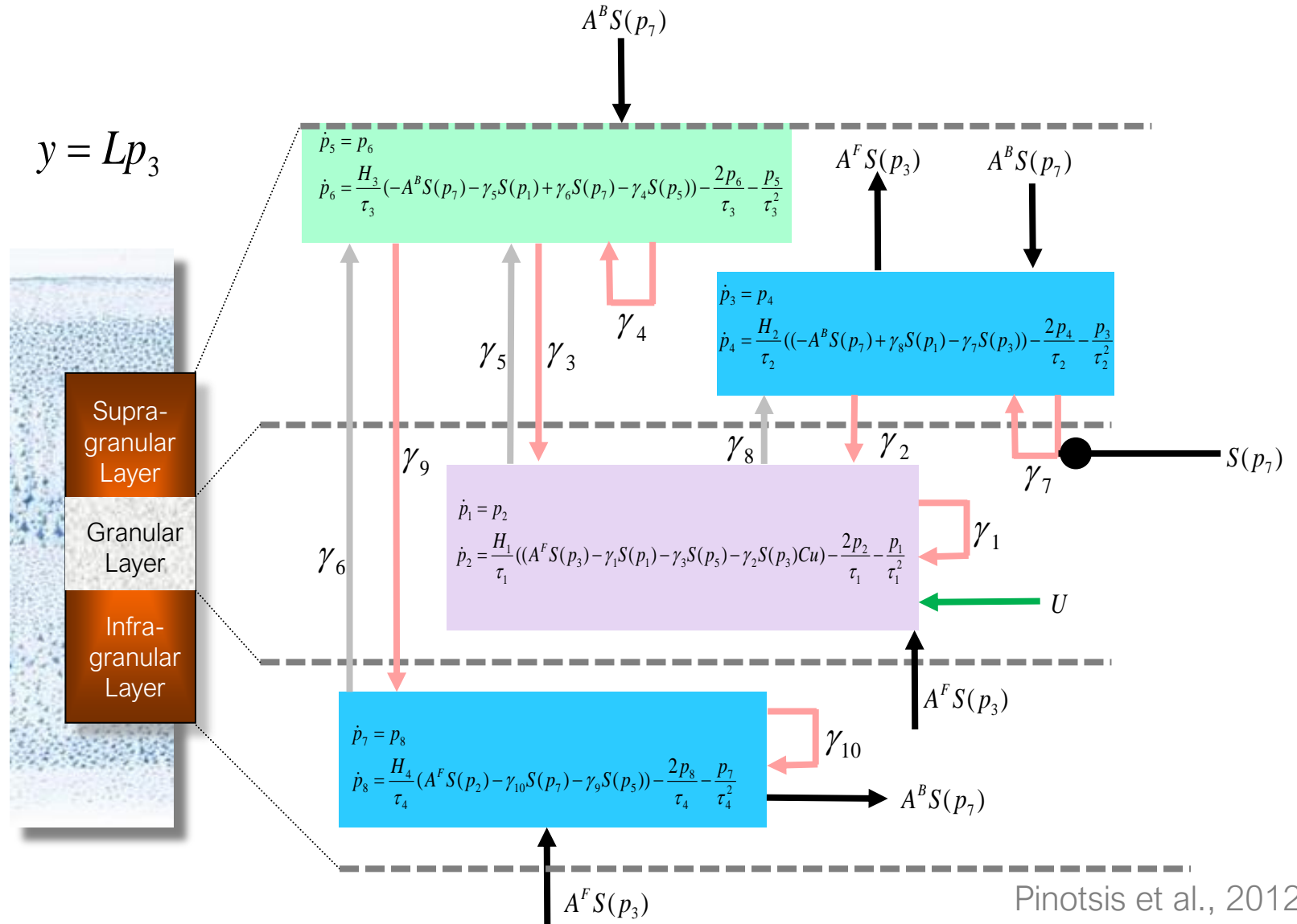
H, τ Kernels: pre-synaptic inputs \rightarrow post-synaptic membrane potentials

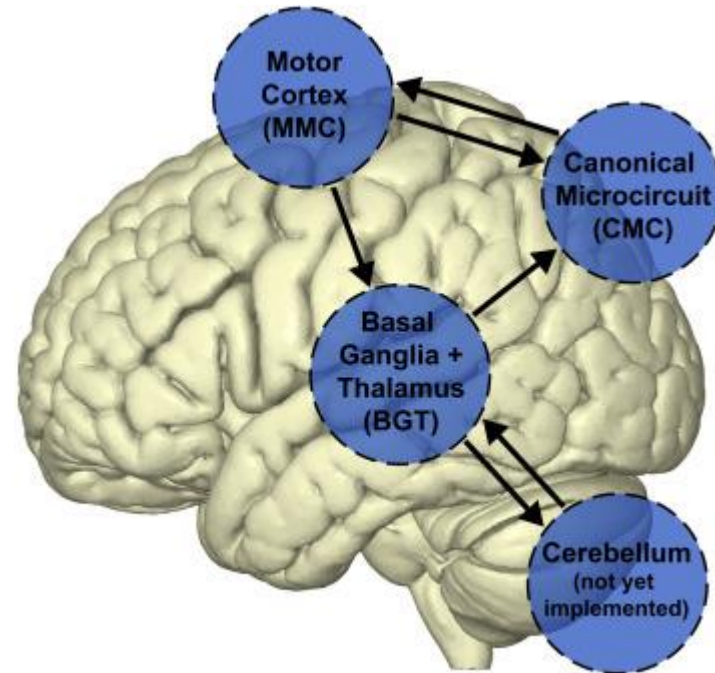
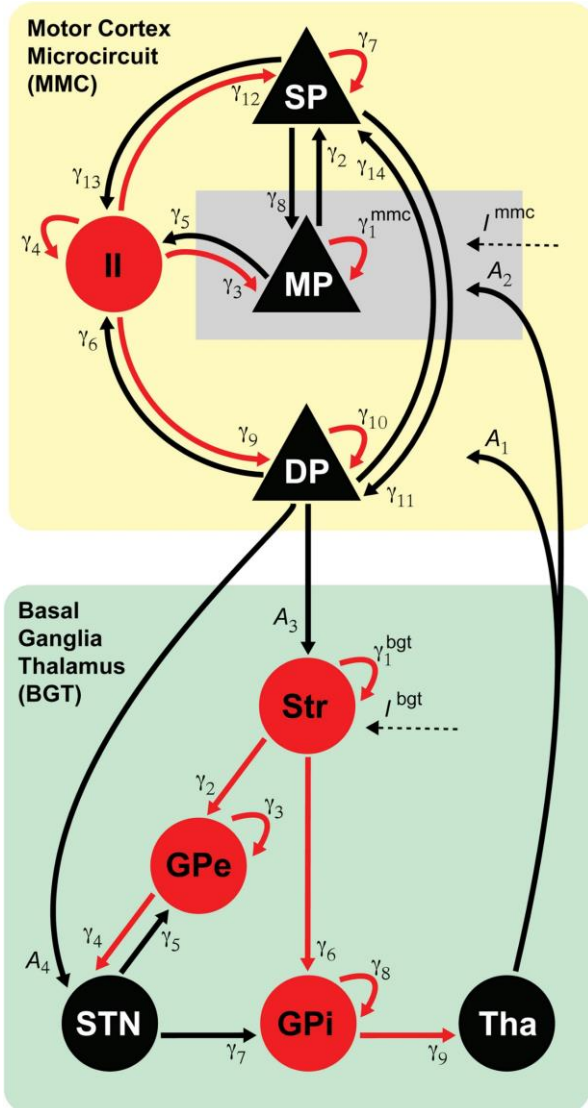
[H : max PSP; τ : rate constant]

S Sigmoid operator: PSP \rightarrow firing rate

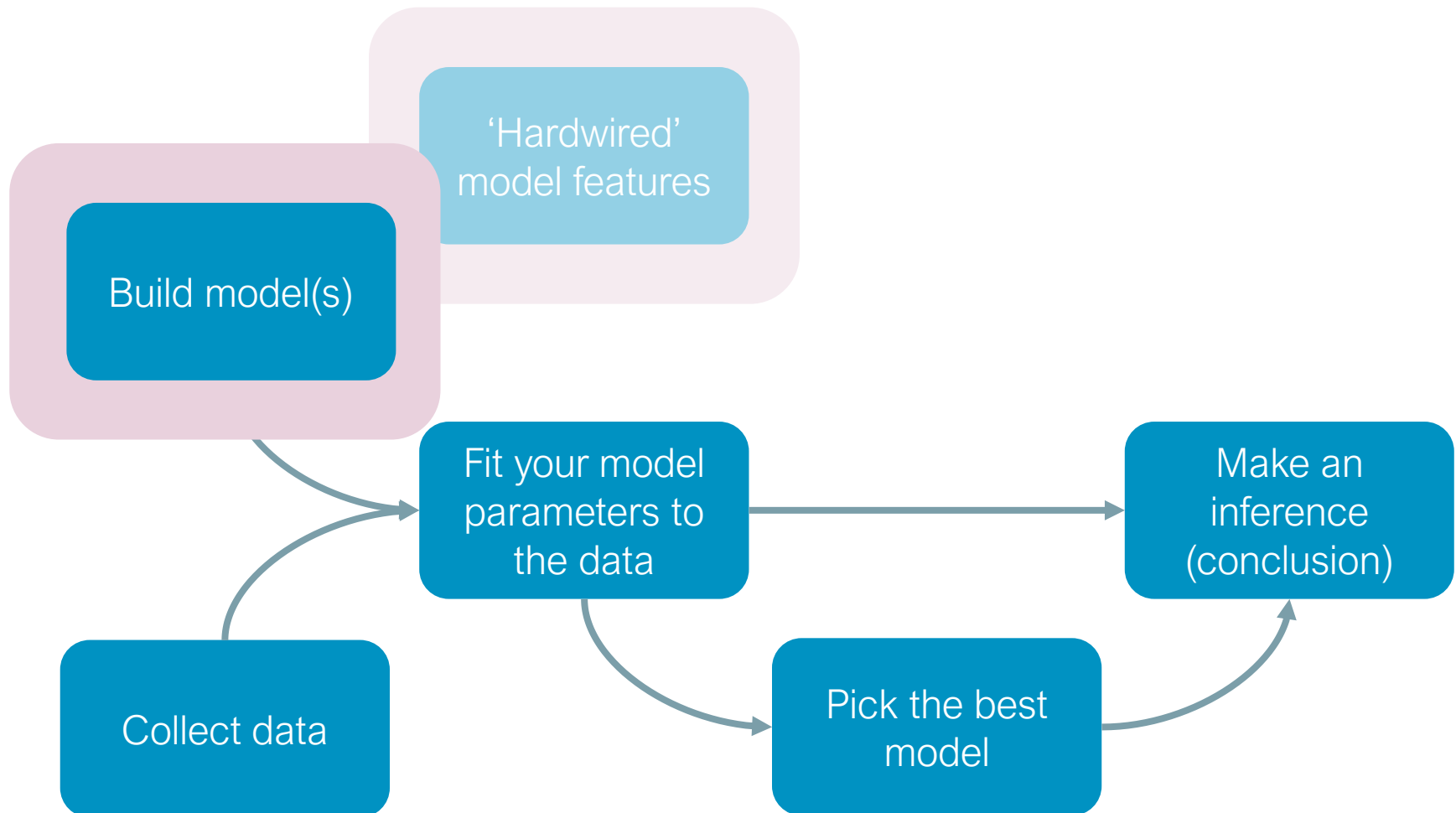


Canonical Microcircuit Model ('CMC')





The DCM analysis pathway



electromagnetic model

source names and locations: prior mean (mm)

right A1	46 -14 8
left A1	-42 -22 7
right STG	56 -40 18
left STG	-60 -48 20
right IPS	34 -66 46

neuronal model

forward back Modulatory input

dipolar symmetry
 optimise source locations
 lock trial-specific effects
 trial-specific inputs

Wavelet transform
 frequency window Hz:
 wavelet number:

ERPs (mode)

5

4

3

2

1

electromagnetic model

source names and locations: prior mean (mm)

right A1	46 -14 8
left A1	-42 -22 7
right STG	56 -40 18
left STG	-60 -48 20
right IPS	34 -66 46

onsets (ms): 20
duration (sd): 16

neuronal model

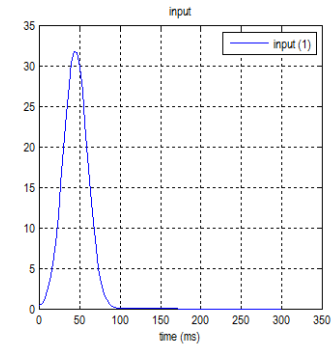
forward back Modulatory **input**

B att-noatt B dev-std

dipolar symmetry
 optimise source locations
 lock trial-specific effects
 trial-specific inputs

Wavelet transform frequency window Hz: 4 48 wavelet number: 7 image API

ERPs (mode) initialise priors BMS post hoc reduce



electromagnetic model

source names and locations: prior mean (mm)

right A1	46 -14 8
left A1	-42 -22 7
right STG	56 -40 18
left STG	-60 -48 20
right IPS	34 -66 46

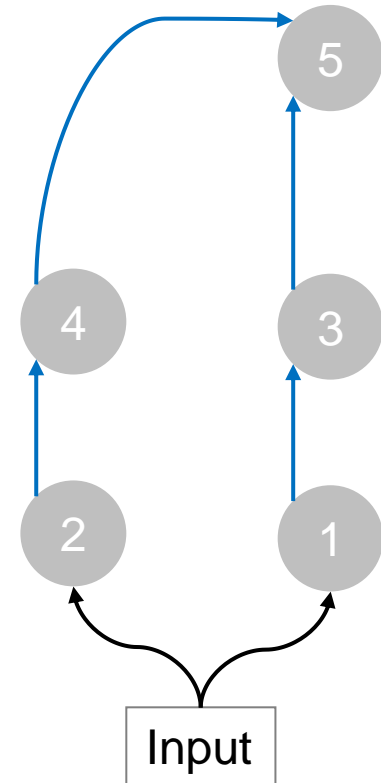
20
 16

neuronal model

forward back Modulatory input

dipolar symmetry optimise source locations lock trial-specific effects trial-specific inputs

frequency window Hz: wavelet number:



electromagnetic model

source names and locations: prior mean (mm)

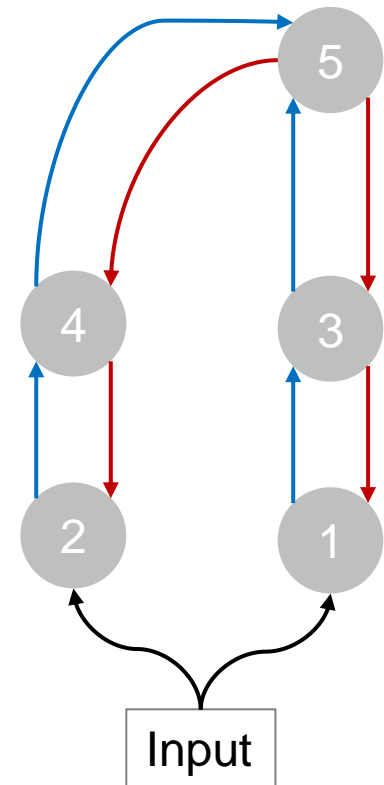
right A1	46	-14	8
left A1	-42	-22	7
right STG	56	-40	18
left STG	-60	-48	20
right IPS	34	-66	46

neuronal model

forward back Modulatory input

dipolar symmetry optimise source locations lock trial-specific effects trial-specific inputs

Wavelet transform frequency window Hz: wavelet number:



electromagnetic model

source names and locations: prior mean (mm)

right A1	46 -14 8
left A1	-42 -22 7
right STG	56 -40 18
left STG	-60 -48 20
right IPS	34 -66 46

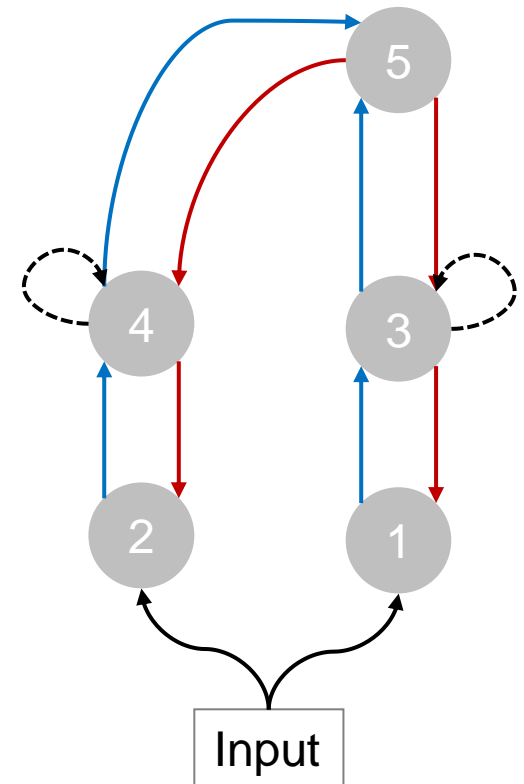
onsets (ms): 20
duration (sd): 16

neuronal model

Modulatory

frequency window Hz: 4 48
wavelet number: 7

initialise priors BMS post hoc reduce



electromagnetic model

source names and locations: prior mean (mm)

right A1	46	-14	8
left A1	-42	-22	7
right STG	56	-40	18
left STG	-60	-48	20
right IPS	34	-66	46

onsets (ms): 20
duration (sd): 16

neuronal model

forward back Modulatory input

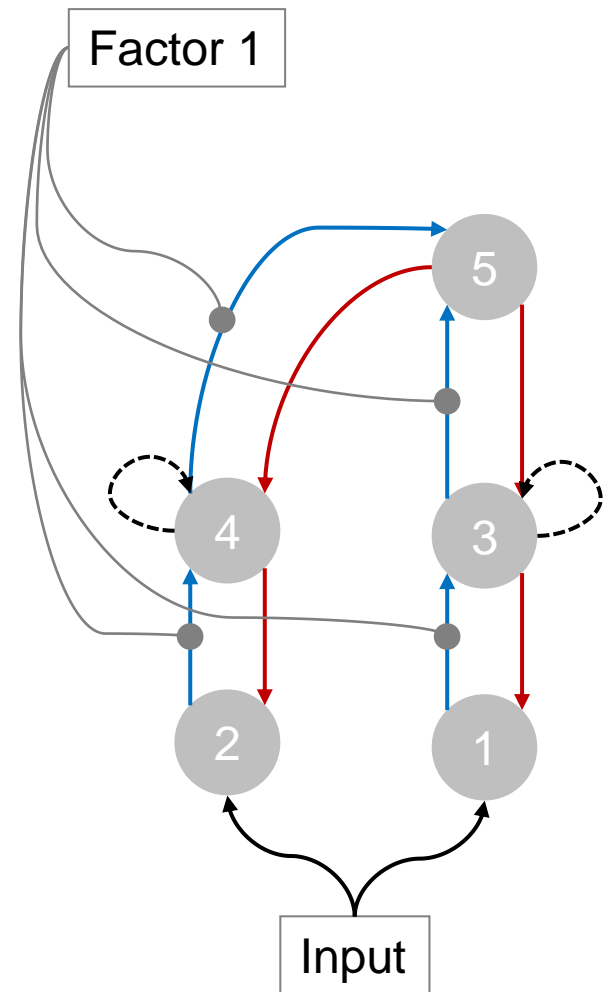
B att-noatt

invert DCM

dipolar symmetry
 optimise source locations
 lock trial-specific effects
 trial-specific inputs

Wavelet transform frequency window Hz: 4 48 wavelet number: 7 image API

ERPs (mode) initialise priors BMS post hoc reduce



electromagnetic model

source names and locations: prior mean (mm)

right A1	46	-14	8
left A1	-42	-22	7
right STG	56	-40	18
left STG	-60	-48	20
right IPS	34	-66	46

onsets (ms): 20

duration (sd): 16

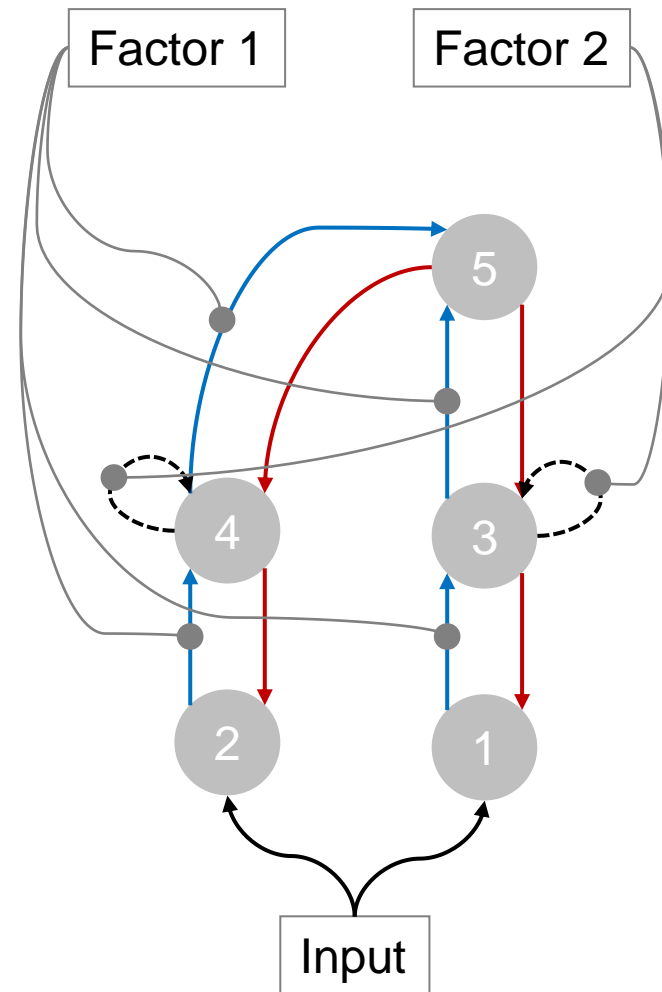
neuronal model

forward back Modulatory input

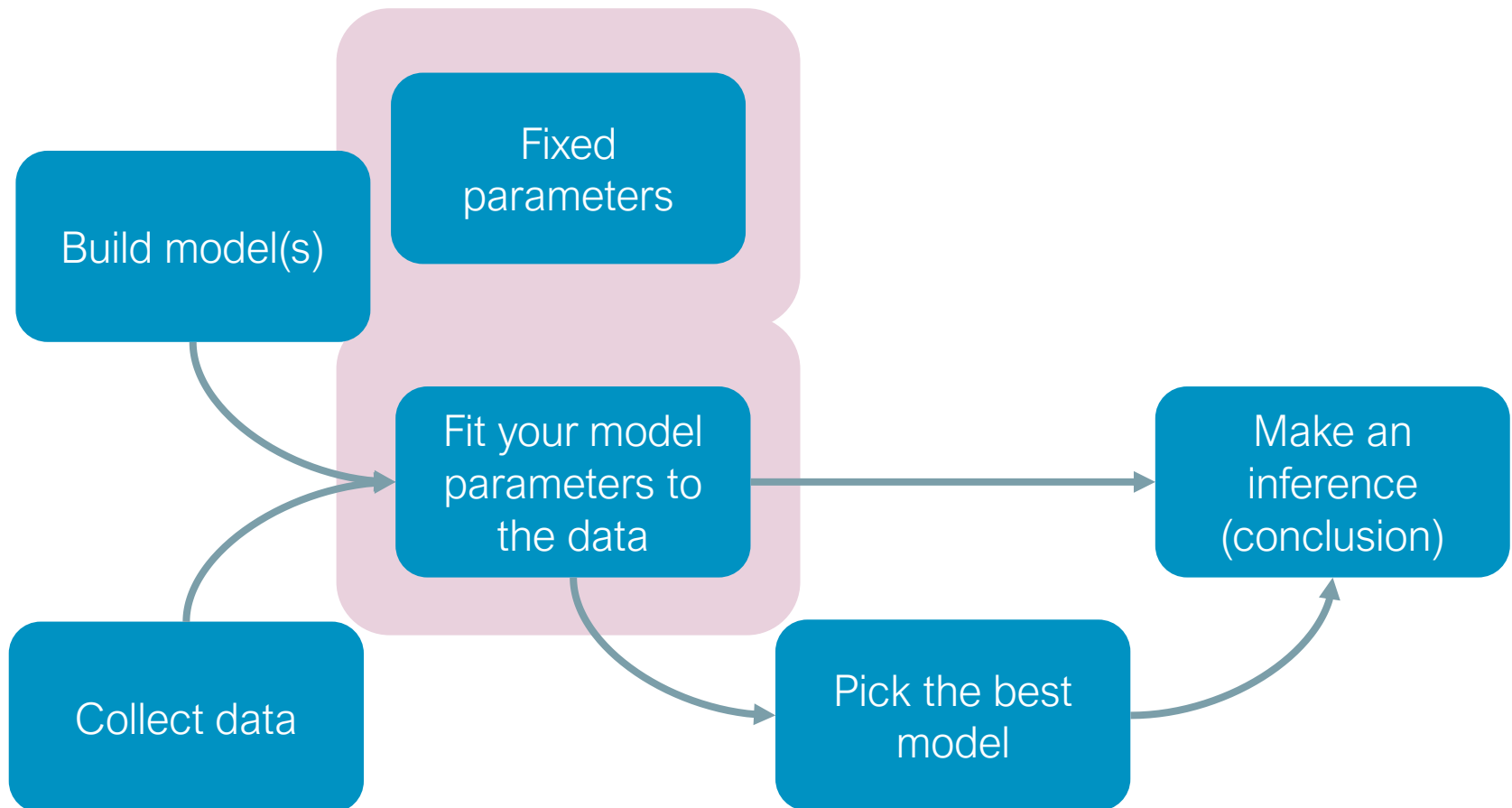
B att-noatt B dev-std

frequency window Hz: 4 48 wavelet number: 7

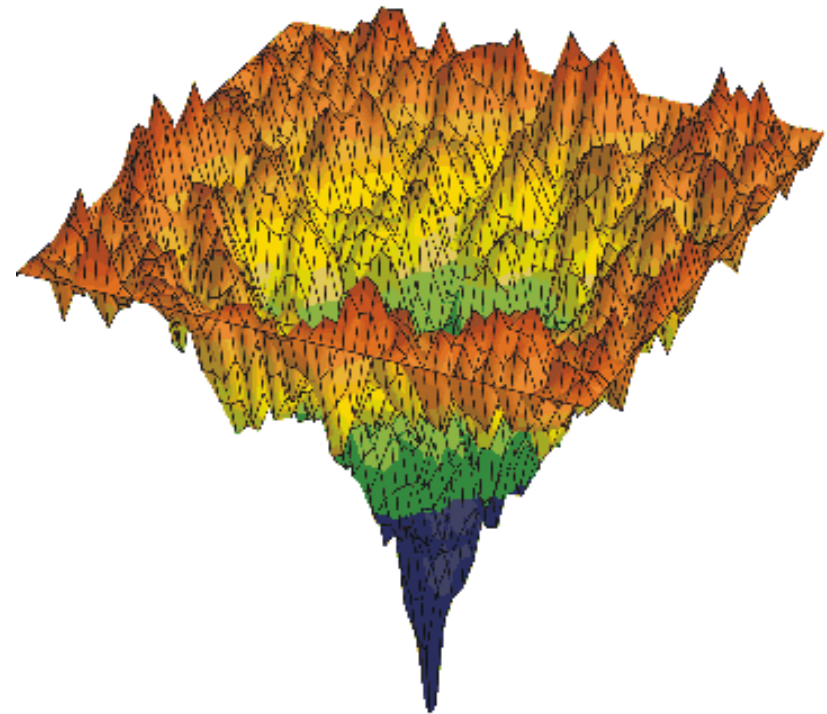
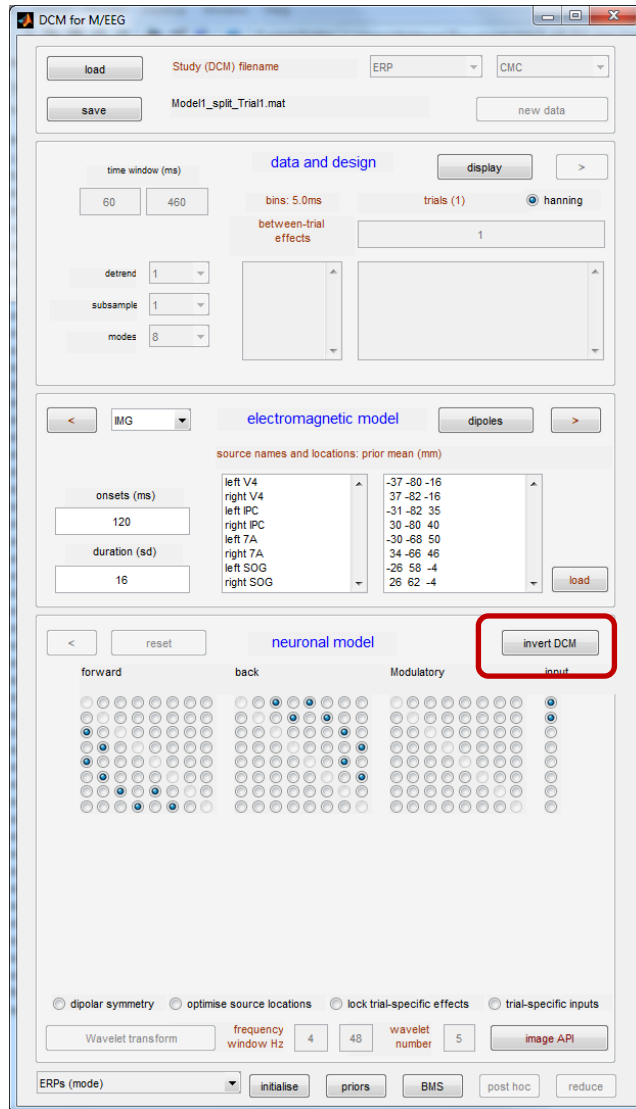
ERPs (mode) initialise priors BMS post hoc reduce



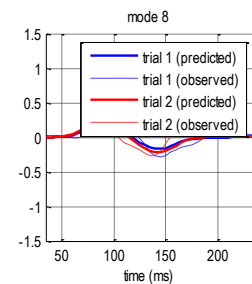
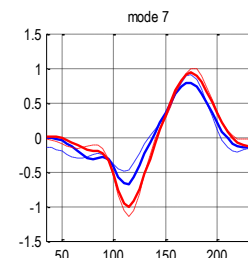
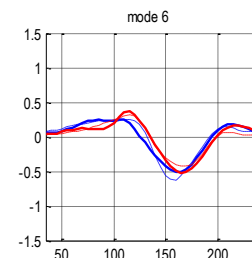
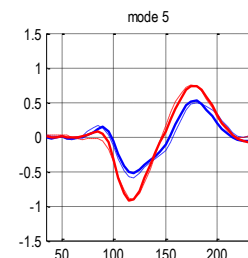
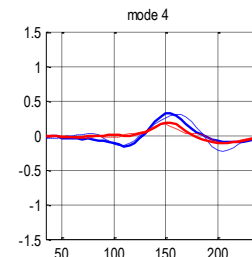
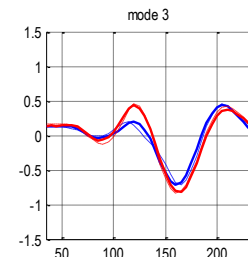
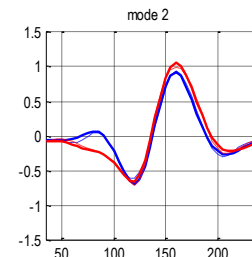
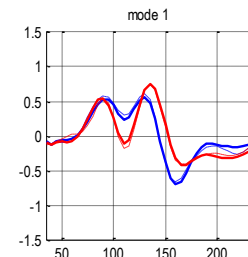
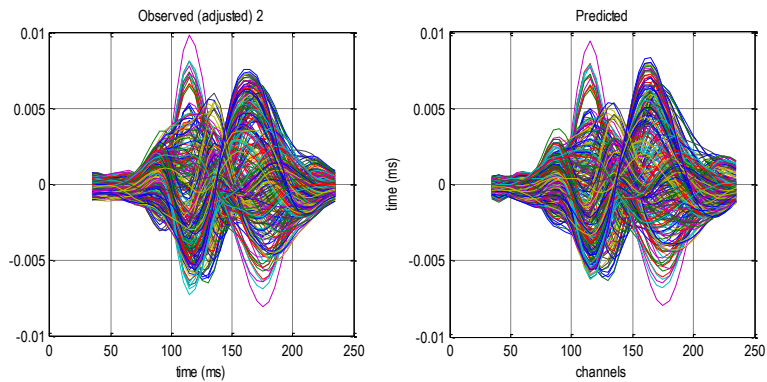
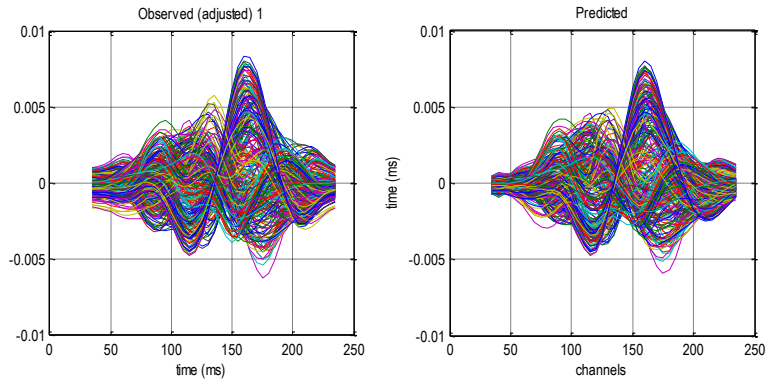
The DCM analysis pathway



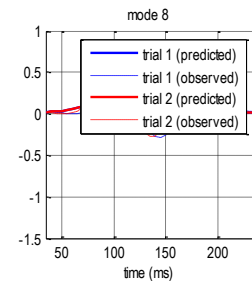
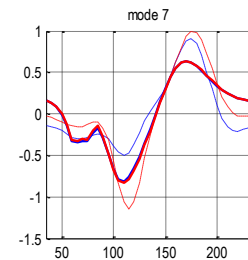
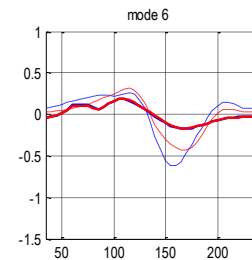
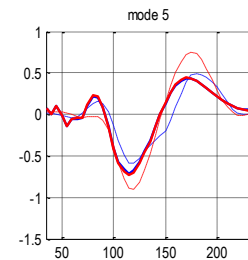
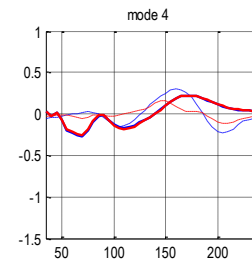
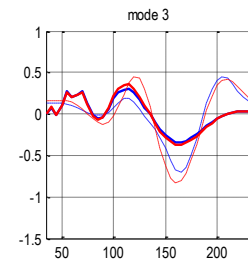
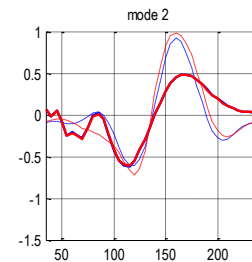
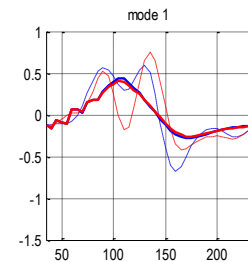
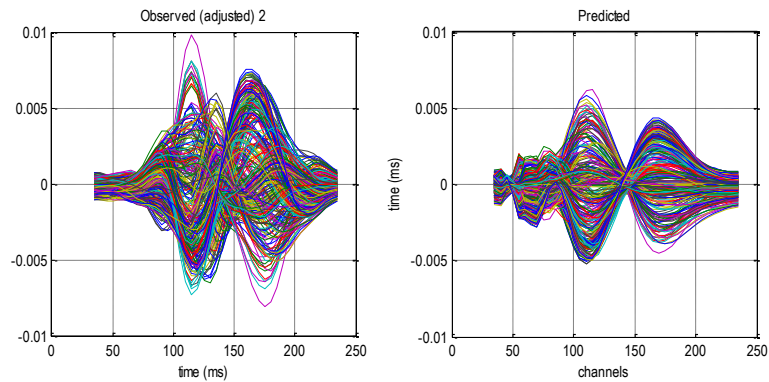
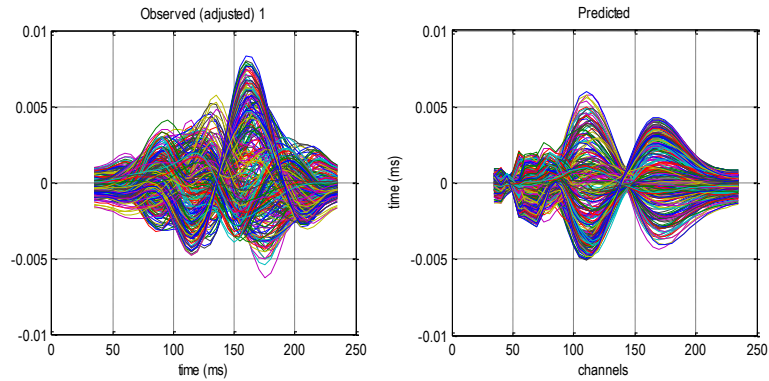
Fitting DCMs to data



Fitting DCMs to data

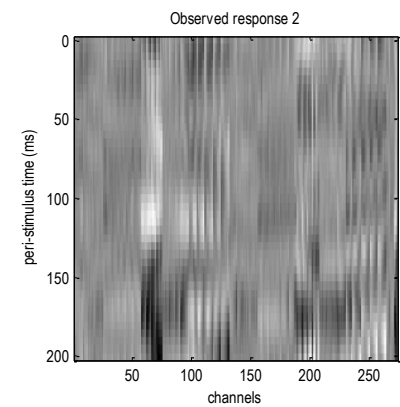
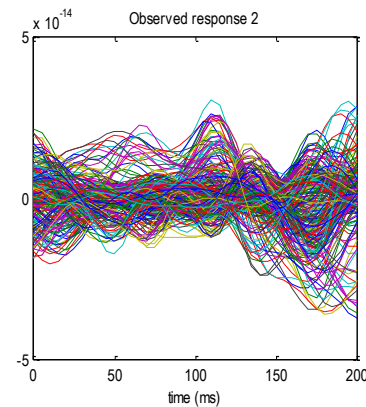
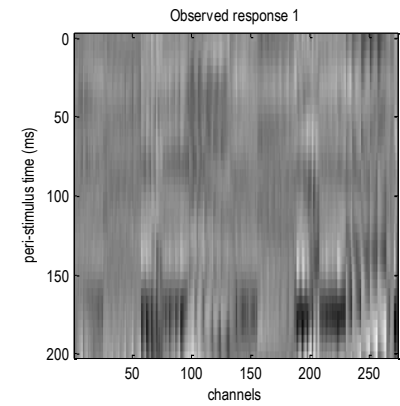
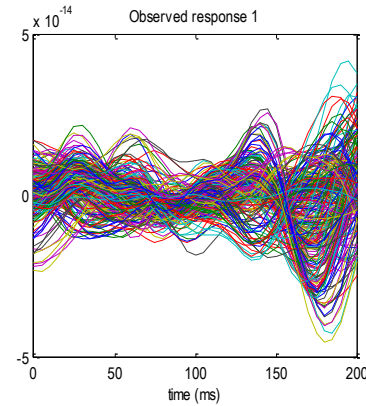


Fitting DCMs to data



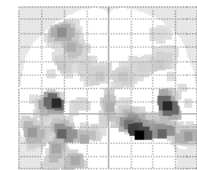
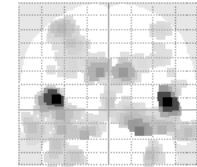
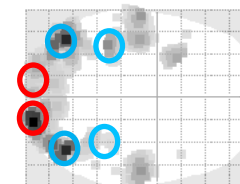
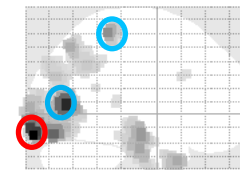
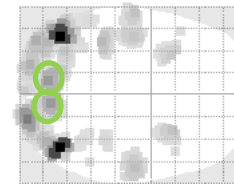
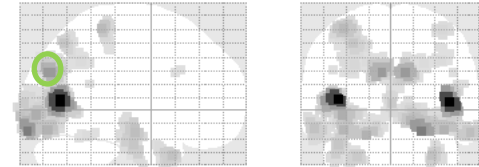
Fitting DCMs to data

1. Check your data



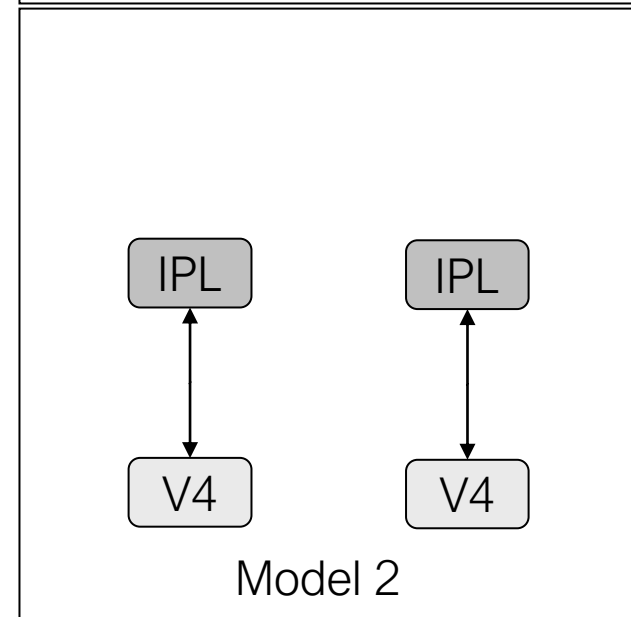
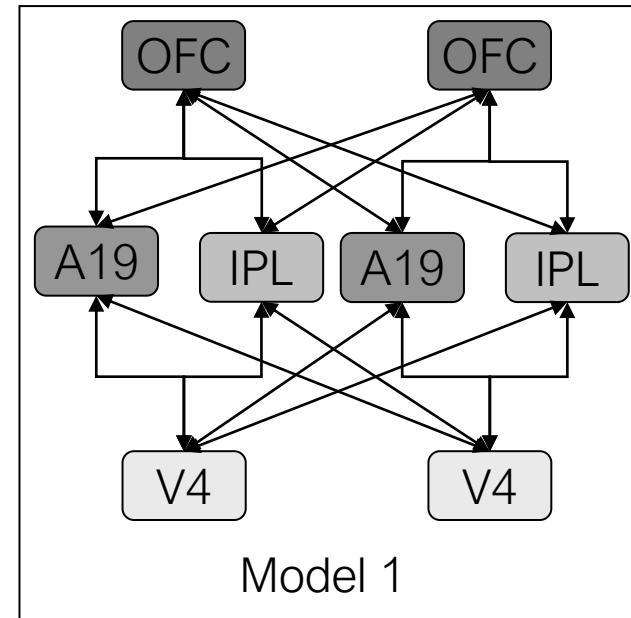
Fitting DCMs to data

1. Check your data
2. Check your sources



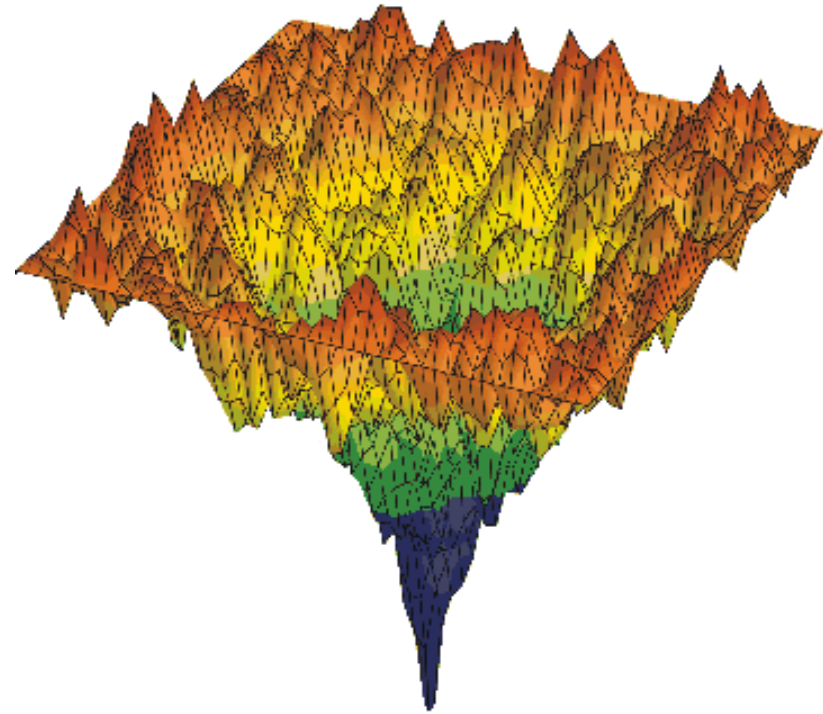
Fitting DCMs to data

1. Check your data
2. Check your sources
3. Check your model

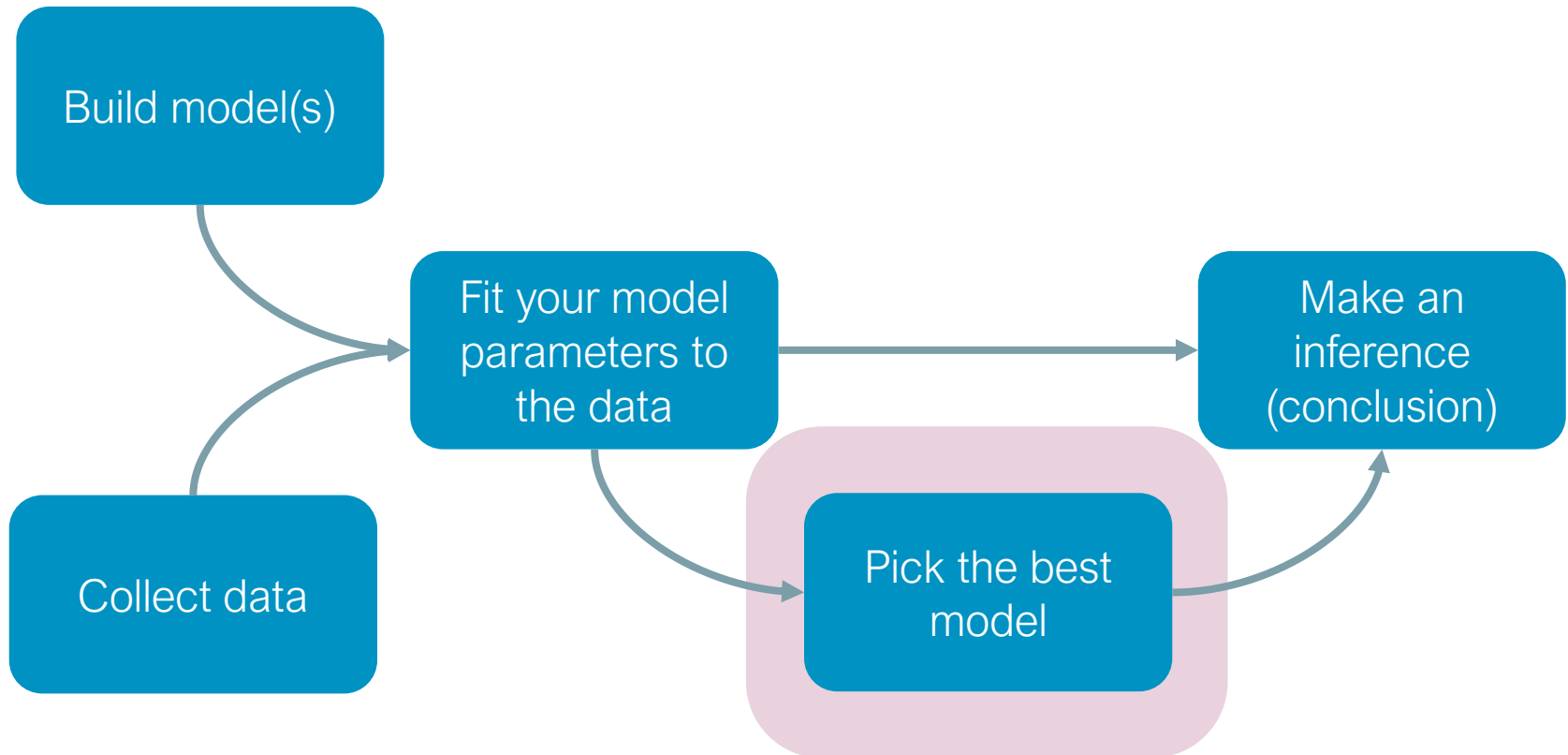


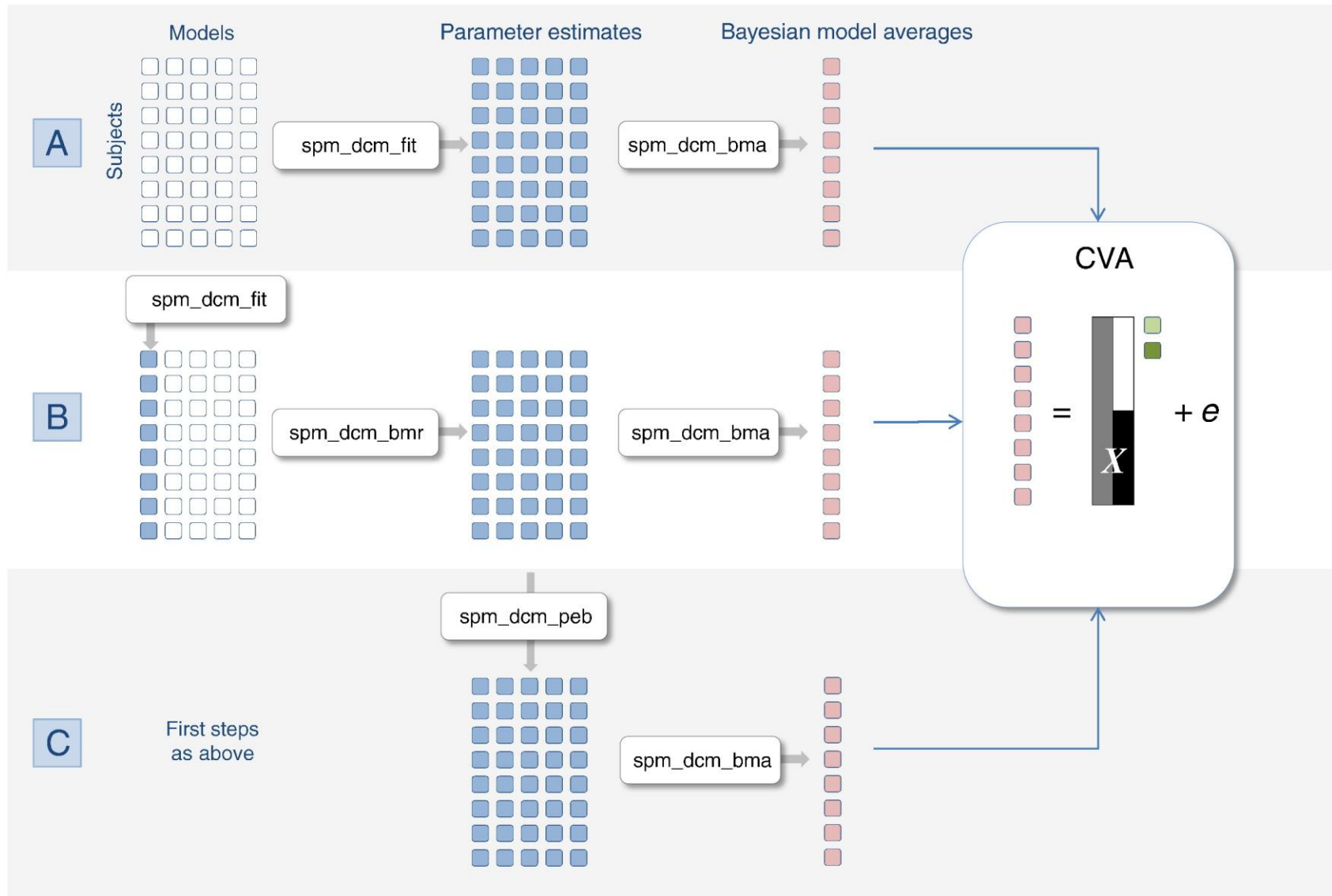
Fitting DCMs to data

1. Check your data
2. Check your sources
3. Check your model
4. Re-run model fitting

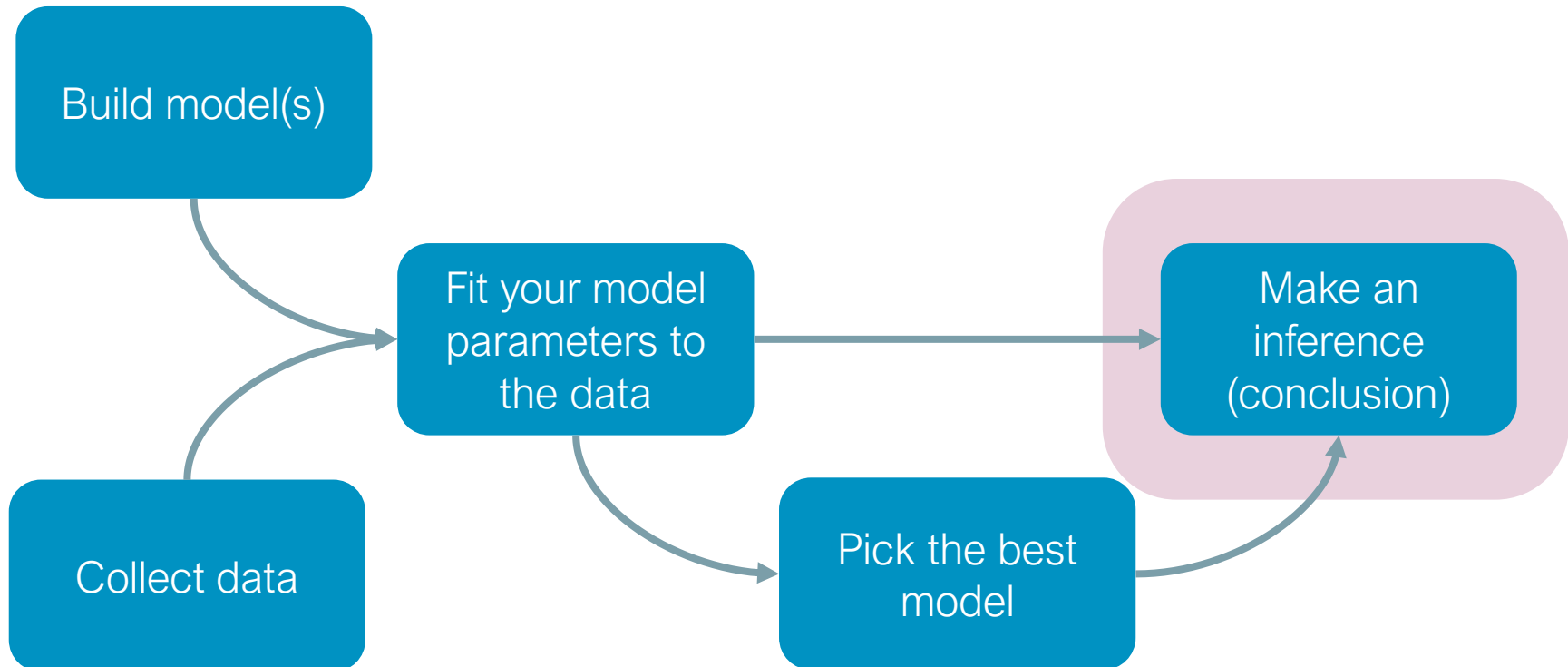


The DCM analysis pathway





The DCM analysis pathway



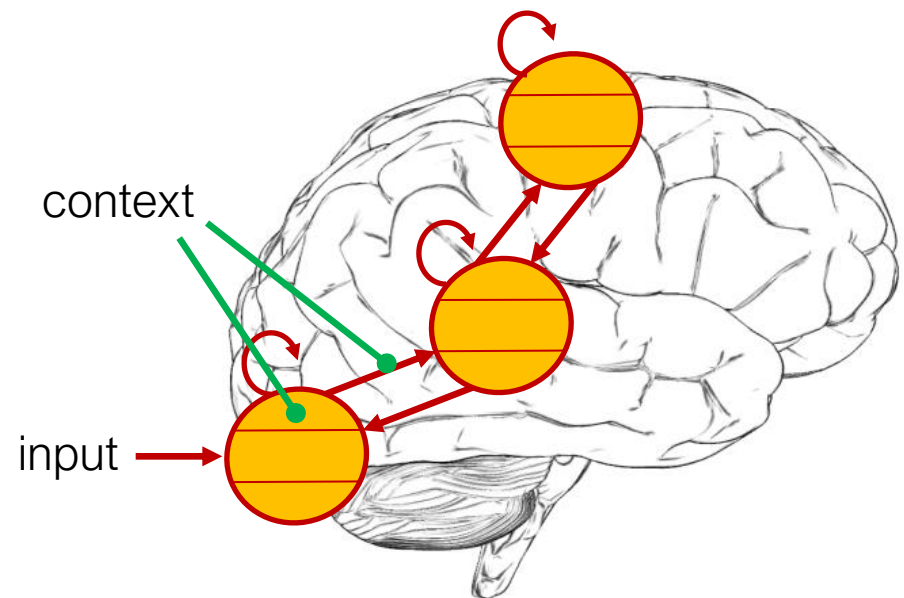
Does network XYZ explain my data better than network XY?

Which XYZ connectivity structure best explains my data?

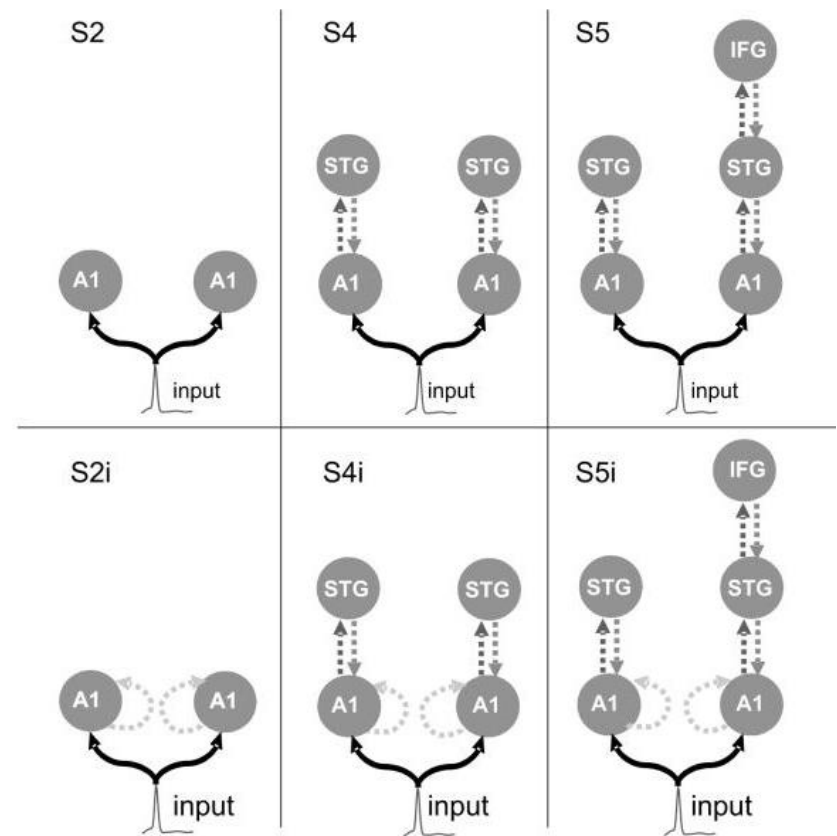
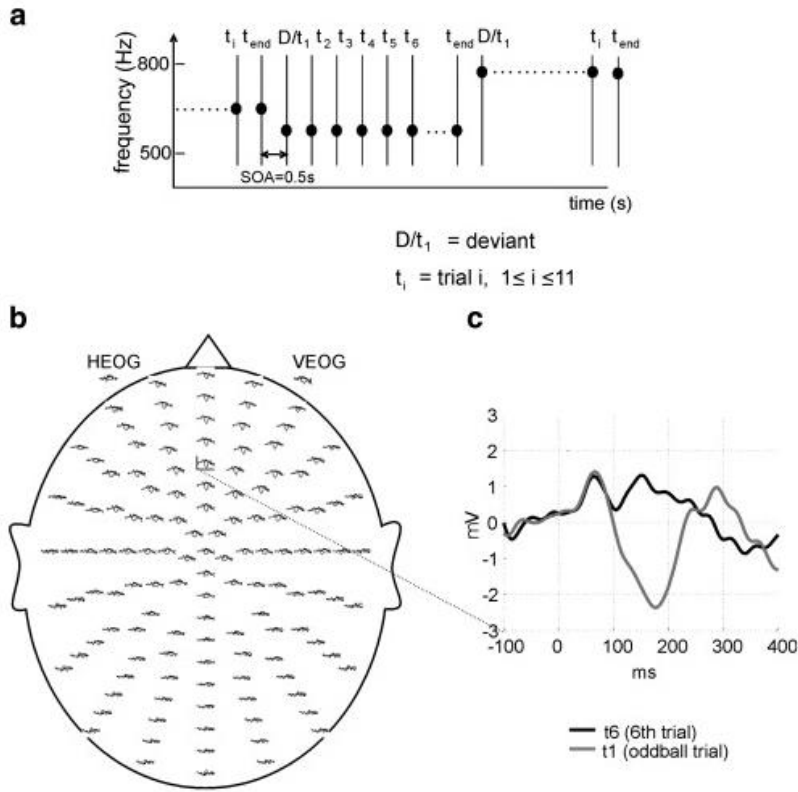
Are X & Y linked in a bottom-up, top-down or recurrent fashion?

Is my effect driven by extrinsic or intrinsic connections?

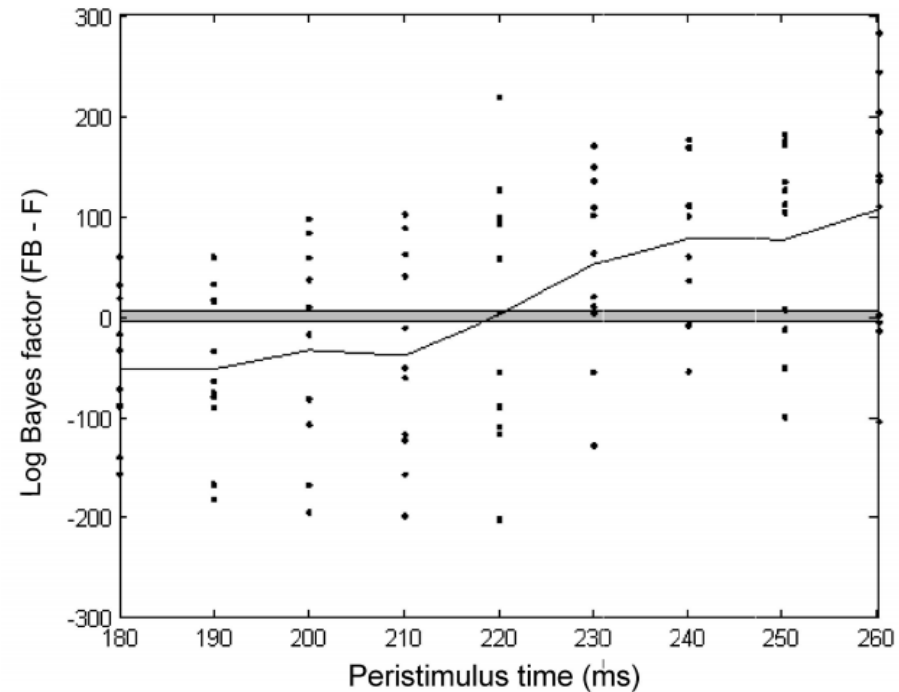
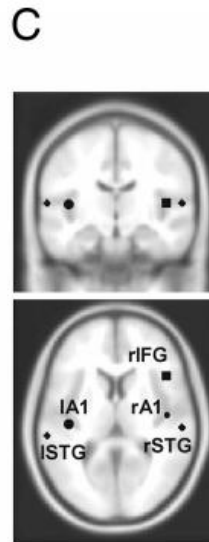
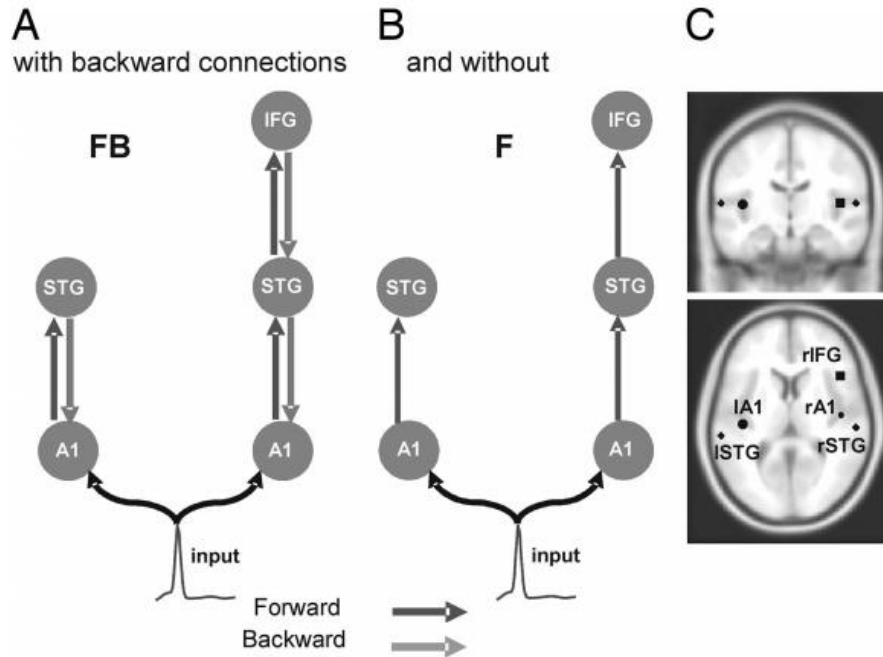
Which connections/populations are affected by contextual factors?



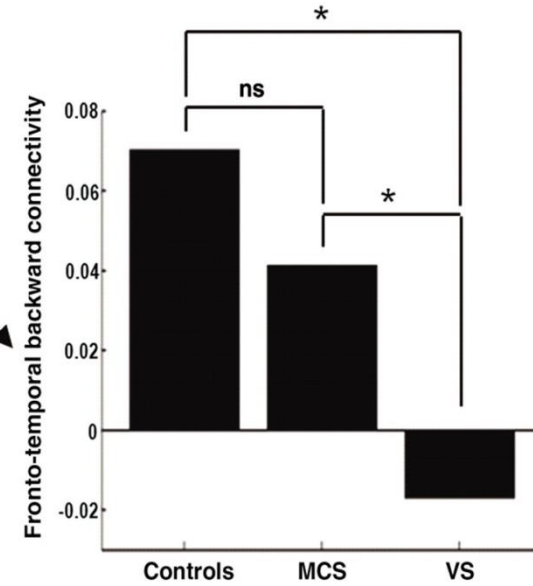
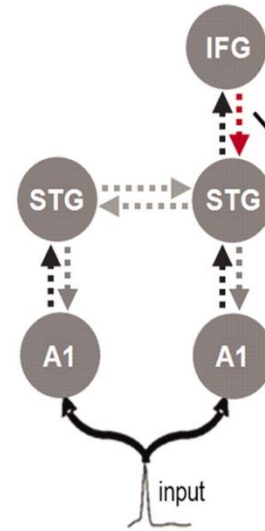
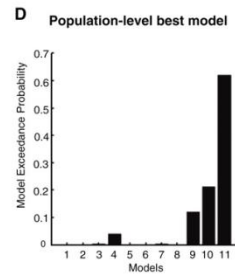
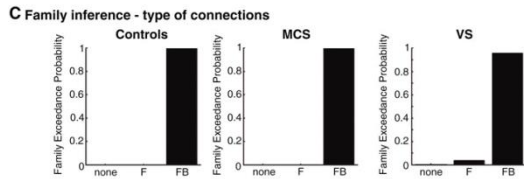
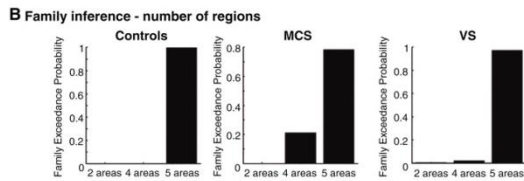
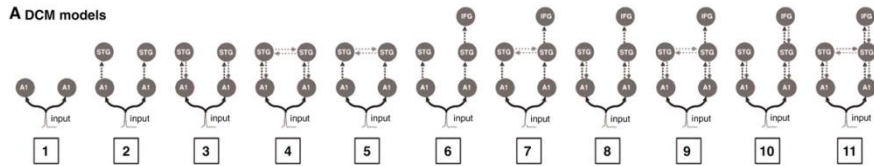
Example #1: Architecture of MMN



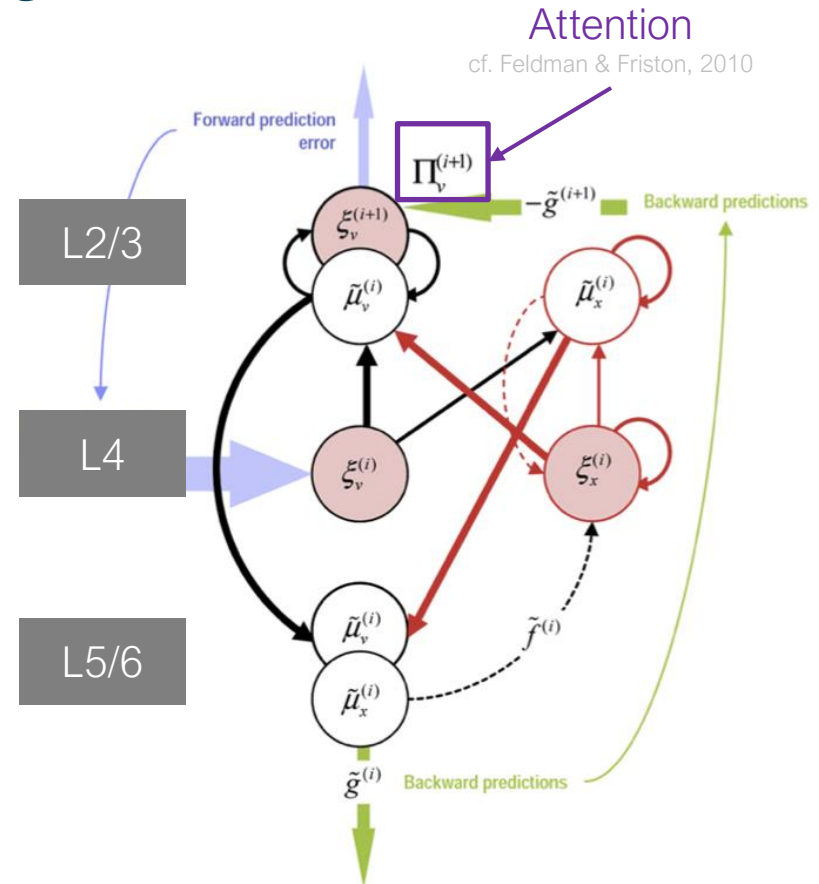
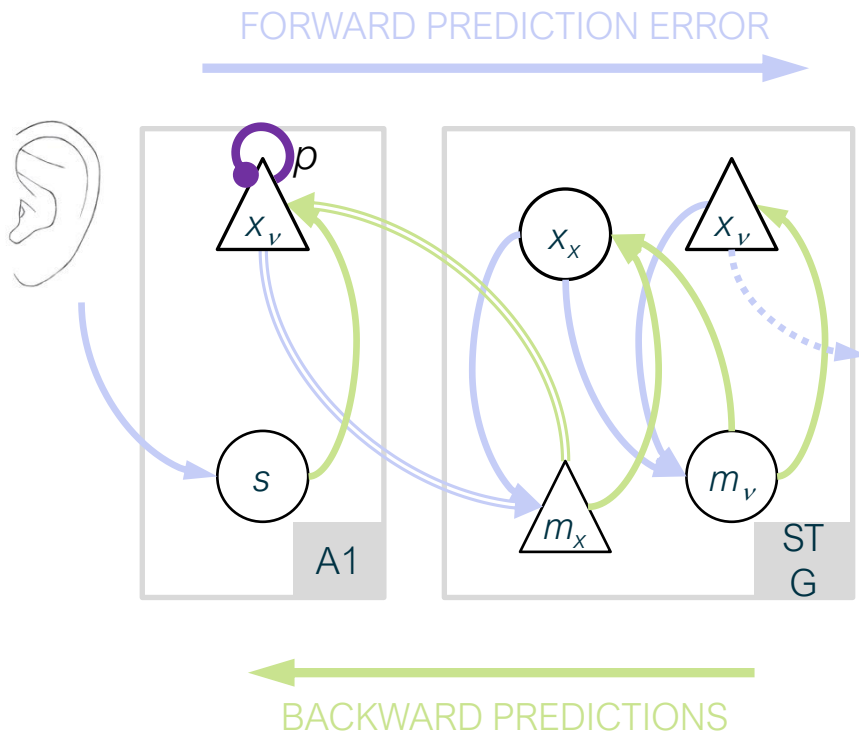
Example #2: Role of feedback connections



Example #3: Group differences

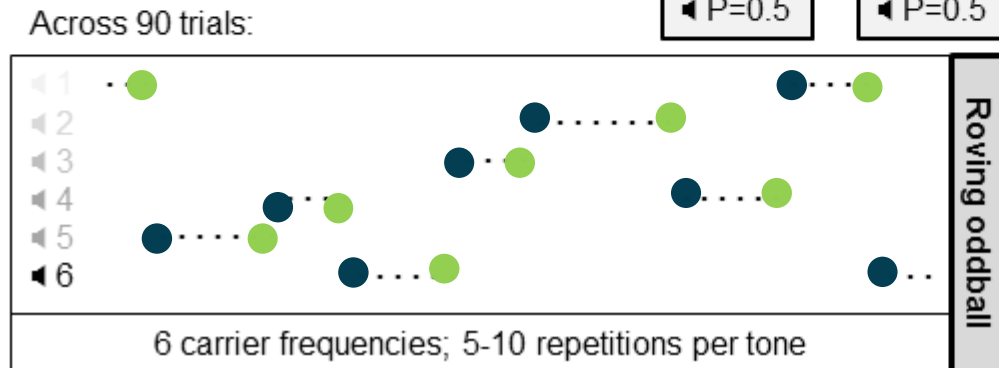
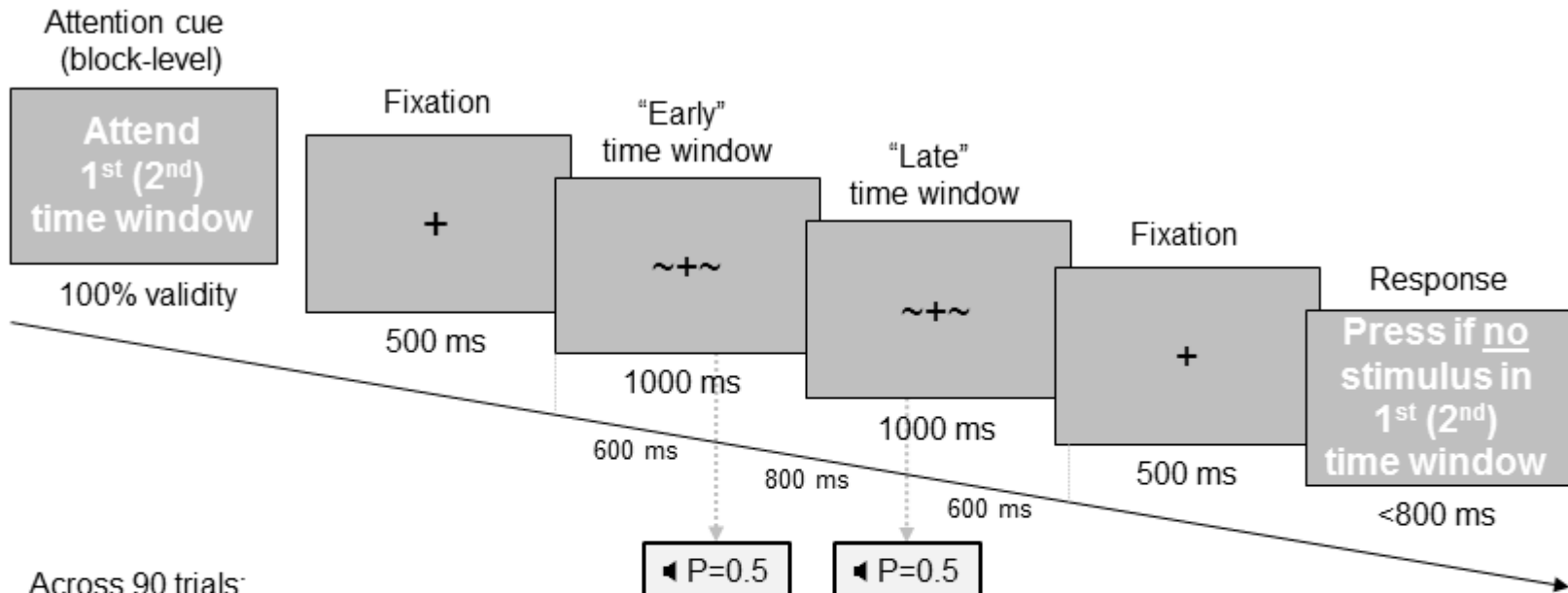


Example #4: Factorial design & CMC



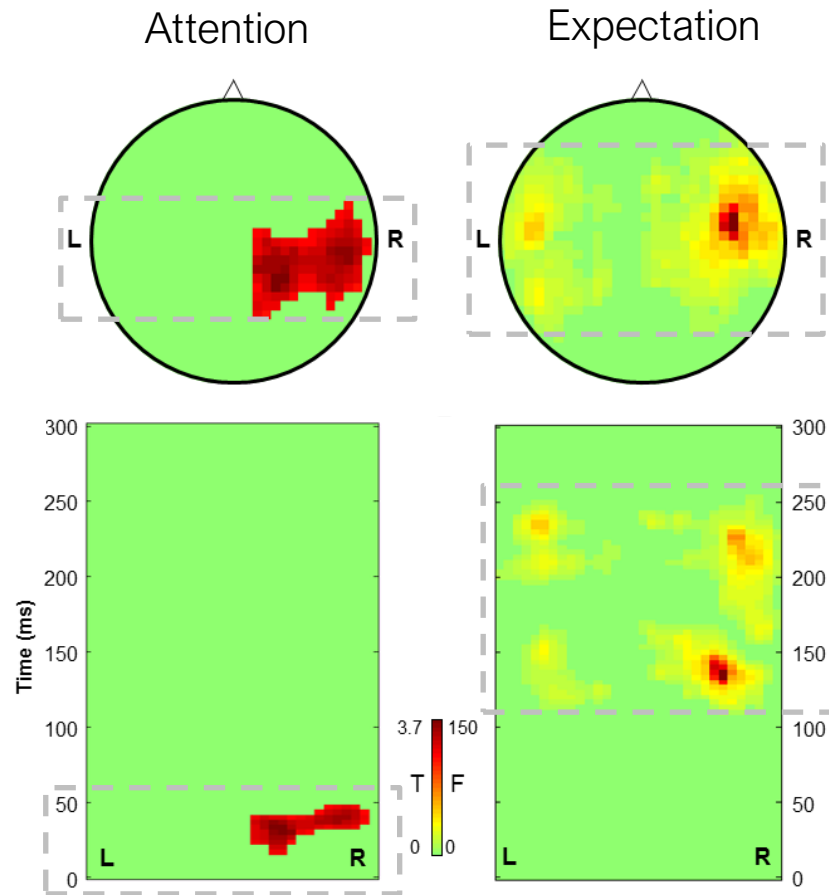
Bastos et al., *Neuron* 2012

Auksztulewicz & Friston, 2015

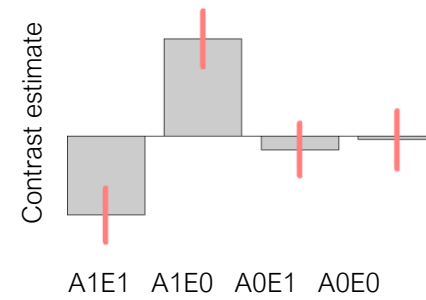
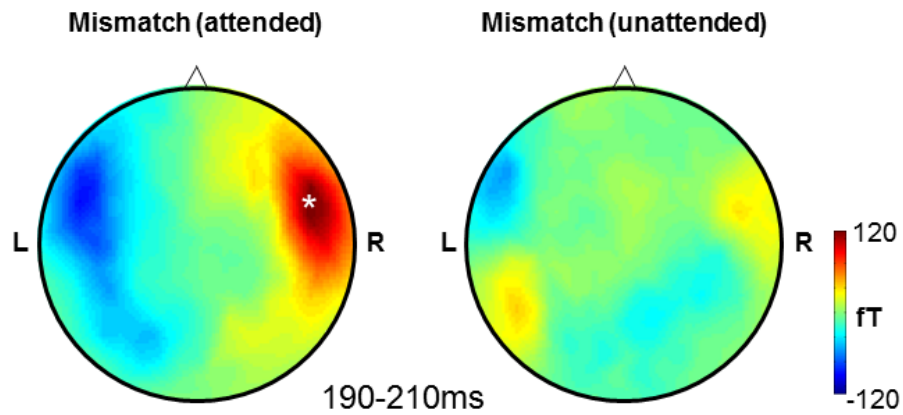


2x2 design:
 Attended vs unattended
 Standard vs deviant
 (Only trials with 2 tones)

N=20



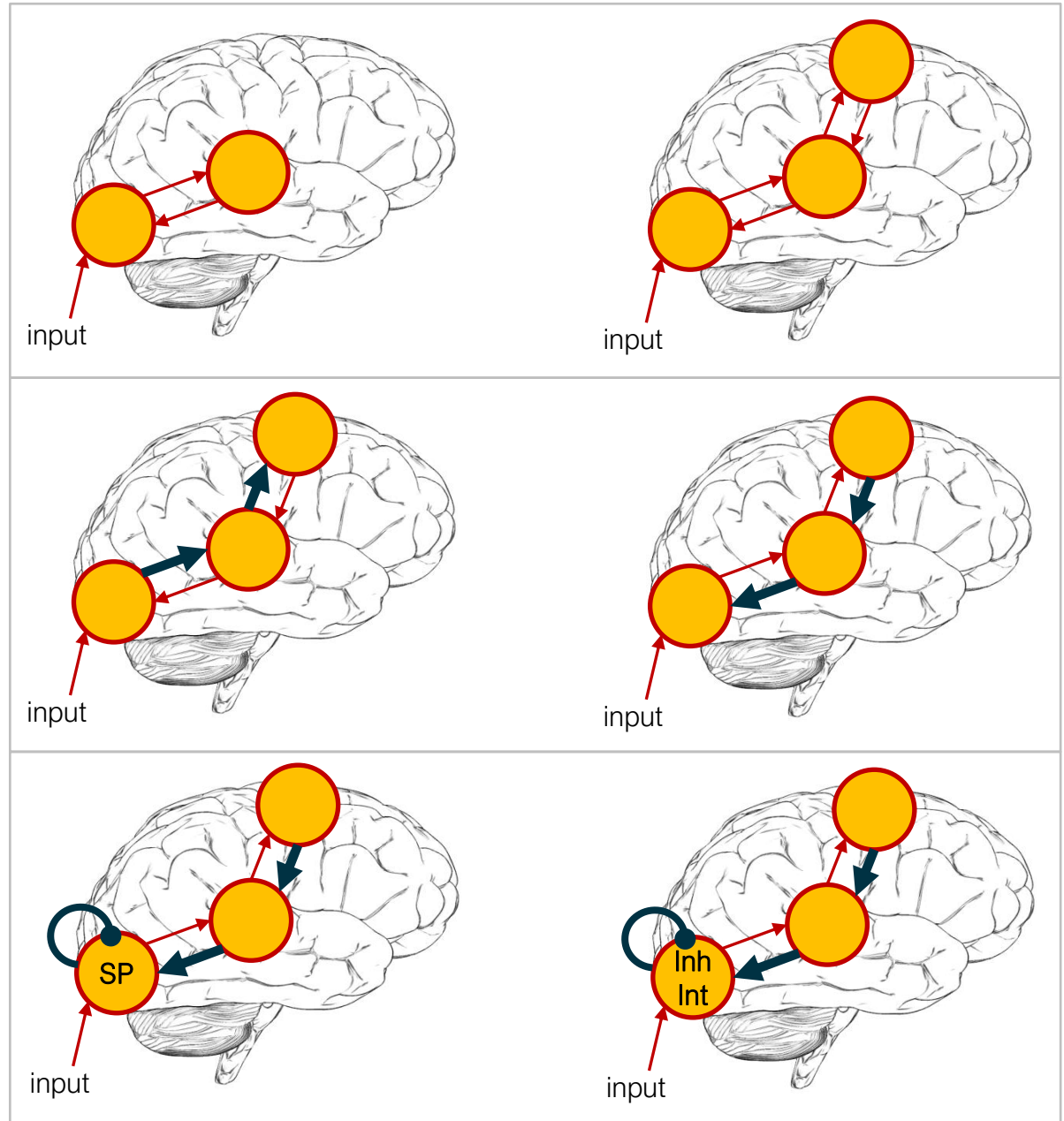
Flexible factorial design
 Thresholded at $p < .005$ peak-level
 Corrected at a cluster-level $pFWE < .05$

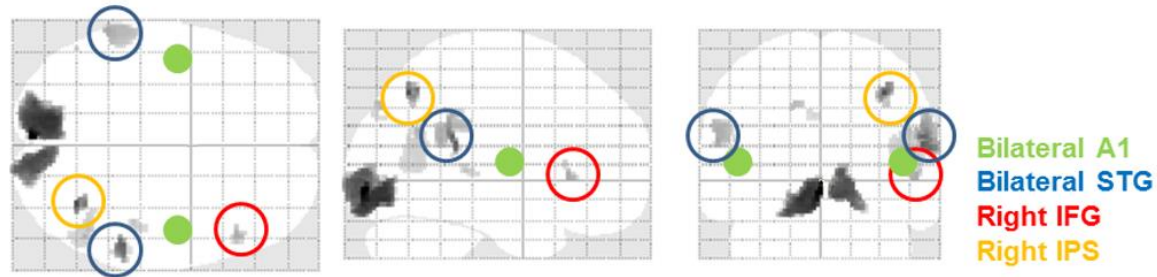


Connectivity structure

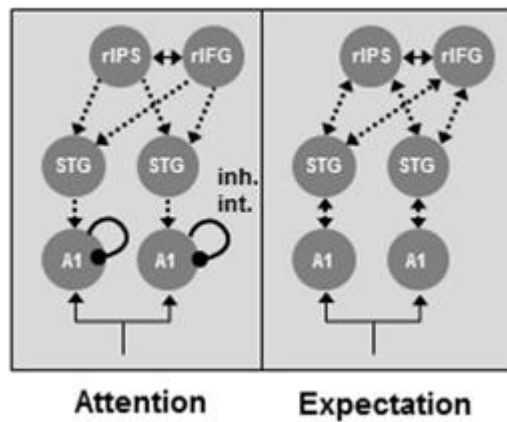
Extrinsic modulation

Intrinsic modulation

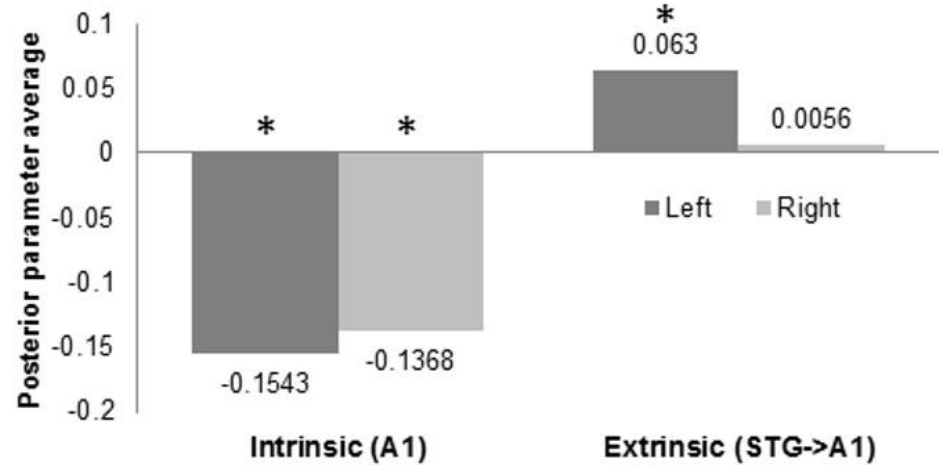


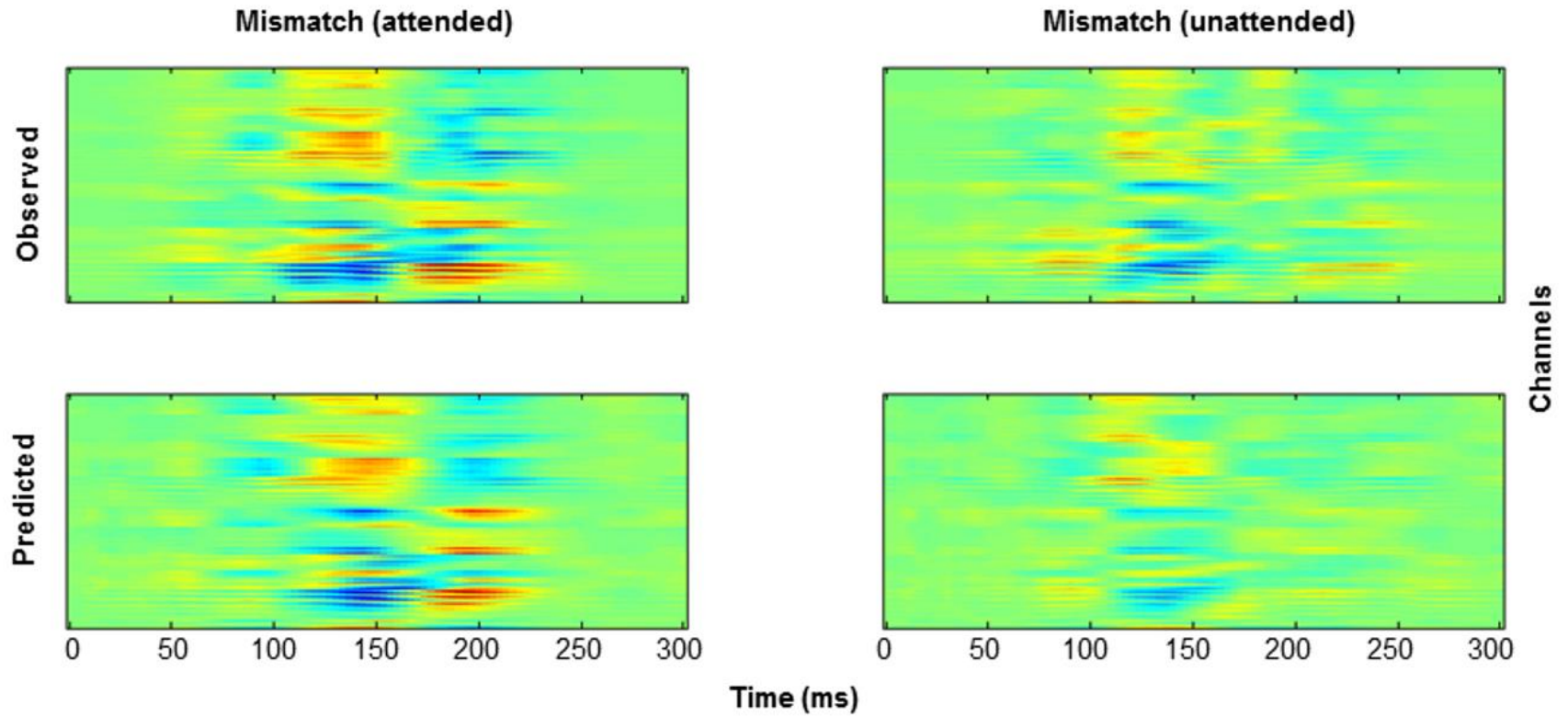


Winning model

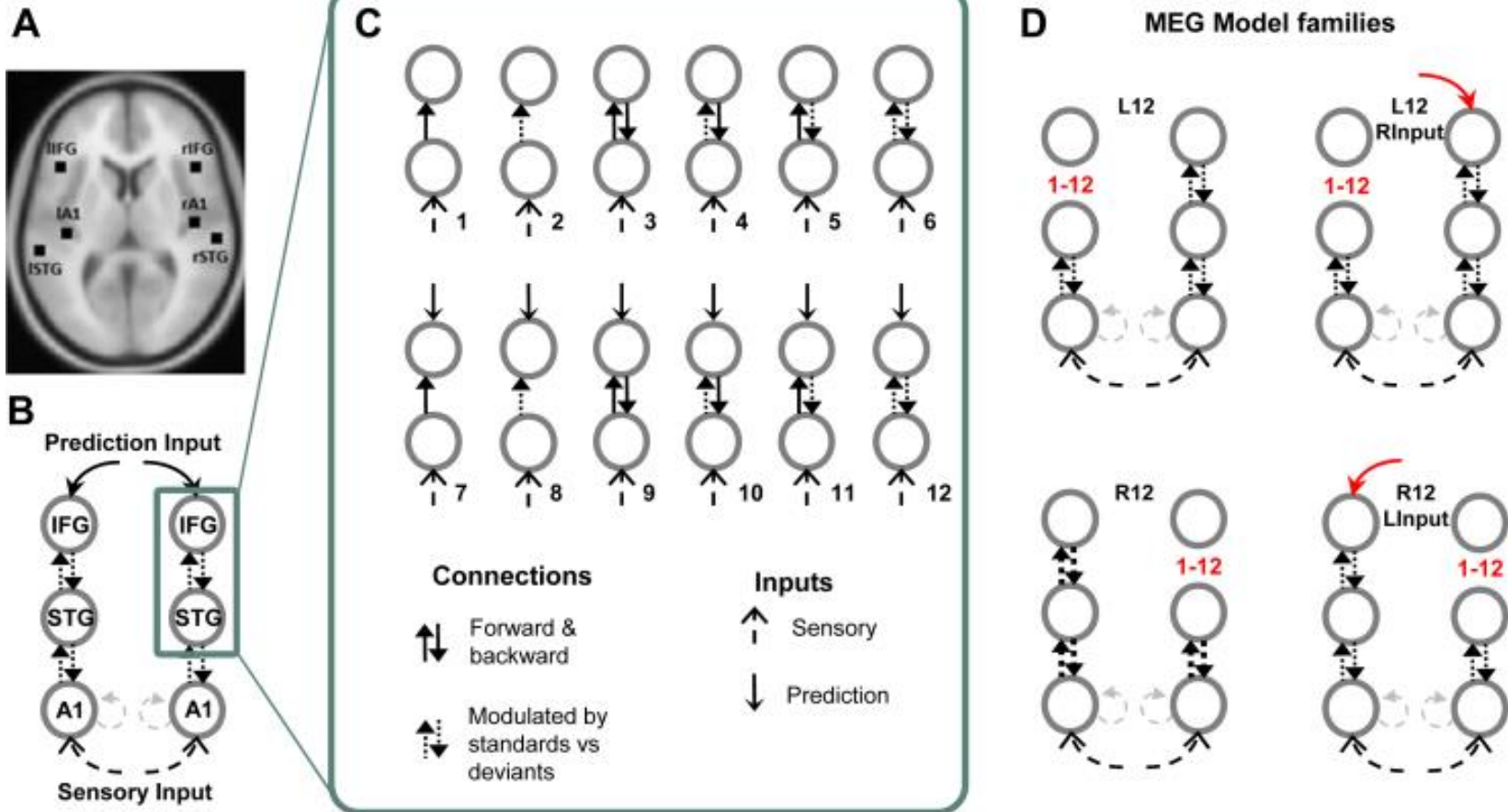


Parameter inference



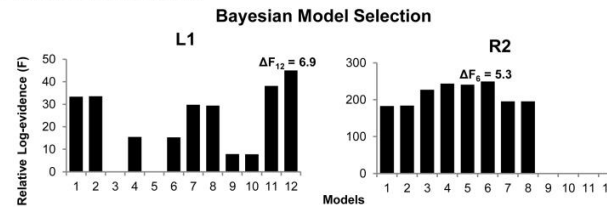


Example #5: Same paradigm, different data

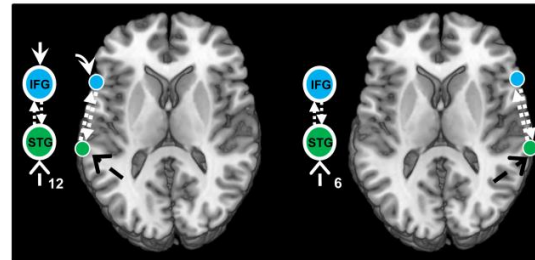


Example #5: Same paradigm, different data

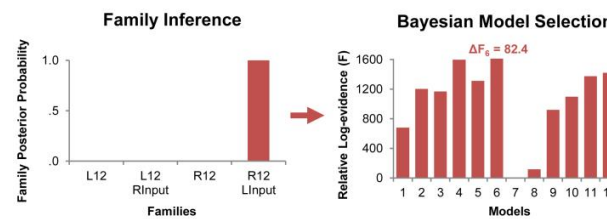
A: ECoG DCM results



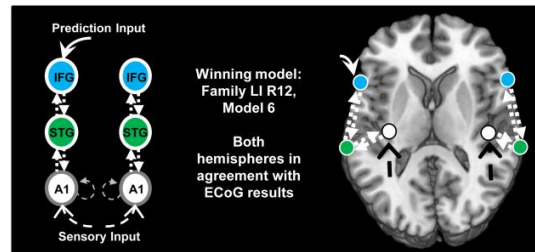
B



C: MEG DCM results

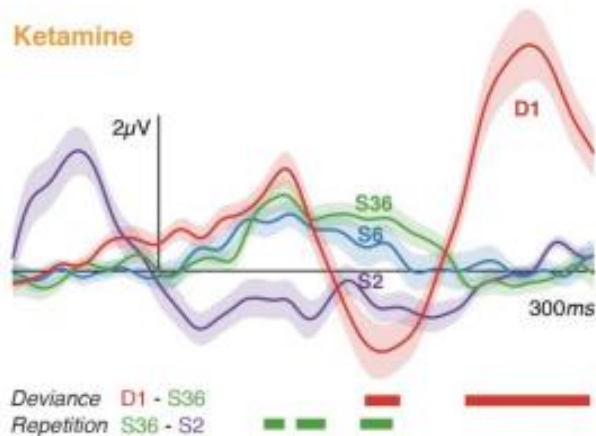
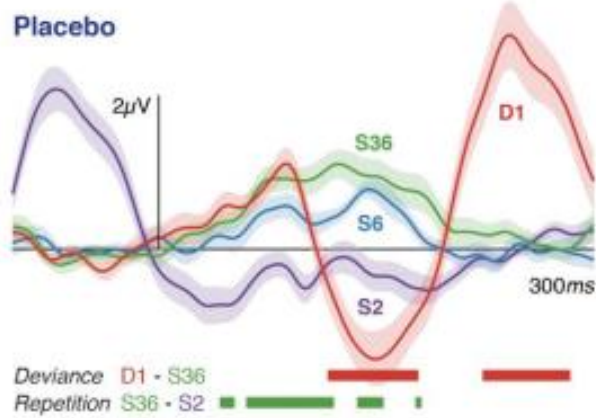


D

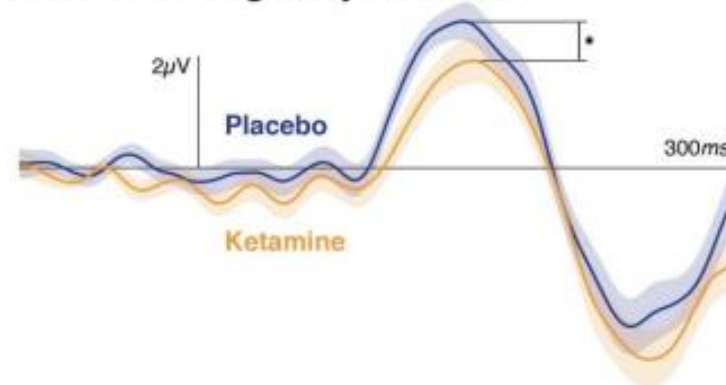


Example #6: Hierarchical modelling

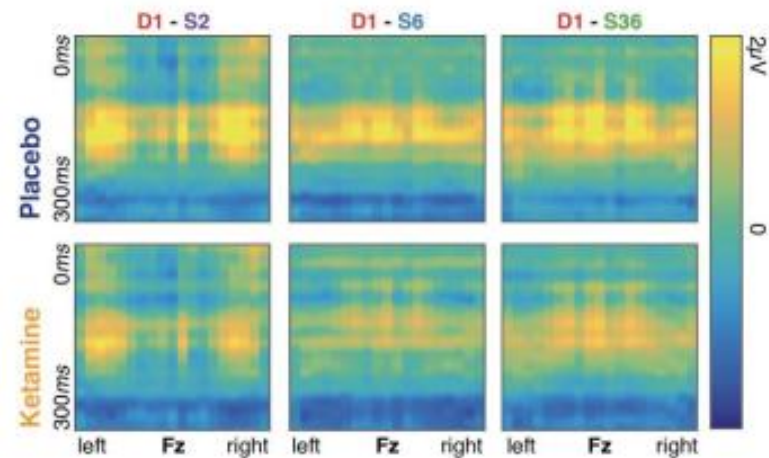
A Evoked response potentials at Fz



B Mismatch negativity waveform

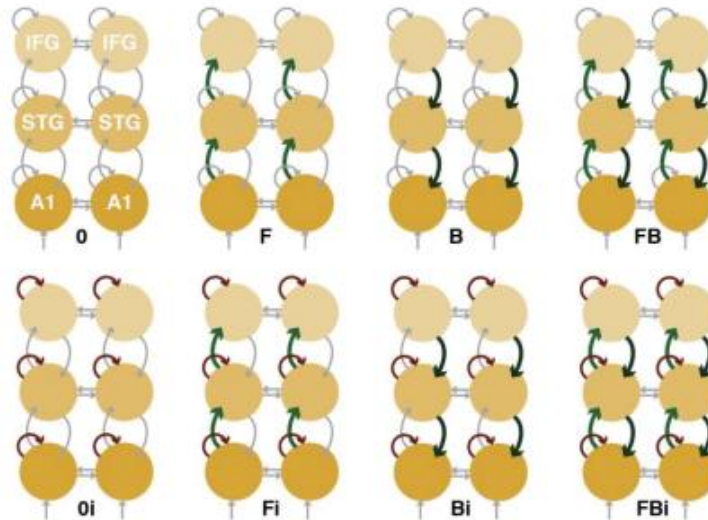


C Scalp topography of mismatch responses



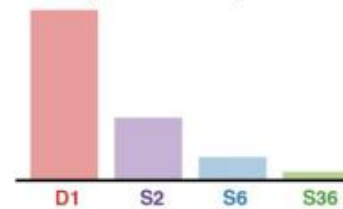
Example #6: Hierarchical modelling

A First level model space: Effects of repetition



Parametric effects of repetition

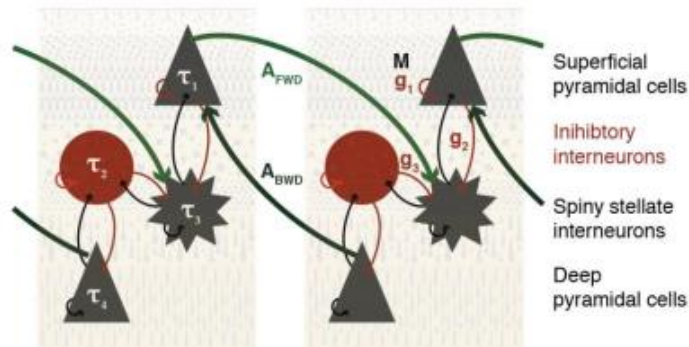
Monophasic Decay



Phasic Effect



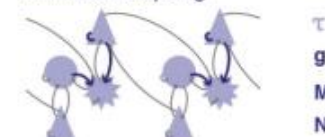
B Second level model space: Effects of ketamine



Extrinsic coupling

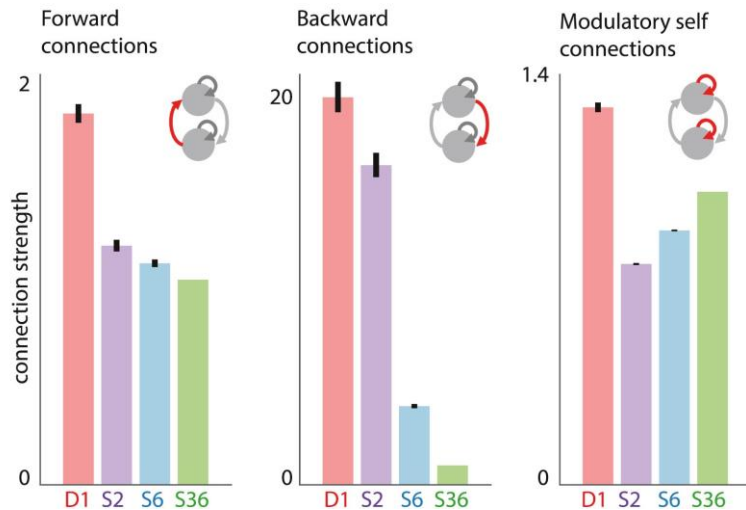


Intrinsic coupling

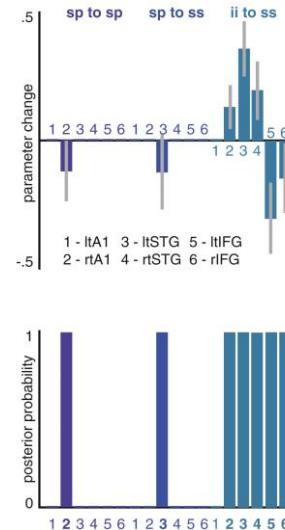


Example #6: Hierarchical modelling

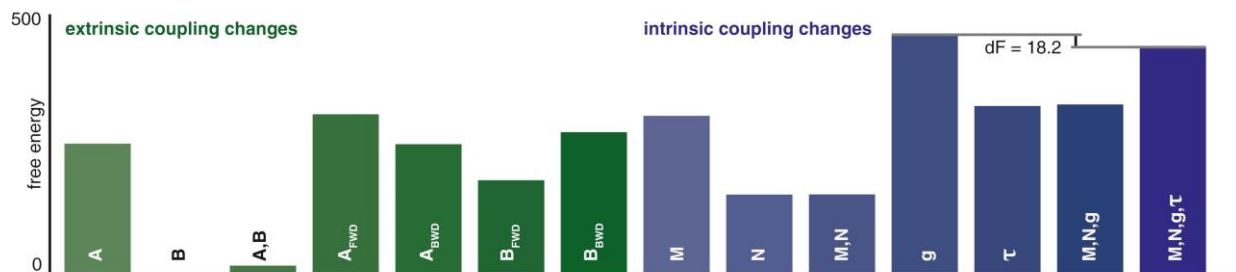
Parameter changes during repeated exposure



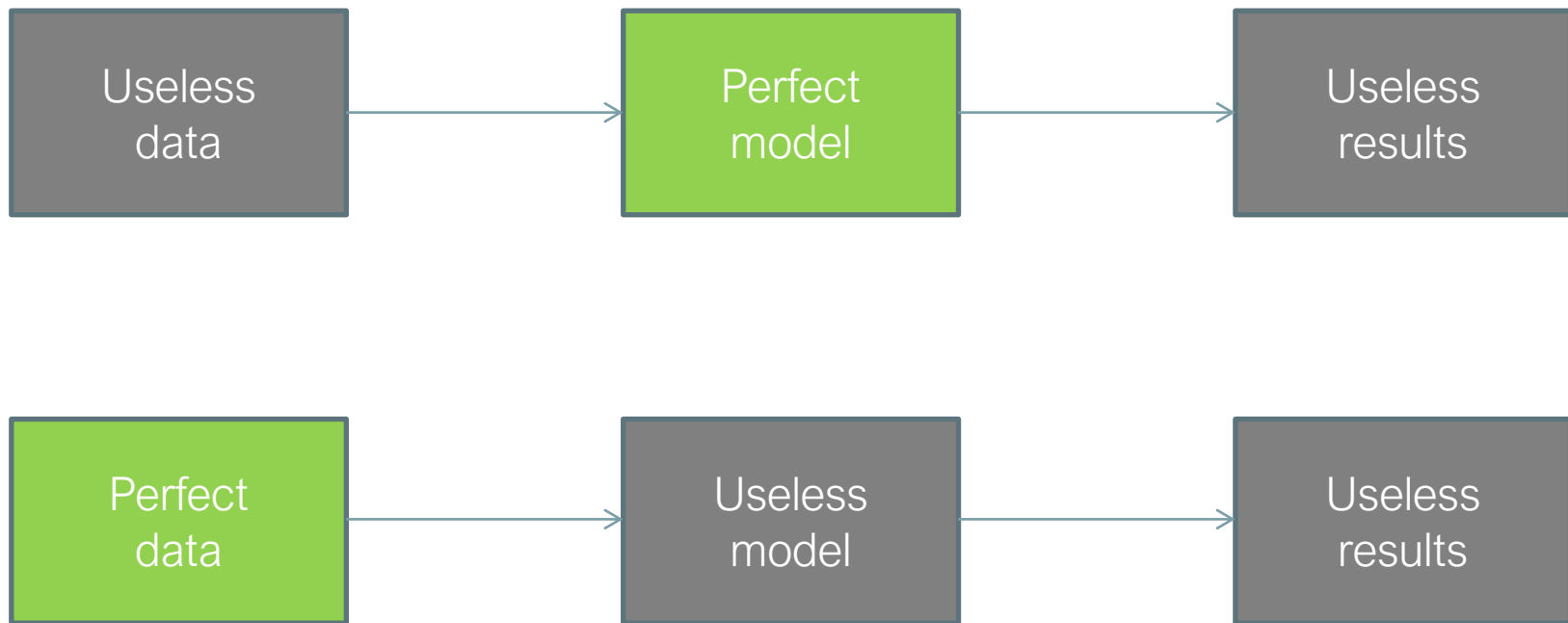
Induced parameter changes



Bayesian model comparison on reduced models explaining ketamine effects



Motivate your assumptions!



References

Overview

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Model specifics

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Group inference

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Examples

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Thank you!

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