

BACKGROUND & RATIONALE

- Dysprosodia** - impairment in perceiving social intent via vocal intonation, features prominently in a number of neurological/psychiatric disorders (e.g. Depression, Parkinson's Disease, Autism Spectrum Disorder, and Schizophrenia). **The neural mechanisms of prosody are poorly understood**
- Cue saliency** refers to the relative presence or absence of particular acoustic features that serve as cues for differentiating emotion. For example, high pitch variability ($F0_{SD}$) is a powerful cue for identifying happiness, while low $F0_{SD}$ signals fear. Similarly, high spectral energy (HF_{500}) is strongly correlated with anger perception
- Previously, using fMRI and a parametric mode, we delineated a temporo-frontal reciprocal circuit for prosodic processing
- We observed that cue saliency (ZCUE) increases were correlated with increased BOLD activation in superior temporal /middle gyrus and amygdala, while decreases in ZCUE correlated with increased frontal activity within IFG

APPROACH

- Temporo-spectral ROI analysis:** A cue-saliency weighted temporo-spectral map at each component
- ROI-2-ROI Connectivity:** Reciprocal activation pattern and connectivity within 9-component prosody network

→ Here we show right Superior Temporal and Inferior Frontal Gyrus (**rSTG** **rIFG**)

Hypothesis: The more emotionally salient the prosodic signal is the more consistent the brain response → high cross trial phase synchrony (PLV see box below). The more ambiguous the stimulus is the more variable the response across trials → low PLV

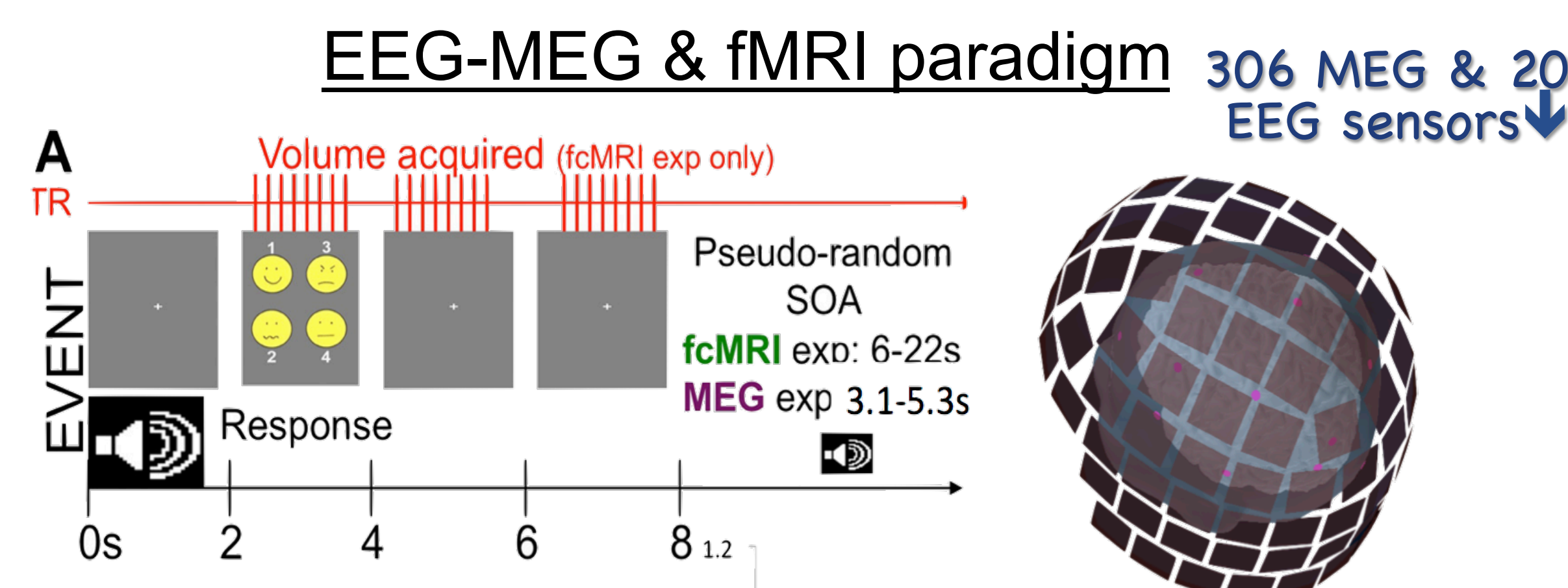
Target- Gamma band oscillations (30-80Hz)

Rationale: 1) Computational models suggest that gamma frequency reflects excitatory-inhibitory balances that allow for local cortical pyramidal columns to act in unison. 2) Prior studies link fMRI-BOLD activation to Gamma synchrony

Subjects: 18 healthy controls (13 males 24.86±7.31 years of age; Education: 14.86±1.81 years)

Task: EEG&MEG: 15 repetitions of 26 (8, happy and fear 10 anger) stimuli = 390 stimuli, divided into five 5-minute blocks
Stimuli: sentence 0.8 to 2 seconds long (i.e. "its eleven o'clock") (see figure above)

Analysis Pipeline: Data: 2.3 second epochs reflecting individual trials (Max N=390) and decimated to 500HZ. Source modeling: **MNE L2 min-norm** sources weighted by fMRI (see handout or web for further methods)



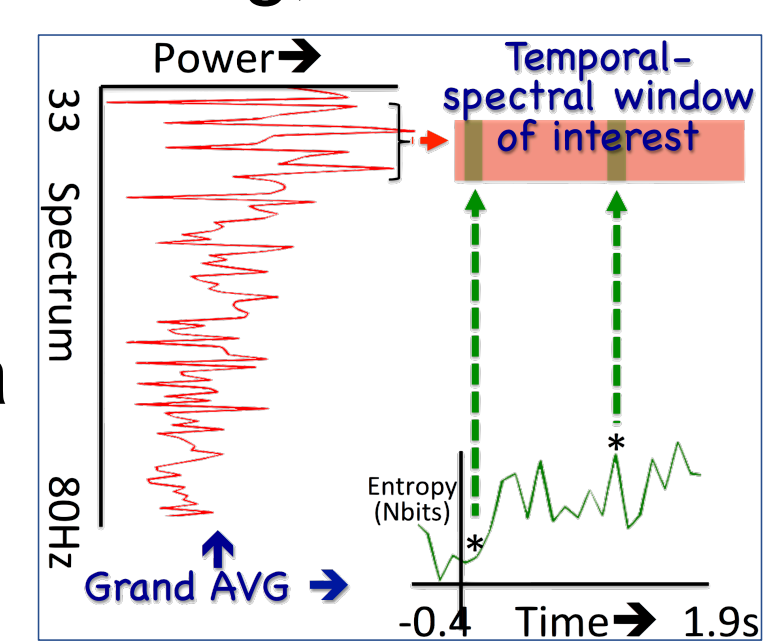
Phase Synchrony across trials (PLV)

- PLV is a coefficient reflecting the degree frequency specific alignment across trials relative to stimulus onset.
- High PLV across trials suggests a high fidelity / reliable response by the neural network. Low PLV would indicate a highly variable response to stimulus.

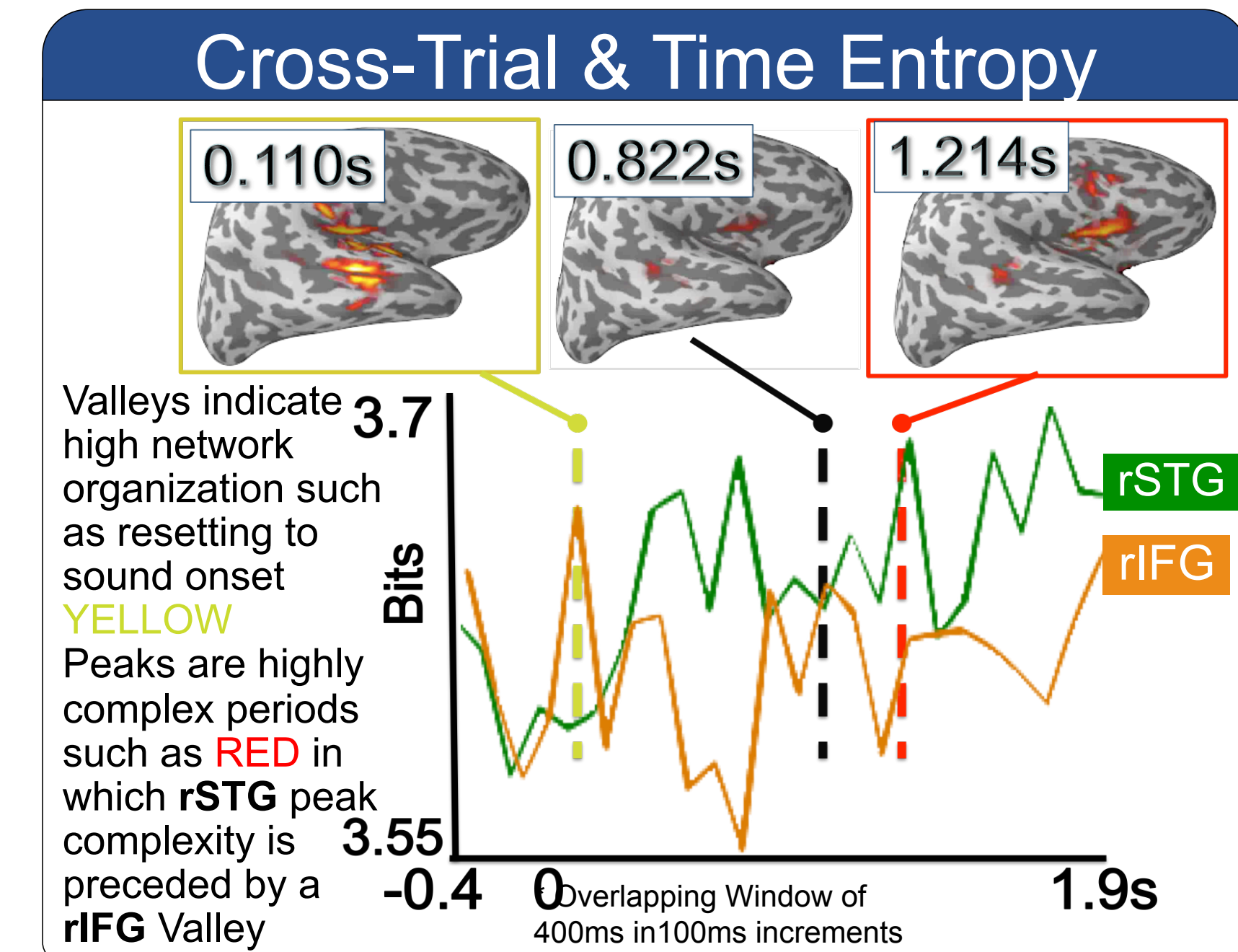
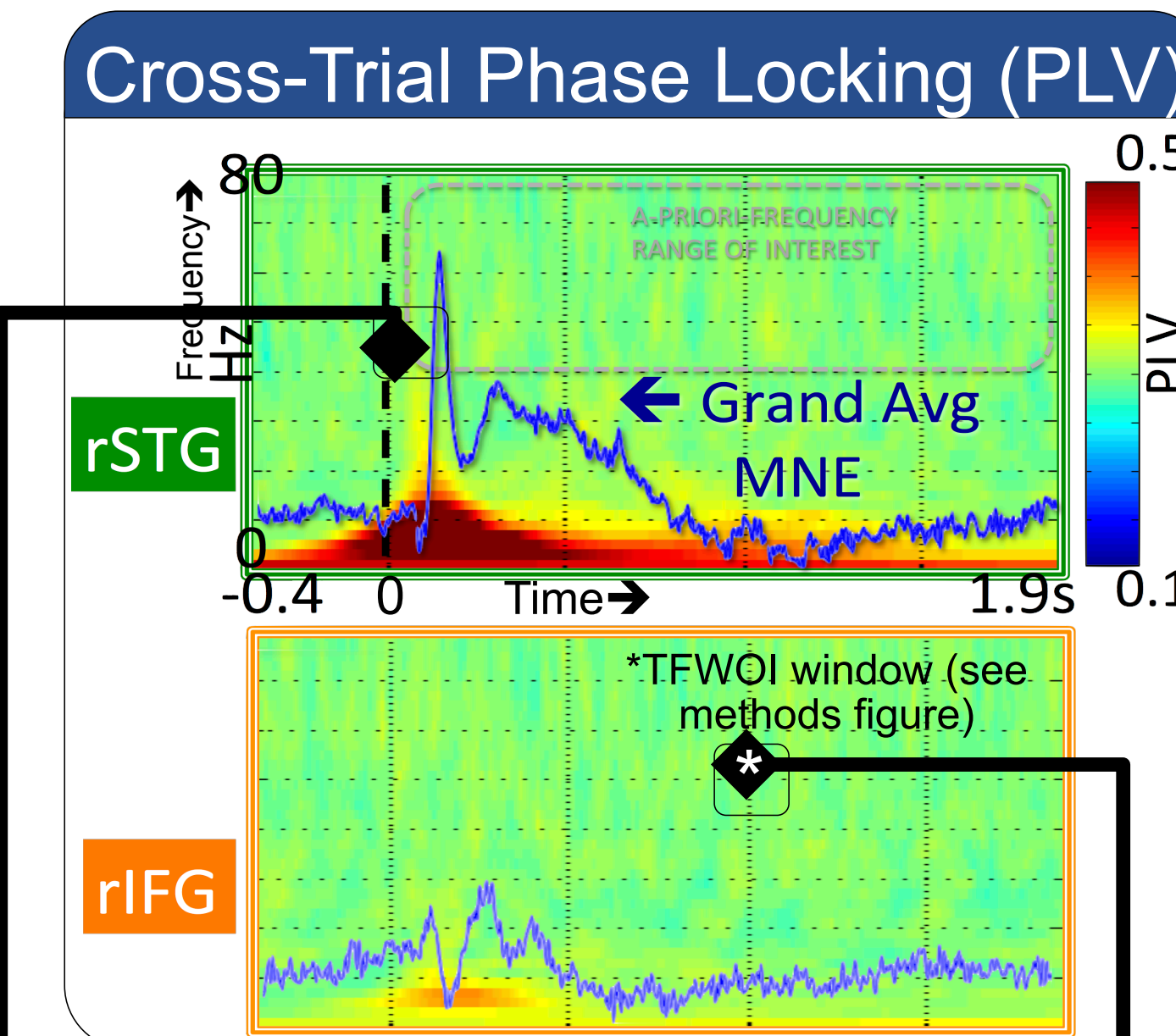
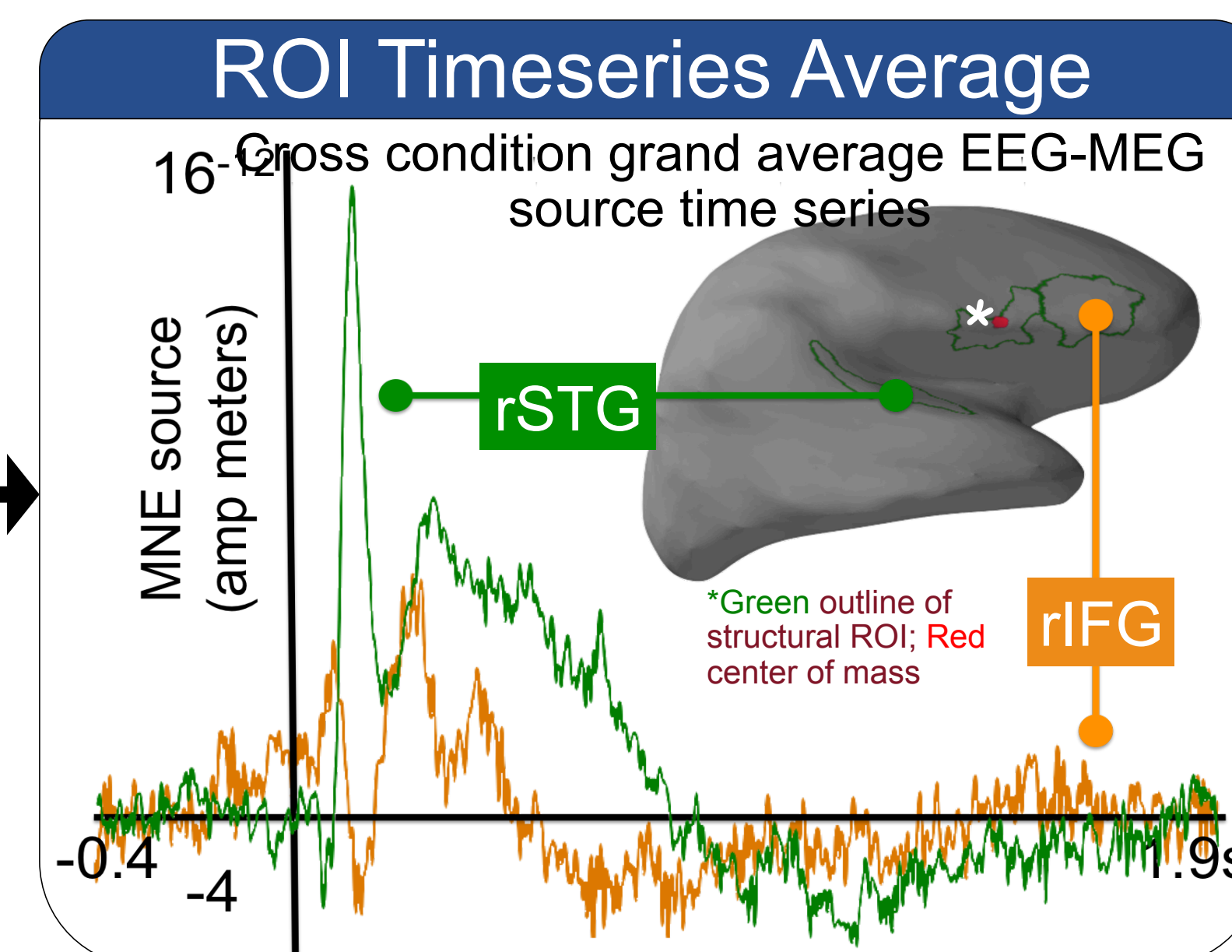
Reducing Data Complexity

Problem: Stimuli and Data are complex: Sentences 2.3 seconds long; 2.3 second TF maps

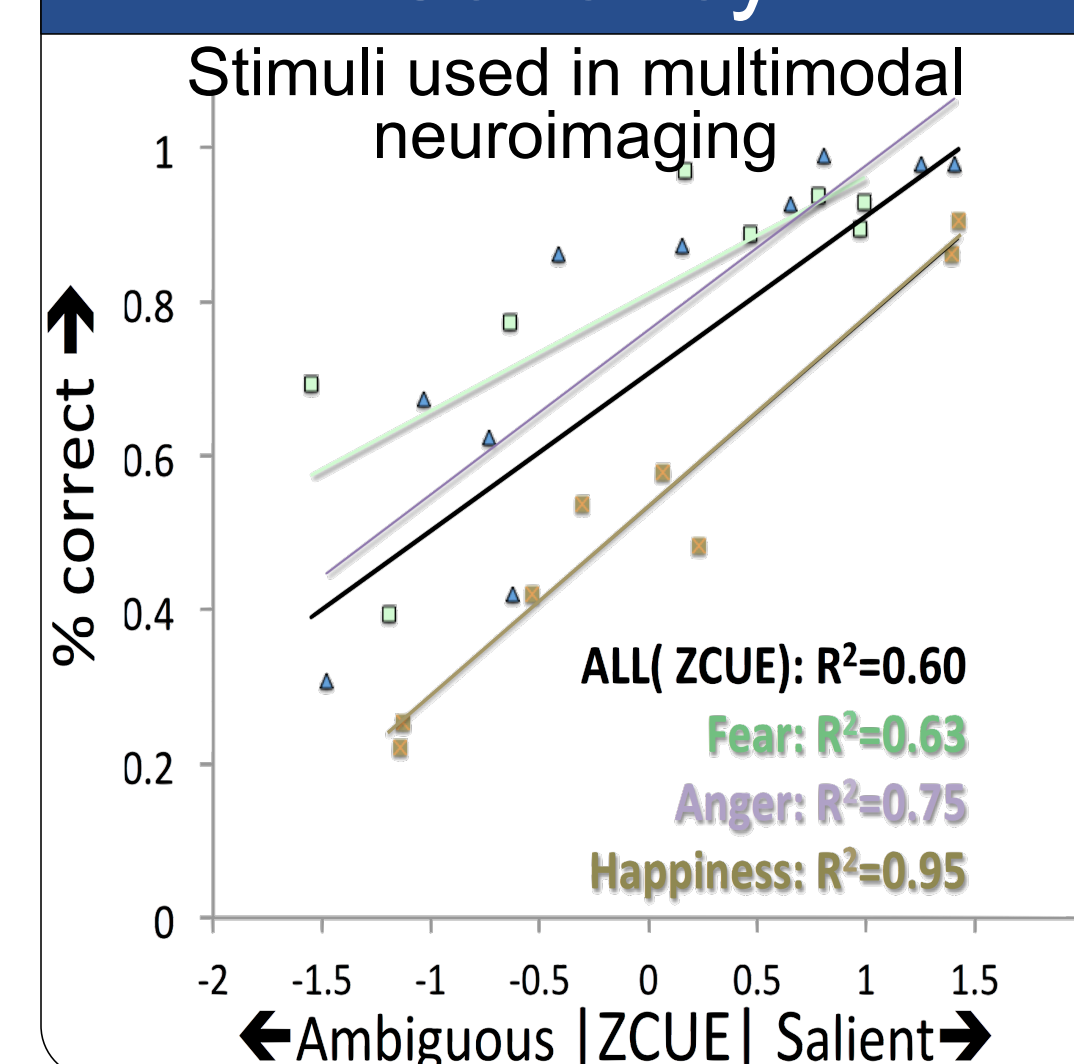
Solution: 1) **Brute Force** cluster correct TF maps at a $p < 0.01$ level using Monte Carlo simulation. 2) Use **information theory** (**entropy** and **mutual information (MI)**) along with overall spectral information to generate TF windows of interest



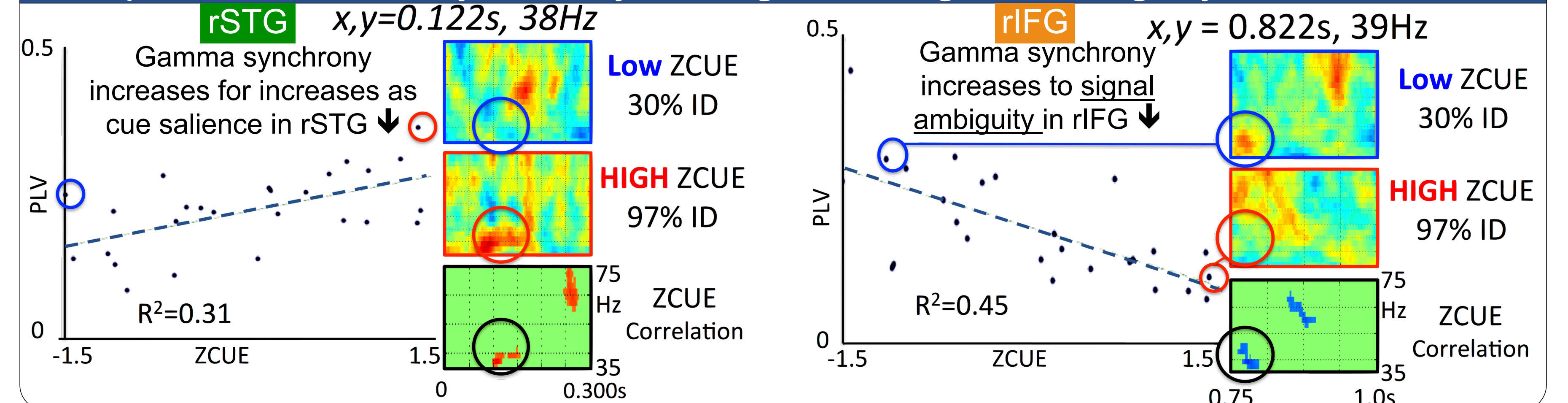
RESULTS



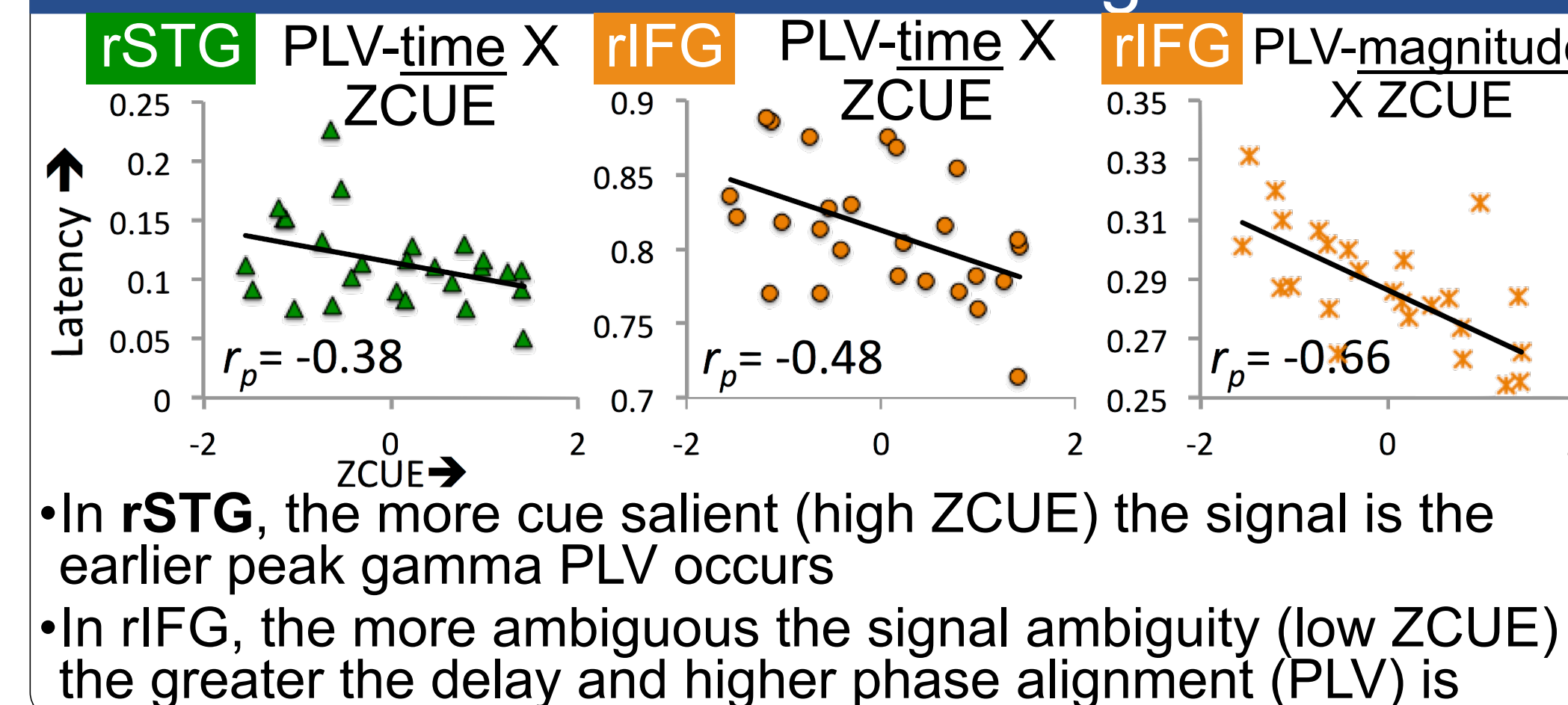
Behavior X Cue Saliency



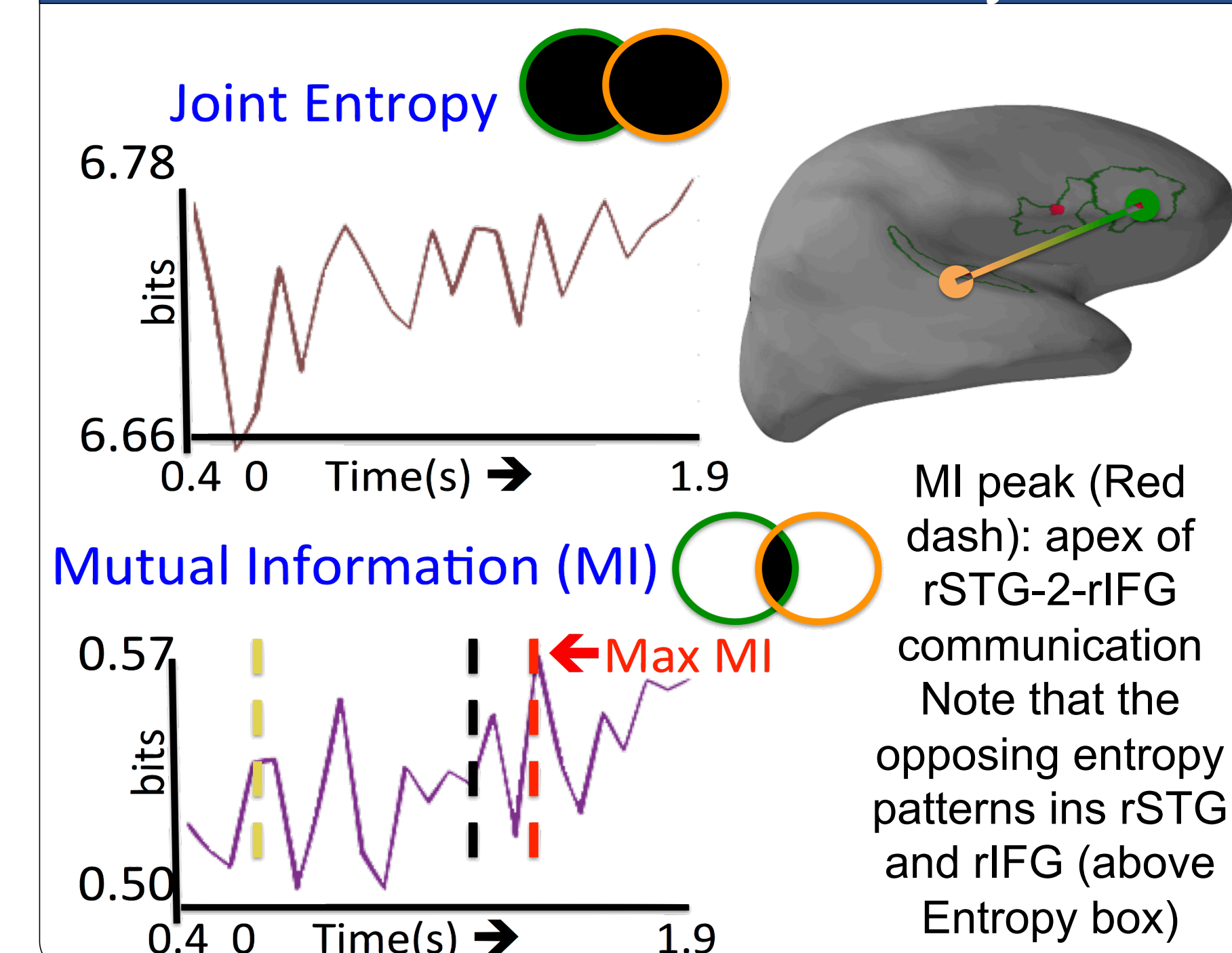
Reciprocal Gamma Synchrony Changes To Signal Ambiguity In rSTG & rIFG



What do ZCUE X PLV changes mean?



ROI-2-ROI Connectivity



for EEG-MEG Prosody MOVIE go to
davidileitman.com/multimodal-neuroimaging-of-prosody/

REMARKS

5-D ROI Activity: Cue-saliency weighted temporo-spectral modulations in gamma band in both rIFG and STG consistent with our temporo-frontal reciprocal circuit model

ROI-2-ROI connectivity: 1) Mutual information between rSTG and IFG were maximal at ~1.2s PSO; 2) MI Peak corresponded to decreased IFG entropy and increased STG entropy consistent with IFG exerting a modulating role on STG

To dos: 1) Extend analysis to all network ROIs; 2) Use diffusion imaging to examine structural-functional relationships