Sundeep Teki

UCL

A network analysis of phonemic perception in patients with persisting aphasia using Dynamic Causal Modeling

Auditory Group (PI: Tim Griffiths)
Wellcome Trust Centre for Neuroimaging

Acknowledgments

Alex Leff

Gareth Barnes

Will Penny

Paul Iverson

Zoe Woodhead

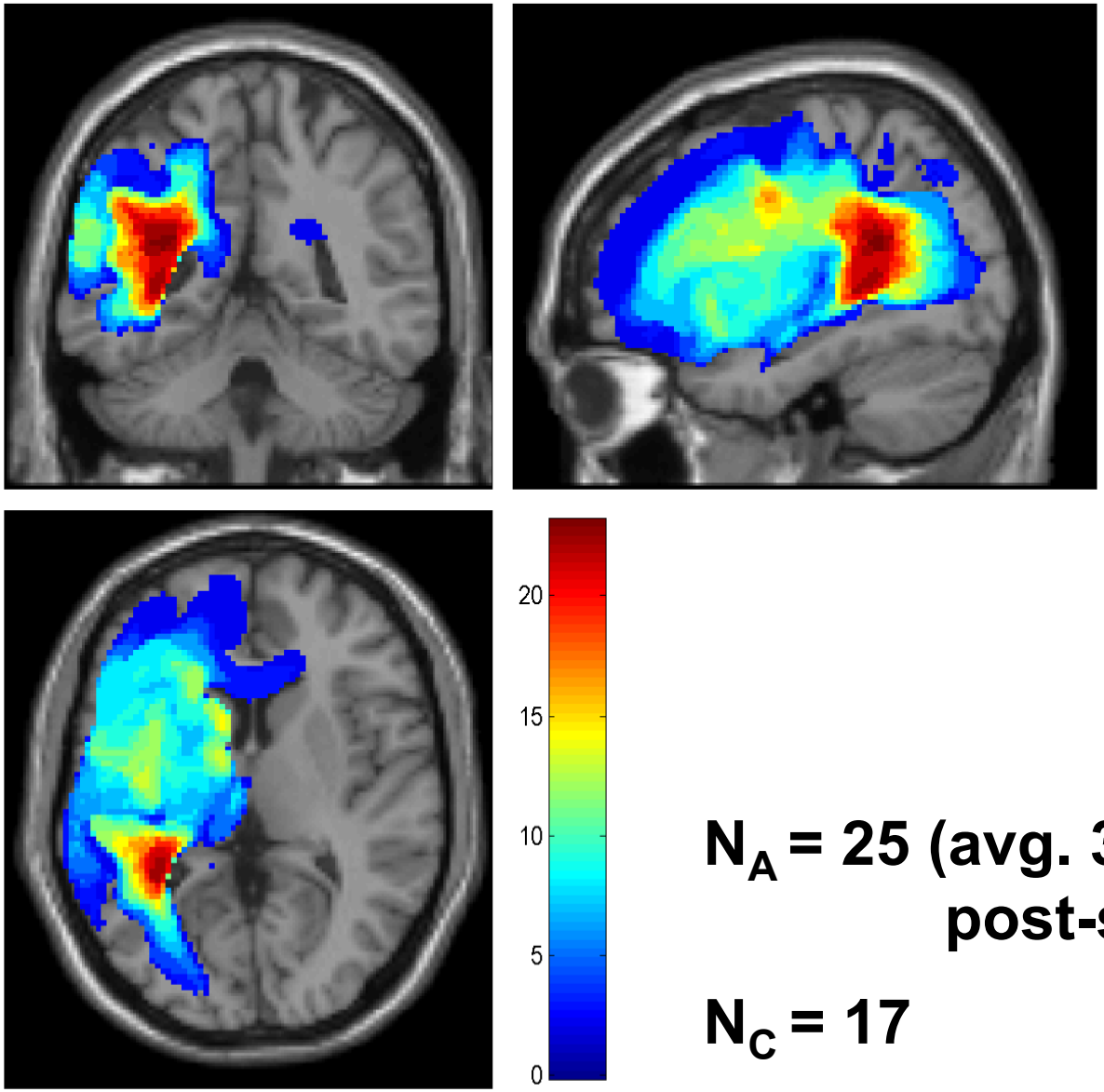
Tim Griffiths

wellcome trust

Take-home message

- Aphasics *do* show robust speech mismatch responses.
- MEG source-space responses indicative of reorganization from left to right hemisphere in aphasics.
- DCM analysis of MEG data suggests distinct speech networks for aphasics vs. controls.
- Speech comprehension deficits in aphasics can be explained by a predictive coding theory of brain function (cf. Friston).
- Left-STG => Right-STG connection strength in aphasics predicts behaviour on phonemic perception tests.

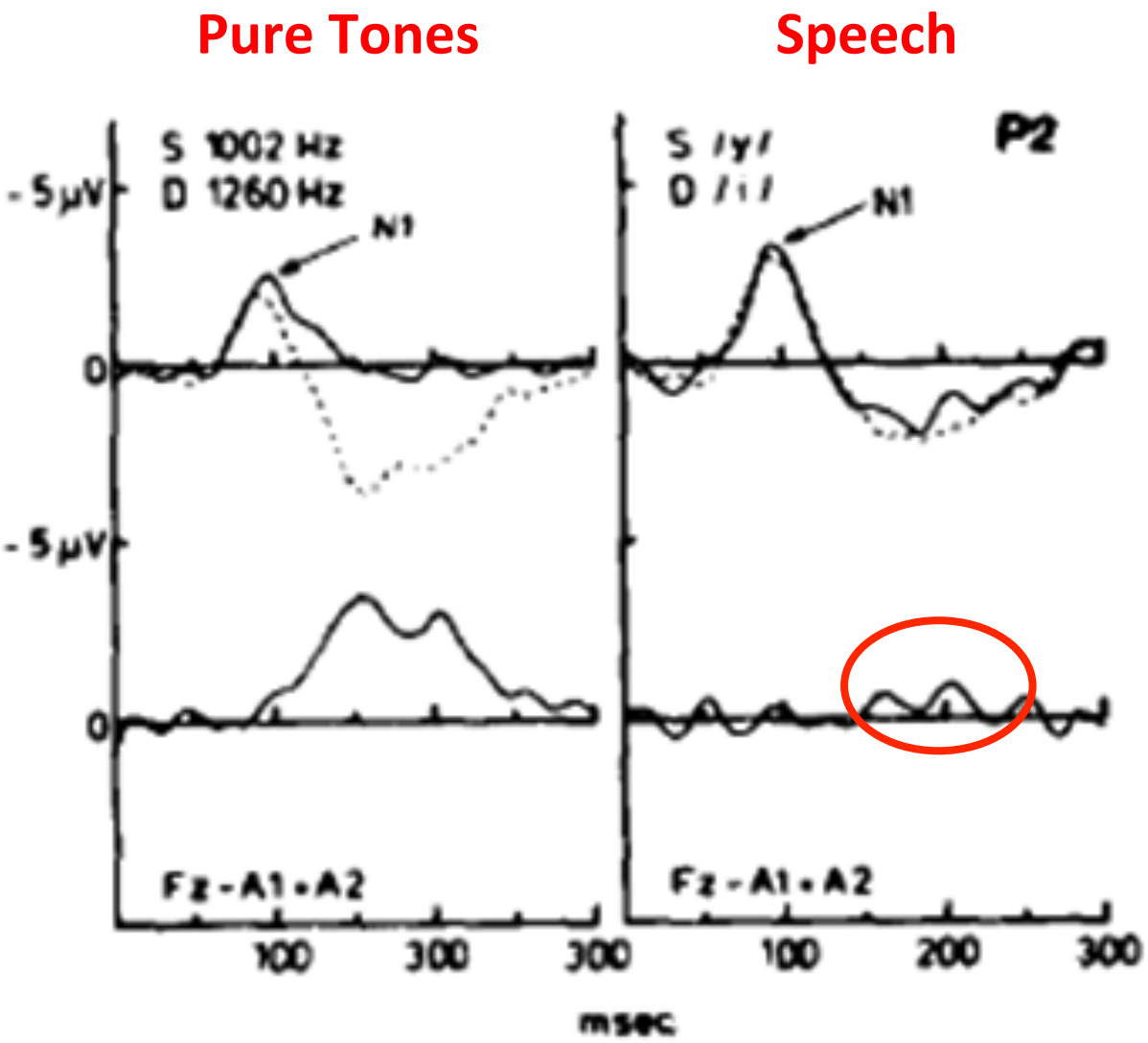
Aphasia



N_A = 25 (avg. 3.6 years post-stroke)

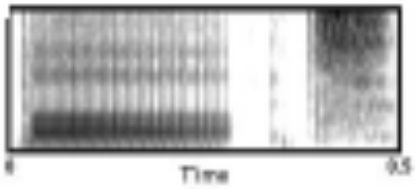
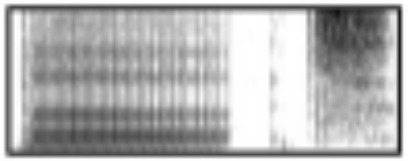
N_C = 17

Aphasia & MMN



Aaltonen et al., 1993

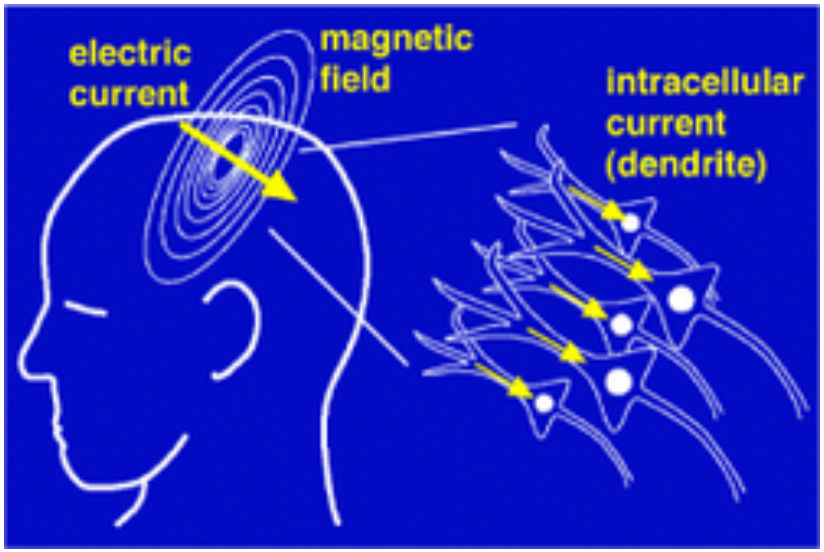
Stimuli

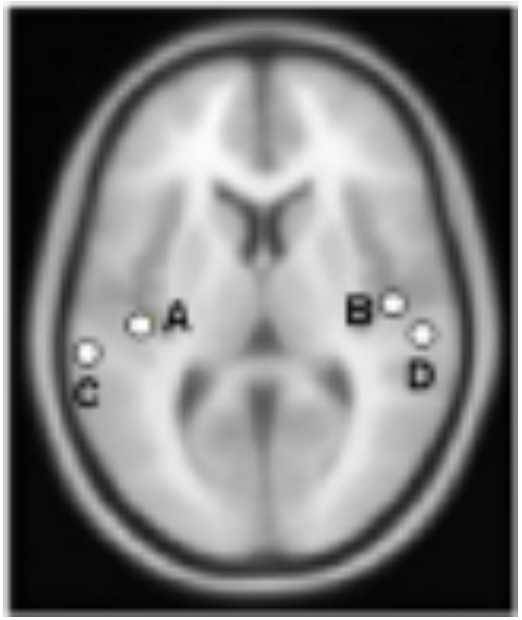
Vowel Stimulus	Percept	Vowels	Formant Frequencies F1 (Hz)	F2 (Hz)	Distance from Standard (ERB)
STD	"Bart"		628	1014	0
D1	"Bart"		565	1144	1.16
D2	"Burt"		507	1287	2.32
D3	"Beat"		237	2522	9.30

D1: **acoustic deviant** (same vowel category)
D2 & D3: **phonemic deviants** (different vowel type)

D2&D3 vs. D1: **phonemic contrast**

MEG





- 274 channel MEG (CTF)
- Fs = 480 Hz
- ISI = 1.08s

- STD:DEV = 4:1, ~60dbSPL
- # Deviants = 120 x 3

- Concurrent visual task

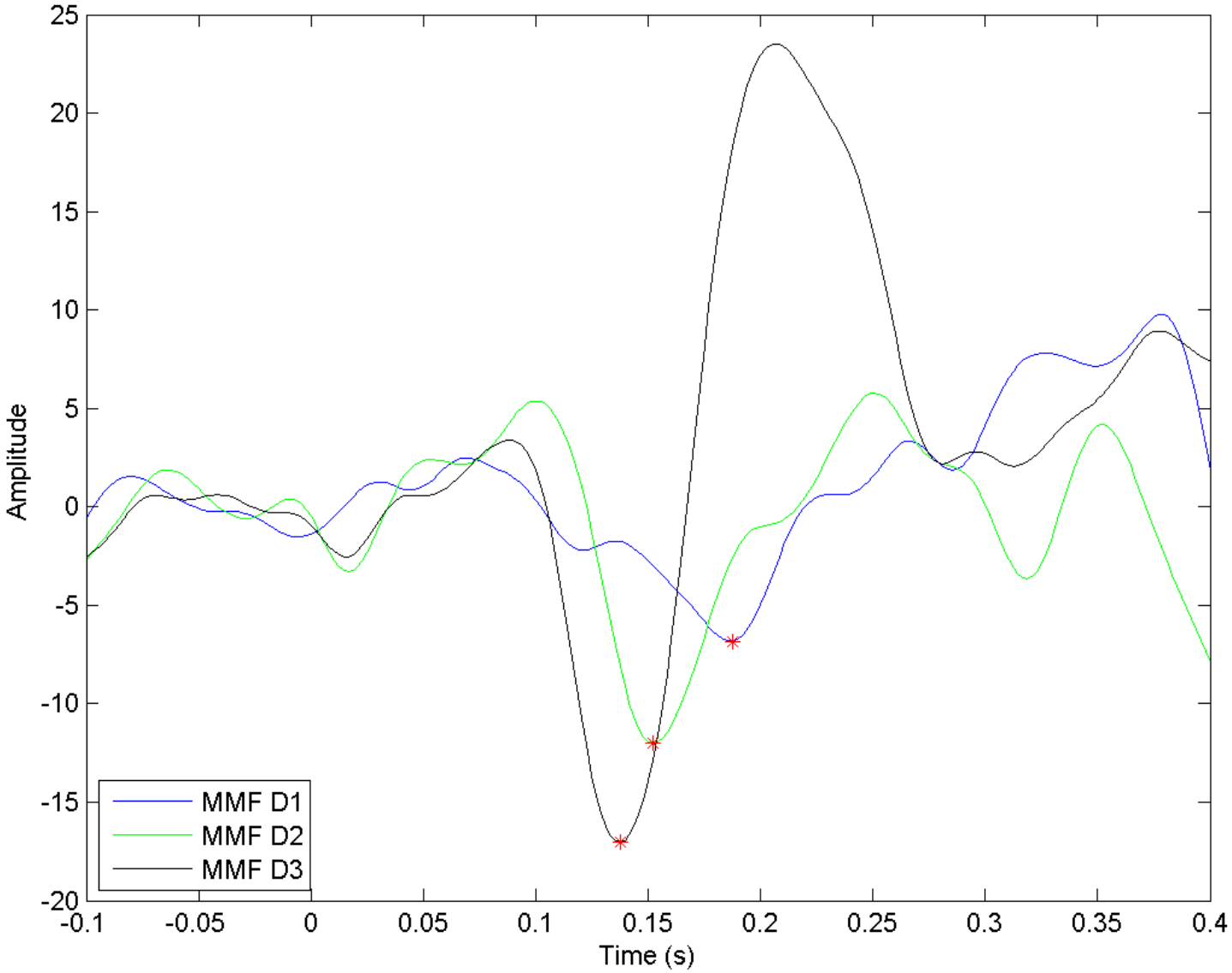
- Best model: 4 sources

  bilateral A1 & STG

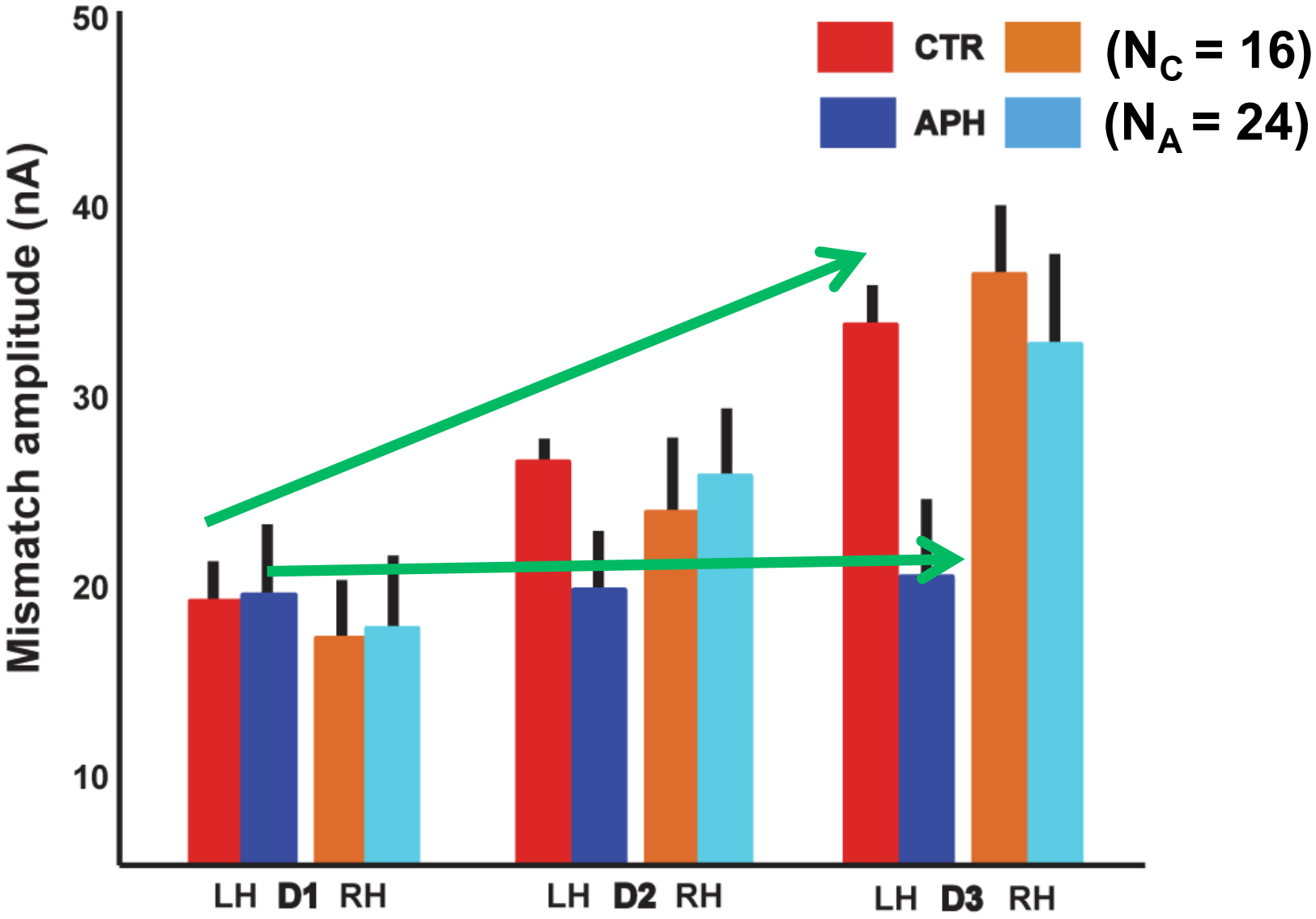
- Aphasics sources constrained by lesion topography

Kiebel et al., 2008

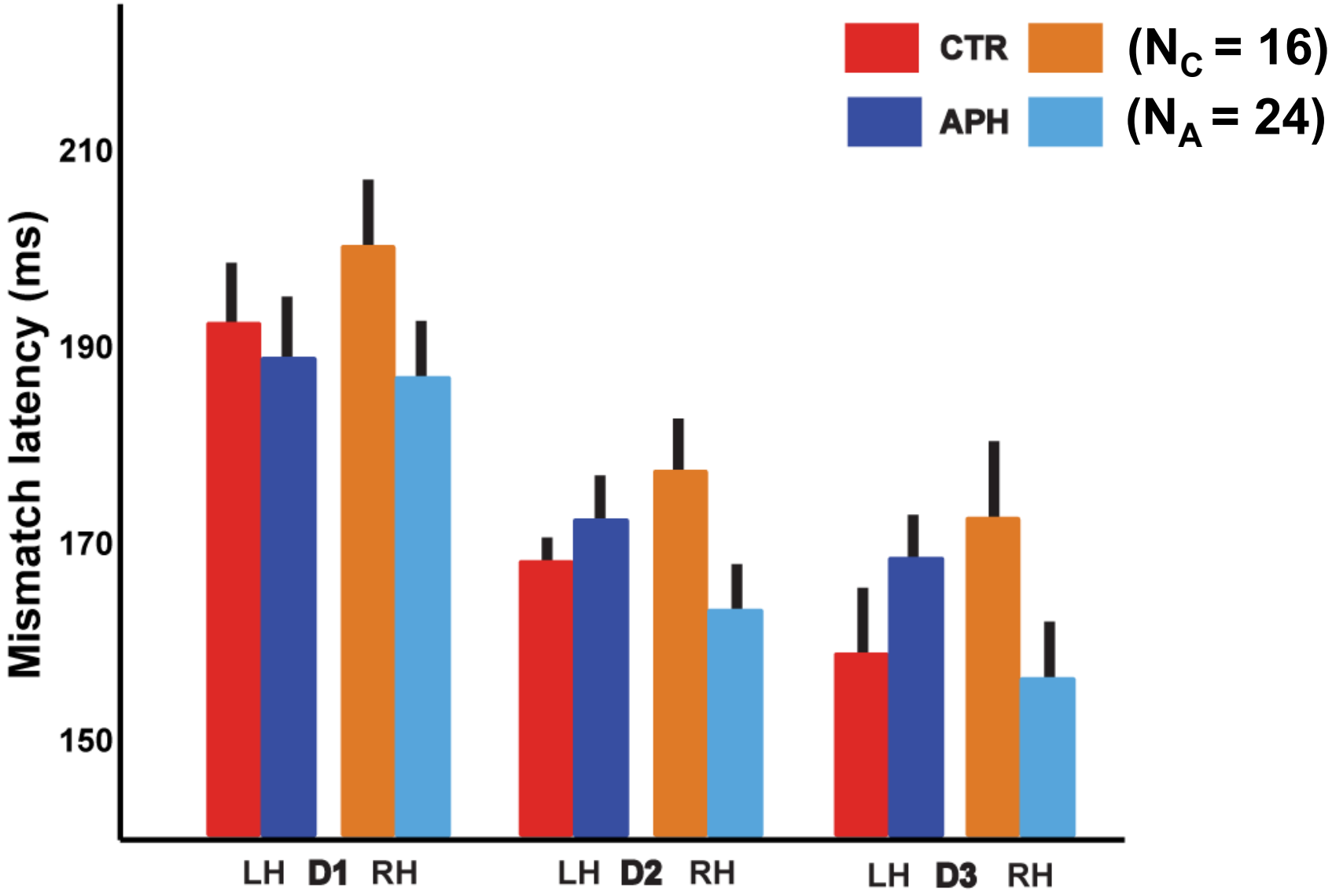
Source-space MMN responses



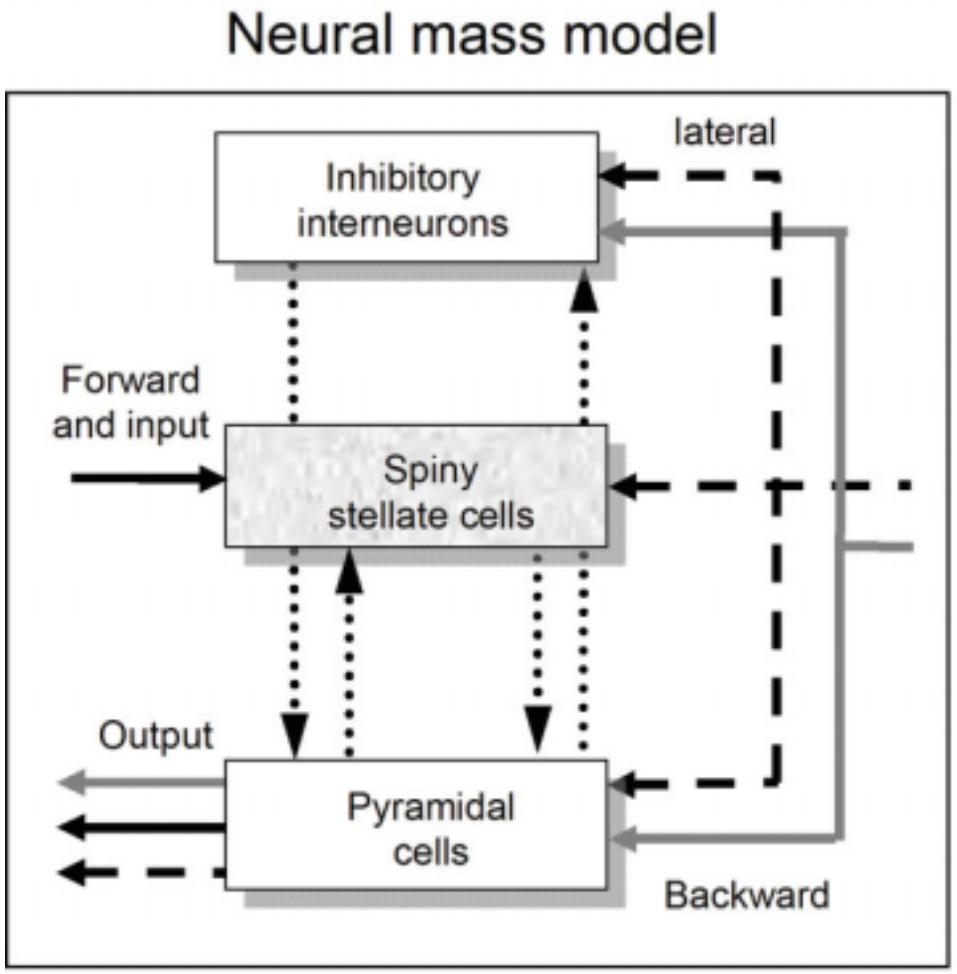
MMN amplitude

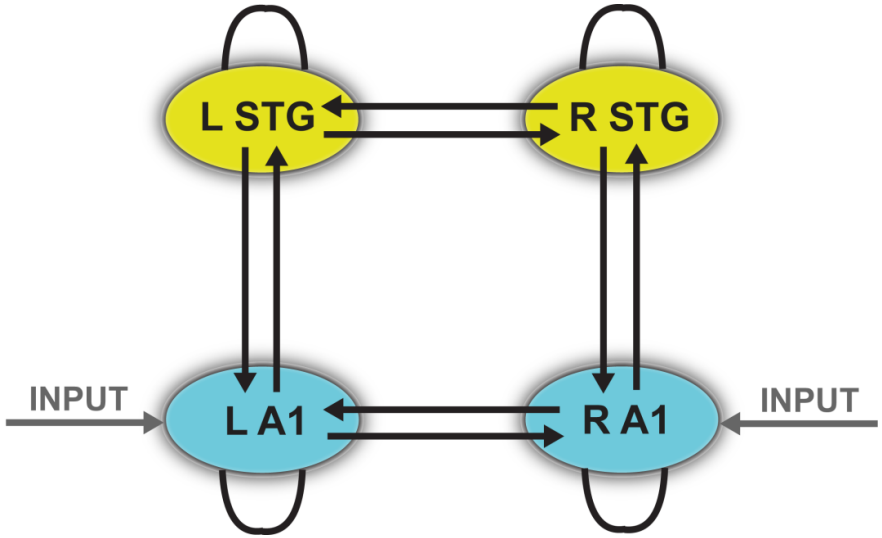


MMN latency


CTR
APH
(N_C = 16)
(N_A = 24)
Mismatch latency (ms)
210
190
170
150
LH D1 RH
LH D2 RH
LH D3 RH

DCM for evoked MEG





Jansen and Rit, 1995
Felleman & Van Essen, 1991

DCM analysis

- **Predictive coding: (Kiebel & Friston, 2009)**

  Prediction error = Predictions - Sensory input

- **Self-connections:**

  sensitivity or precision of neural response to sensory input

- **Forward connections:**

  bottom-up propagation of prediction error from lower to higher level of the hierarchical system

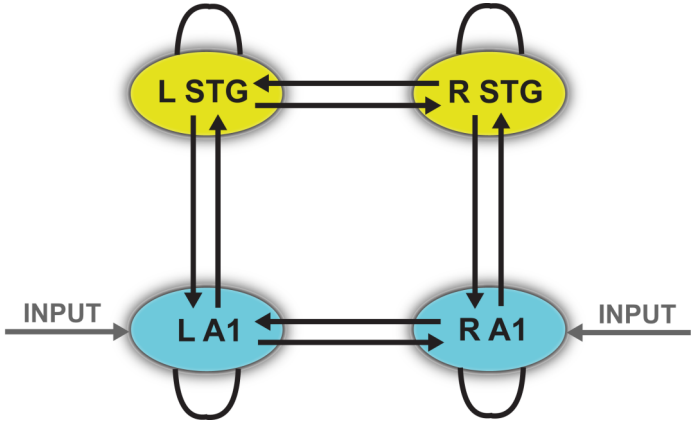
- **Backward connections:**

  top-down predictions from higher to lower levels.

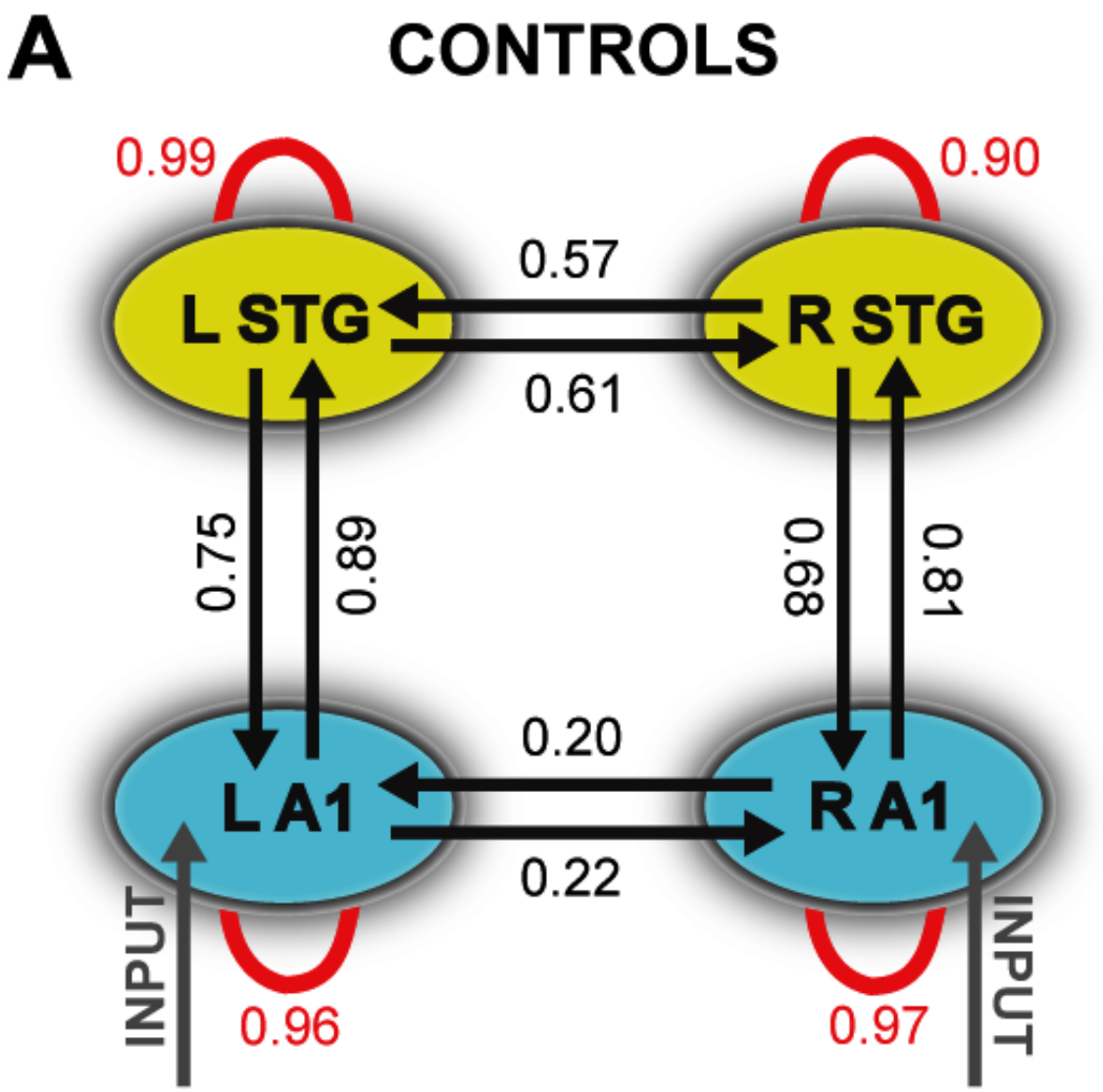
DCM analysis

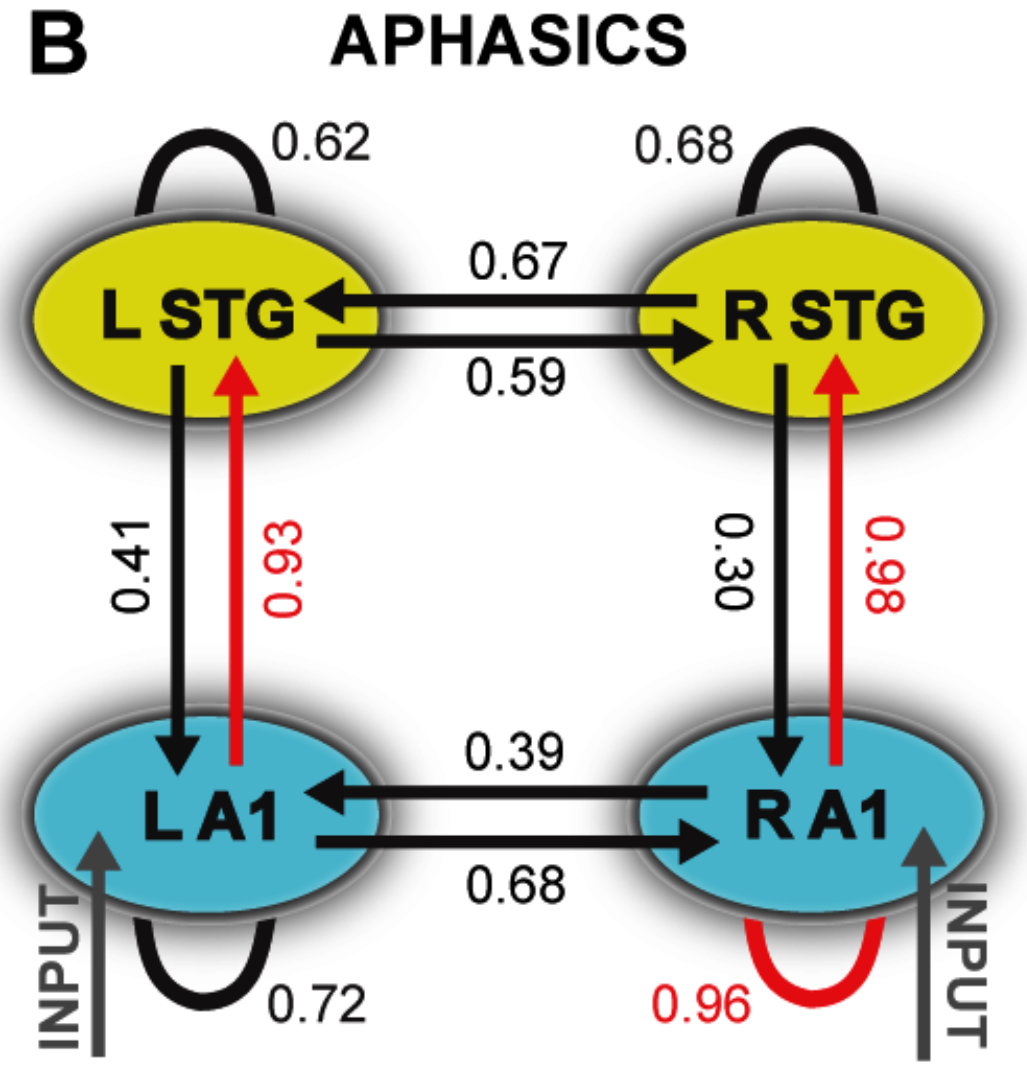
Aim: To investigate modulation of the connections as a function of phonemic deviancy: (D3 & D2) vs. D1

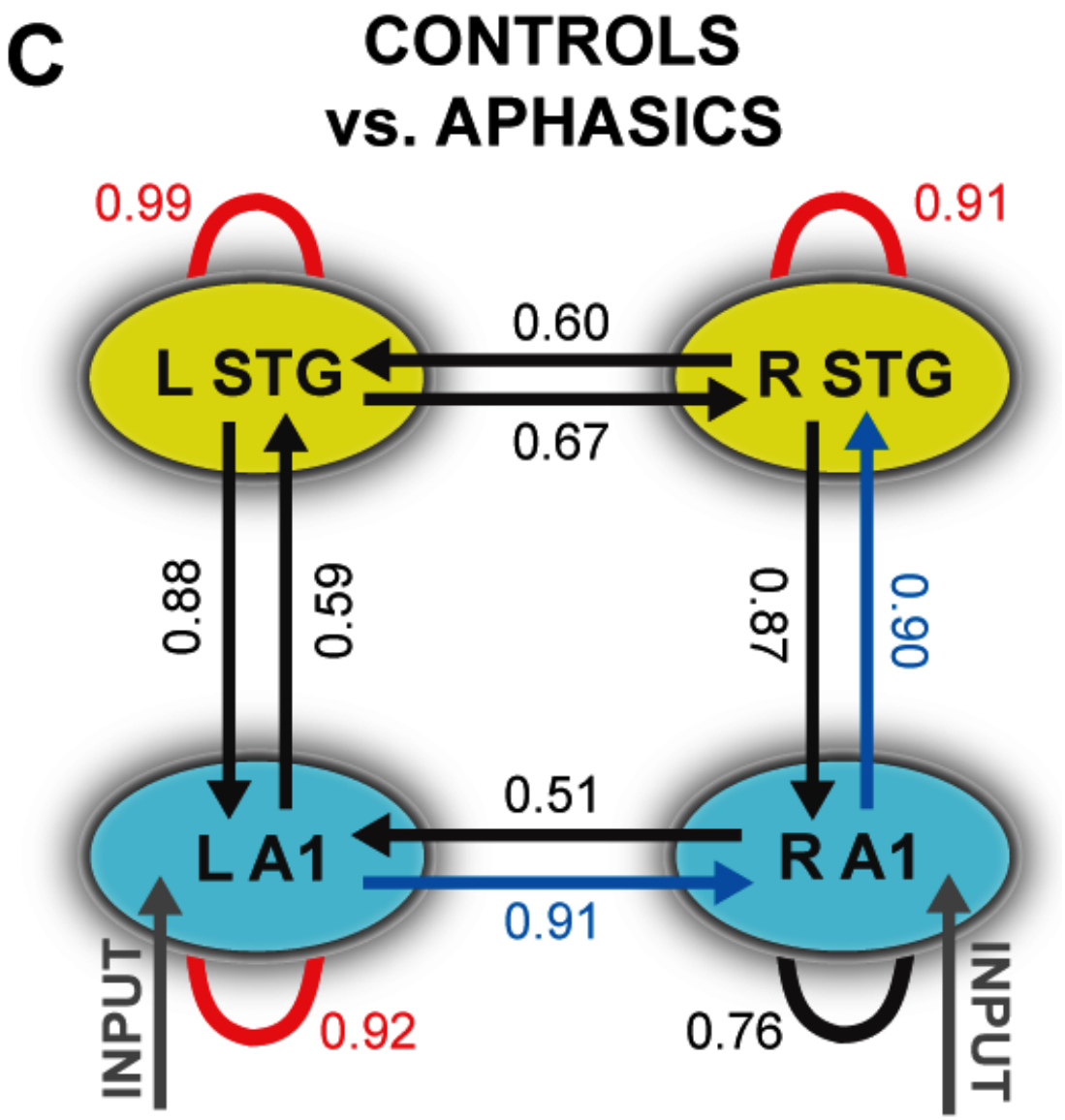
Models: 12 connections between A1 and STG were modelled, yielding 255 models for each participant.



Hypotheses: aphasics may show deficits at the higher level of the network (STG) and impaired left hemisphere function.


A
CONTROLS
0.99
0.90
L STG
R STG
0.57
0.61
0.75
0.89
0.89
0.81
L A1
R A1
0.20
0.22
INPUT
INPUT
0.96
0.97


B
APHASICS
0.62
0.68
L STG
R STG
0.67
0.59
0.41
0.93
0.30
0.98
L A1
R A1
0.39
0.68
INPUT
0.72
0.96
INPUT


C
CONTROLS
vs. APHASICS
0.99
0.91
L STG
R STG
0.60
0.67
0.88
0.59
0.87
0.90
L A1
R A1
0.51
0.91
INPUT
0.92
0.76
INPUT

Summary

- Aphasics *do* show robust speech mismatch responses.
- MEG source-space responses indicative of reorganization from left to right hemisphere in aphasics.
- DCM analysis of MEG data suggests distinct speech networks for aphasics vs. controls.
- Speech comprehension deficits in aphasics can be explained by a predictive coding theory of brain function (cf. Friston).
- Phonemic prediction errors and prediction signals may have different oscillatory signatures (cf. Poeppel/Giraud)
- **Next:** longitudinal analysis following drug/phonological therapy