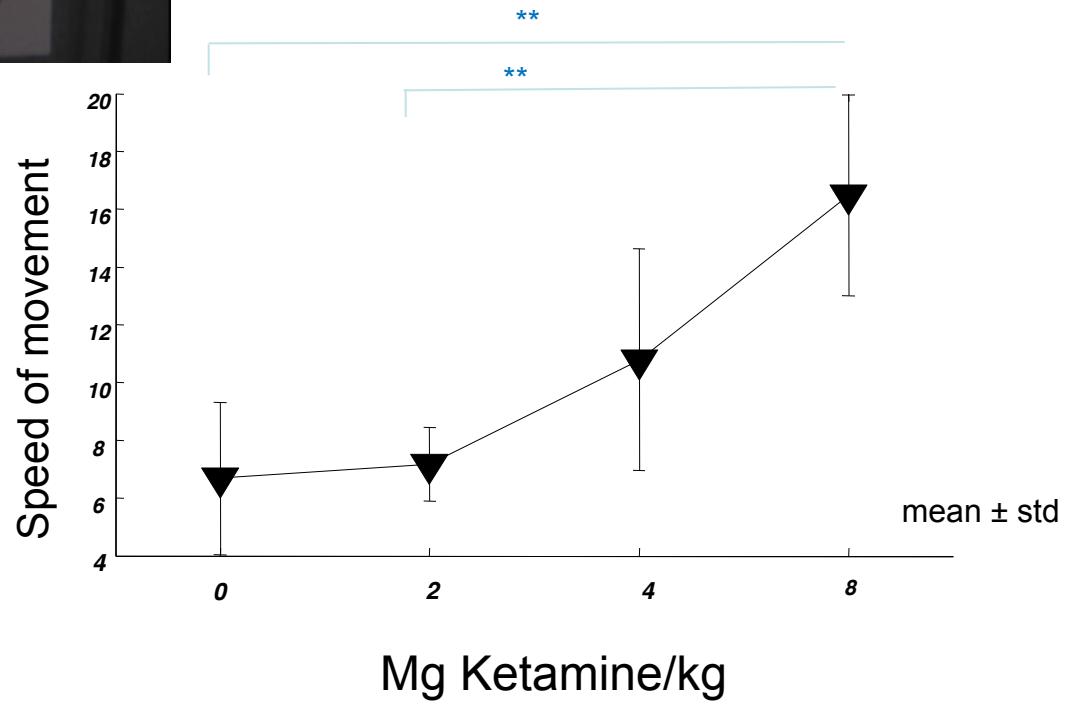


# Dynamic Causal Model for EEG

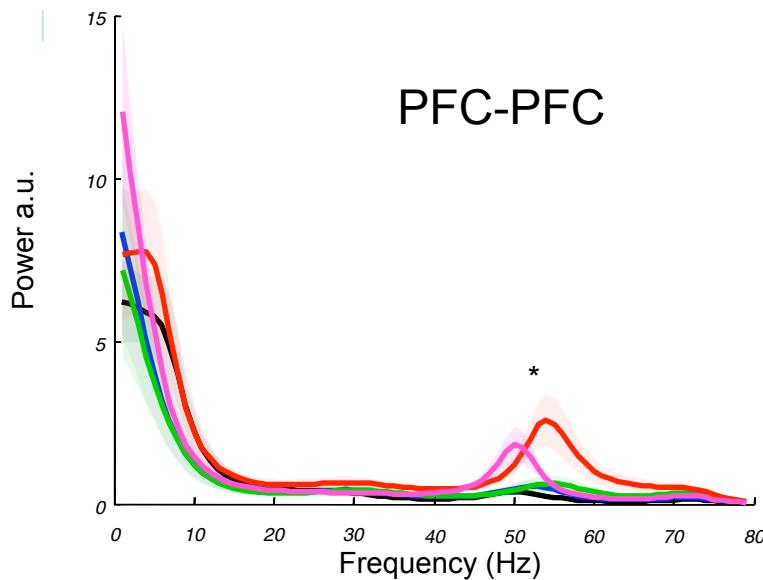
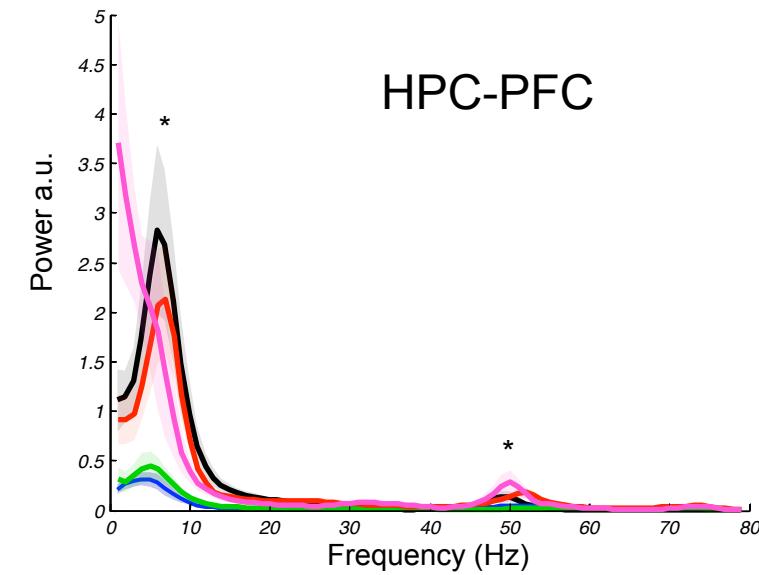
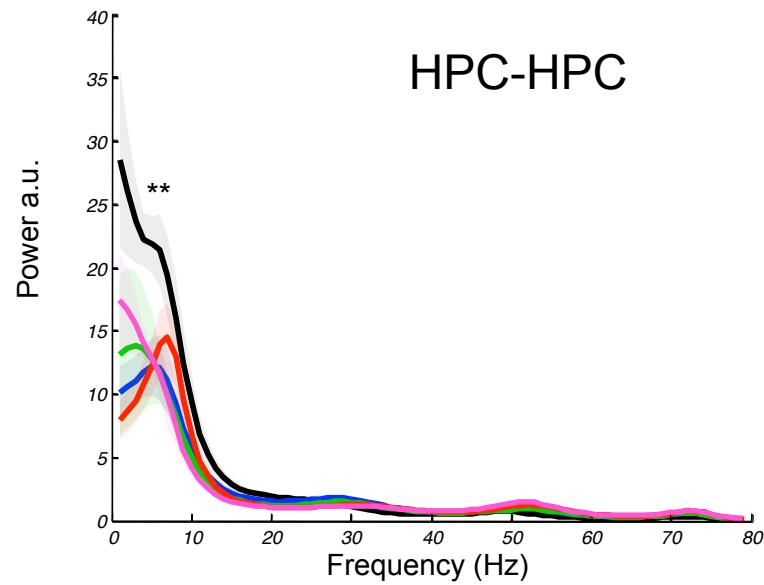
# Ketamine data Set



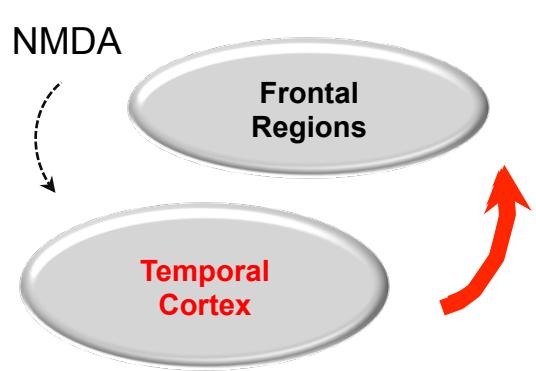
Hyper-locomotion



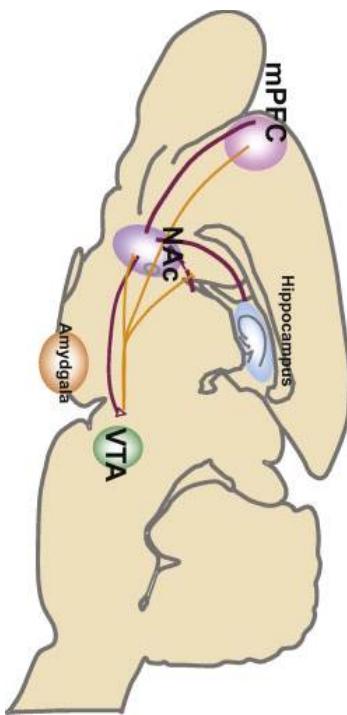
# Oscillatory Characteristics



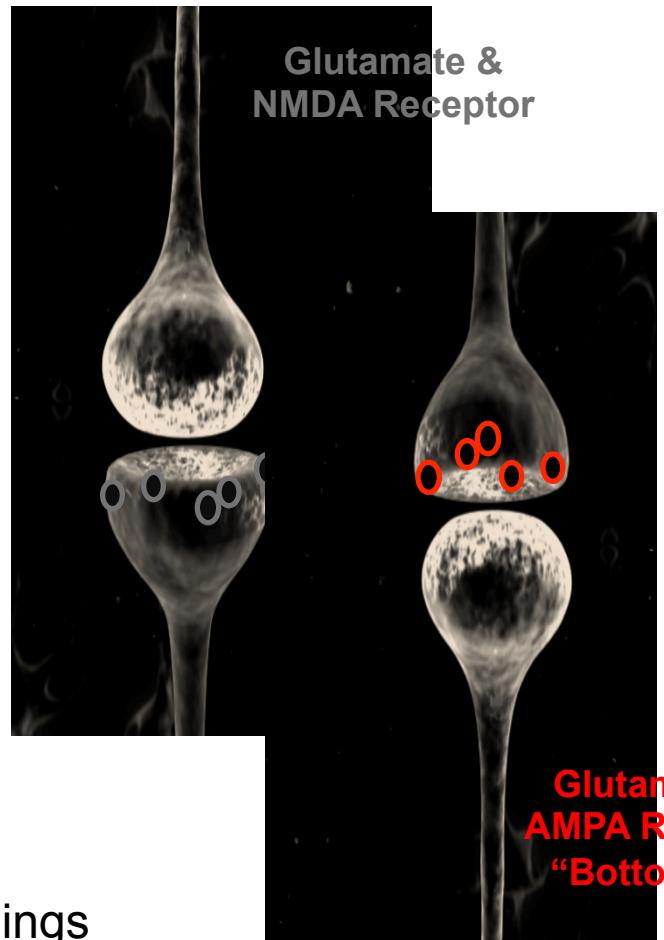
# Hypothesis, Data & Model-based analysis:



Dopamine  
Gamma  
Gaba



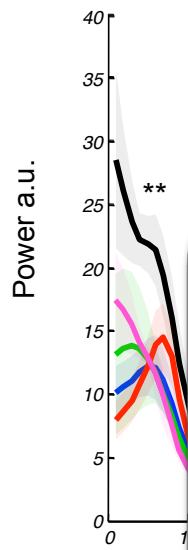
Hippocampal & Prefrontal Recordings



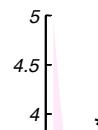
5 mins of recordings from freely moving rat: tetrodes in dCA1 & mPFC

Ketamine Dose: 0, 2, 4, 8, 30 mgkg<sup>-1</sup>

# Hypothesis, Data & Model-based analysis:



HPC-HPC



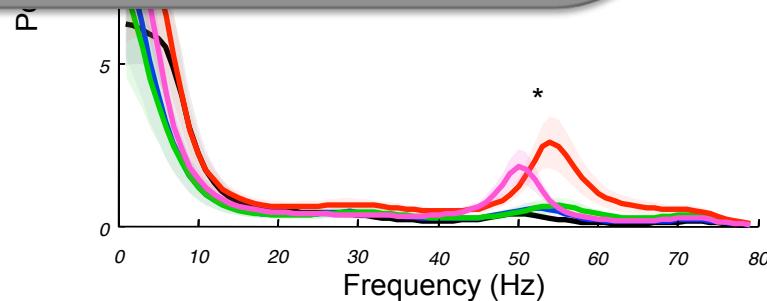
HPC-PFC

What Cortico-Limbic Connectivity Changes are responsible for theta and gamma changes under ketamine?

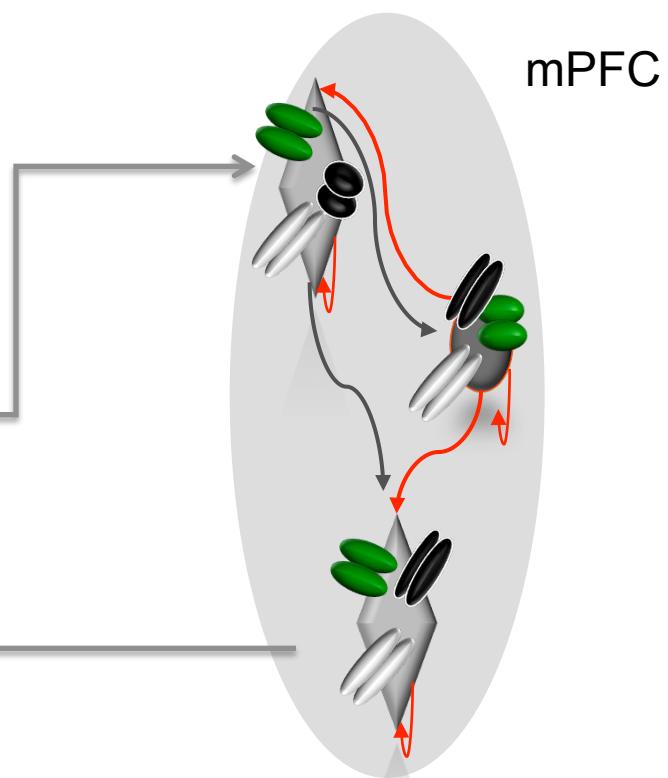
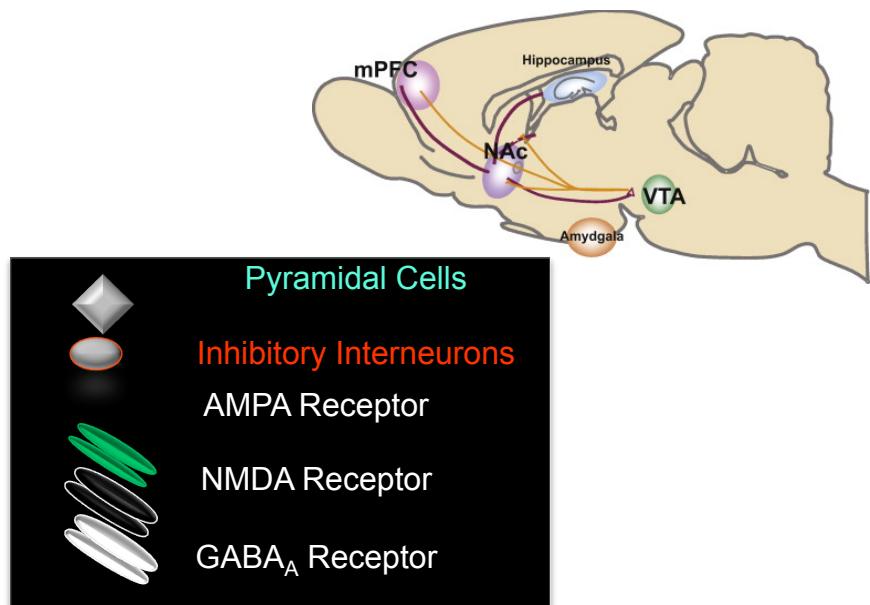
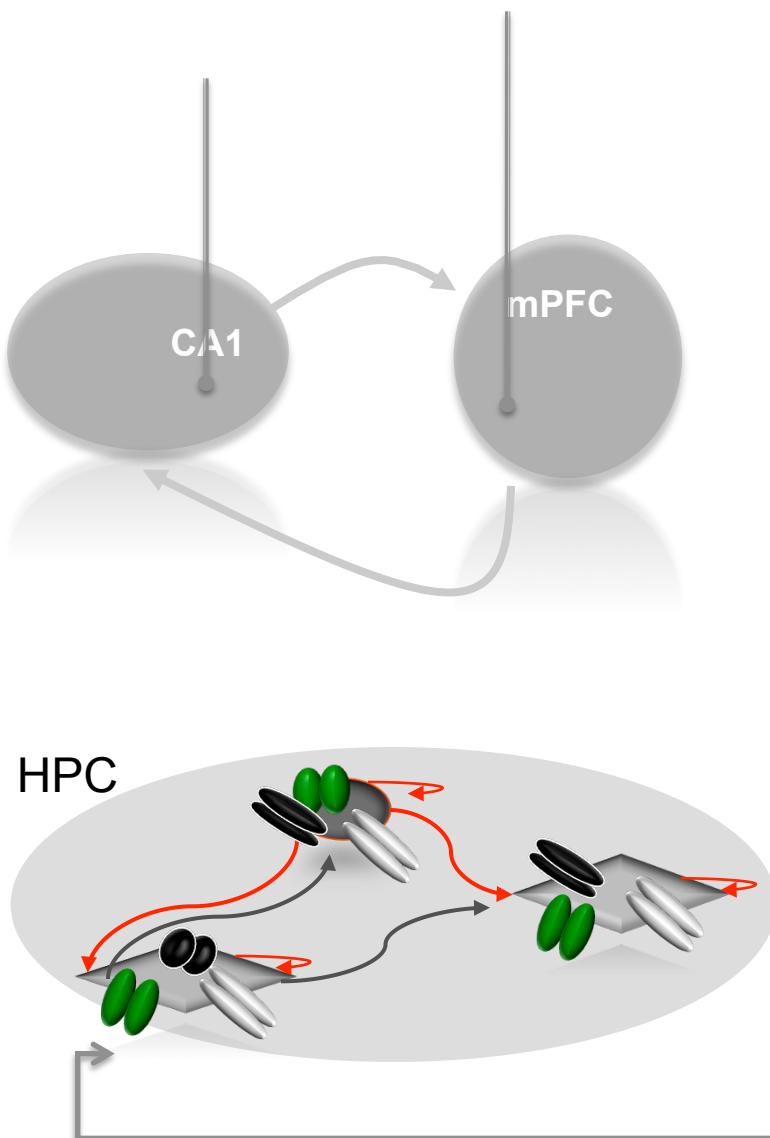
What Intrinsic Connectivity Changes are responsible for theta and gamma changes?

What Receptors are involved at these extrinsic & intrinsic synapses?

5 minutes : freely moving



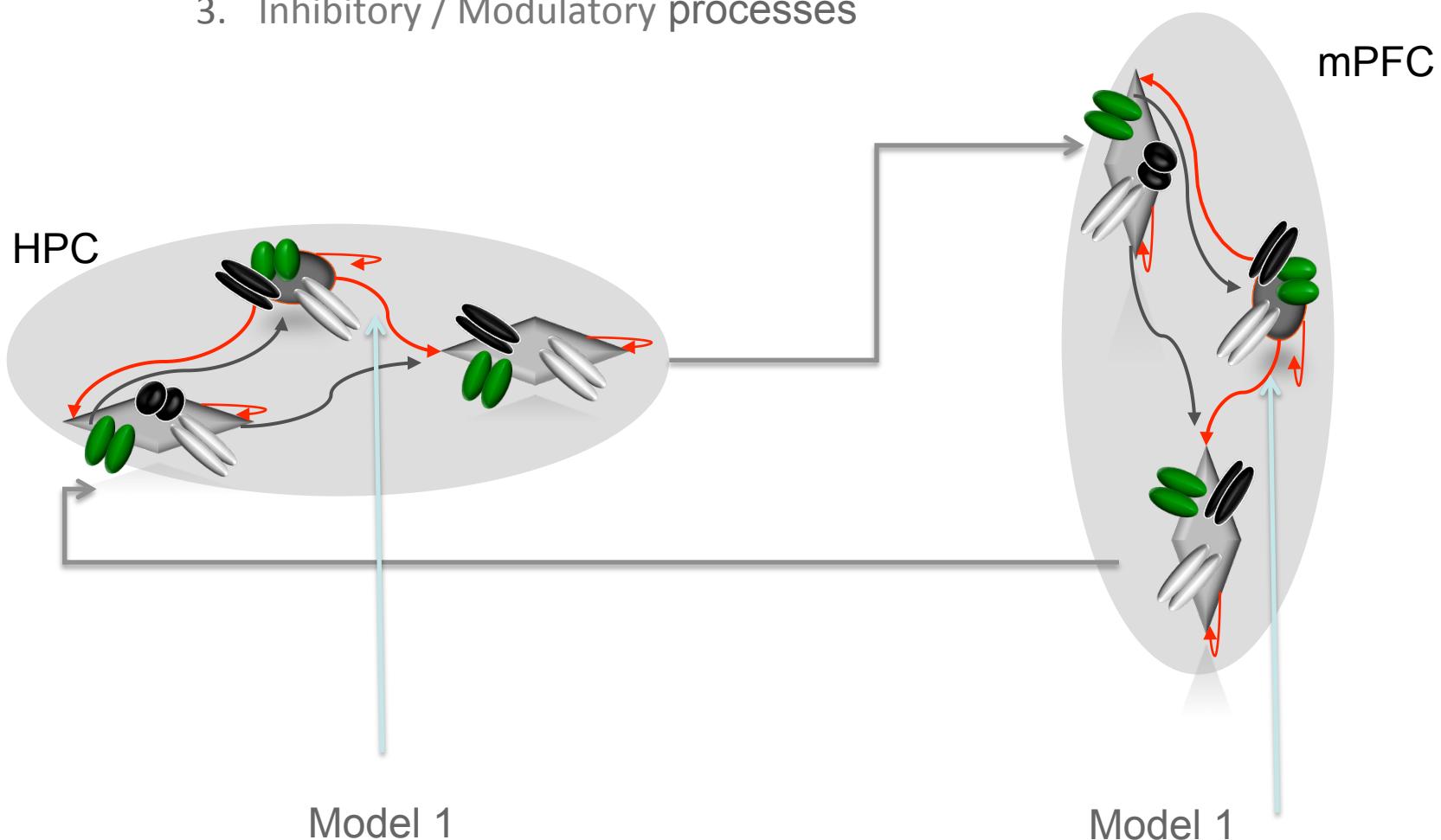
# Proposed Architecture



# Model Comparison

Ketamine modulates:

1. All extrinsic connections,
2. Intrinsic NMDA and
3. Inhibitory / Modulatory processes

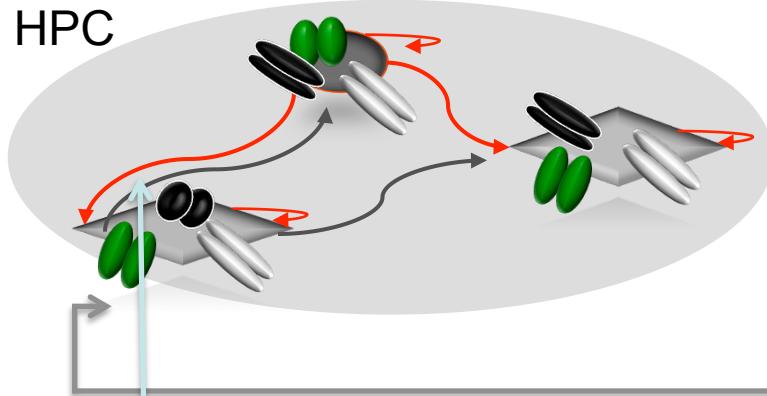


# Model Comparison

Ketamine modulates:

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2. Intrinsic NMDA and
3. Inhibitory / Modulatory processes

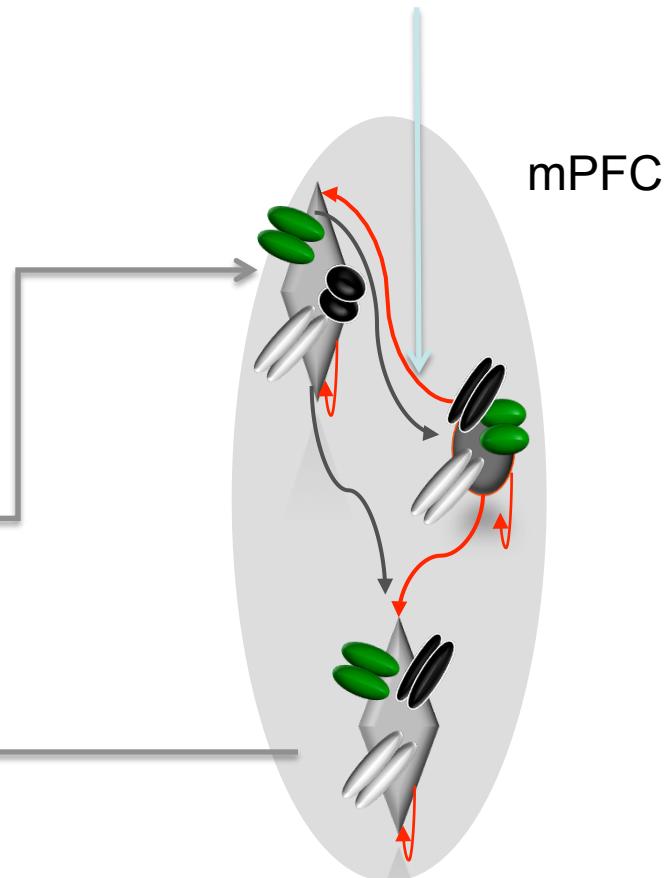
HPC



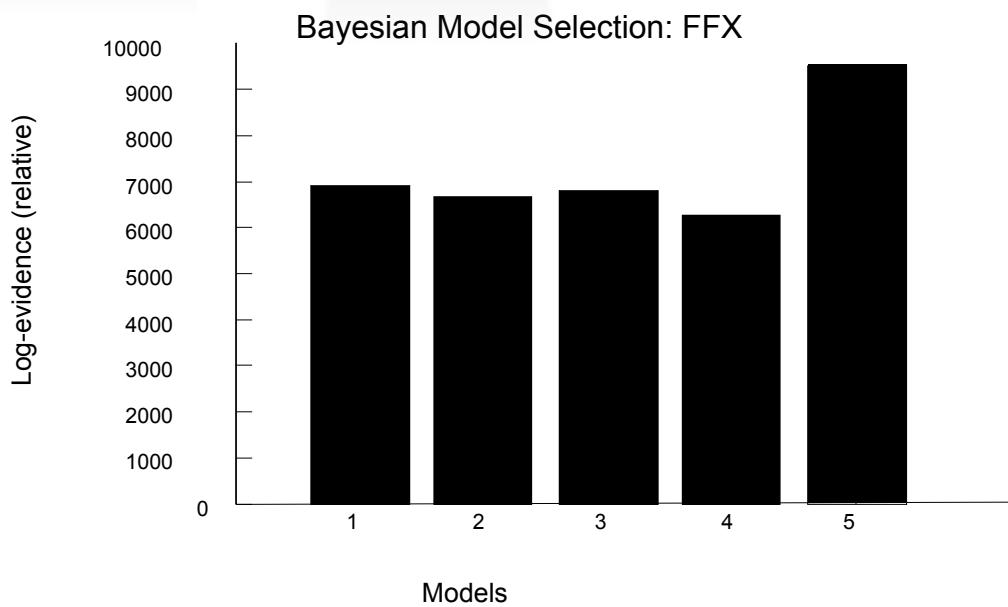
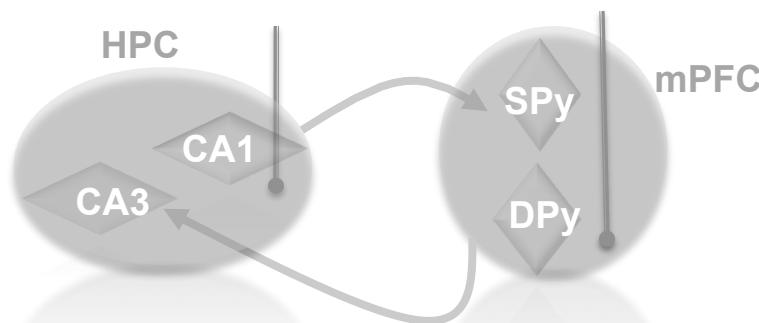
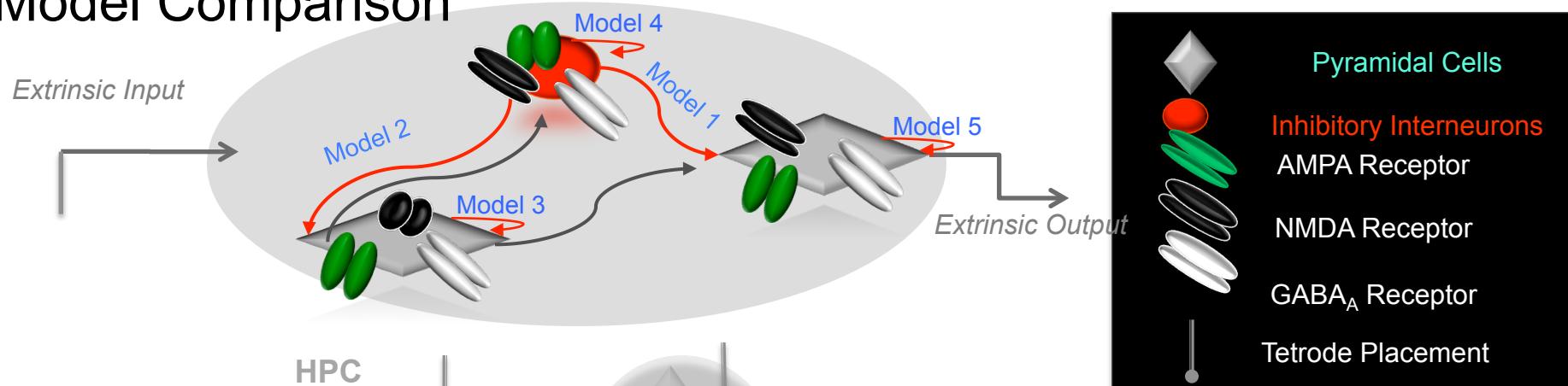
Model 2

Model 2

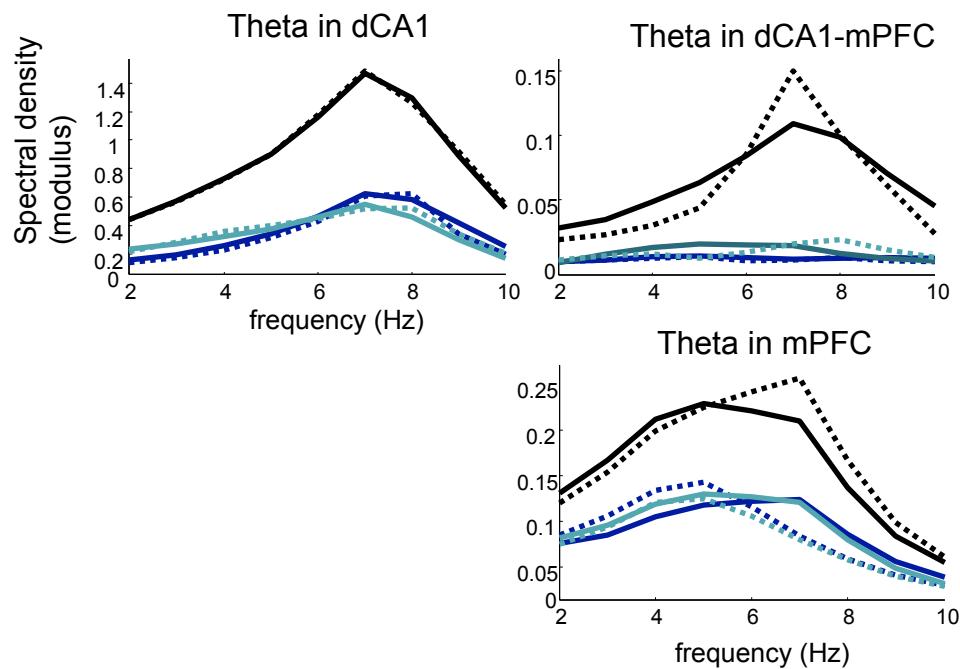
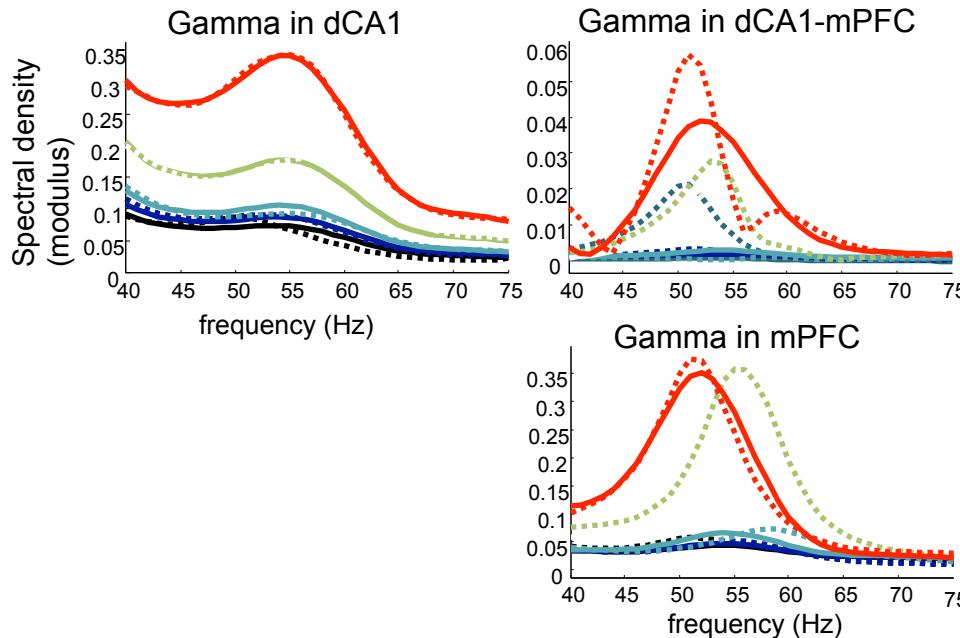
mPFC



# Model Comparison

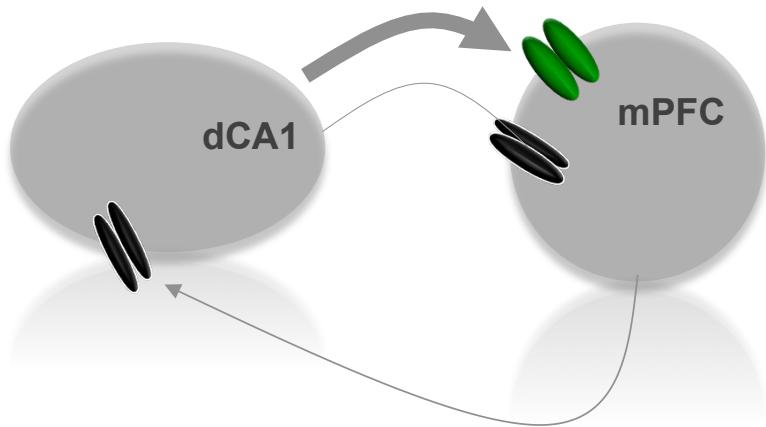


# Model Fits



- predicted: saline vehicle
- observed: saline vehicle
- predicted: 2 mgkg<sup>-1</sup>
- observed: 2 mgkg<sup>-1</sup>
- predicted: 4 mgkg<sup>-1</sup>
- observed: 4 mgkg<sup>-1</sup>
- observed: 8 mgkg<sup>-1</sup>
- observed: 8 mgkg<sup>-1</sup>
- predicted: 30 mgkg<sup>-1</sup>
- observed: 30 mgkg<sup>-1</sup>

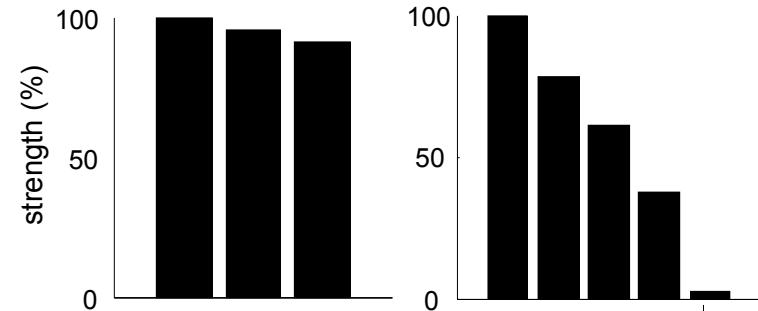
# Extrinsic Connectivity Changes under Ketamine



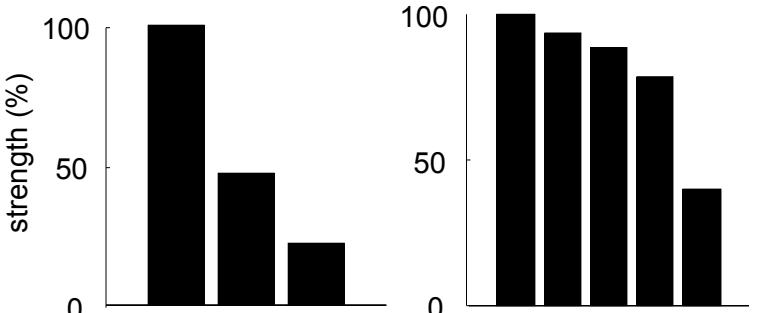
Theta Model

Gamma Model

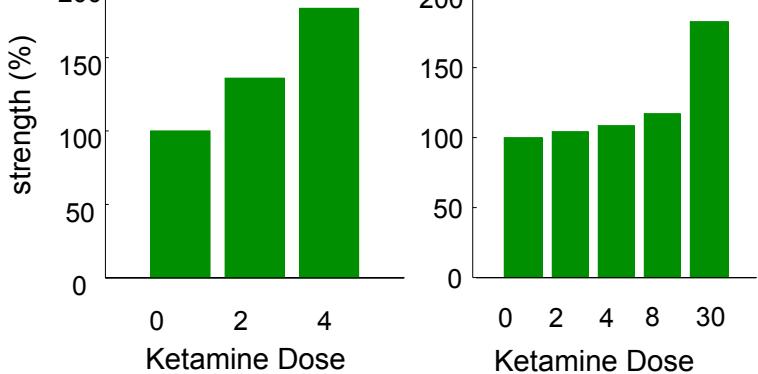
NMDA-mediated input to HPC from mPFC



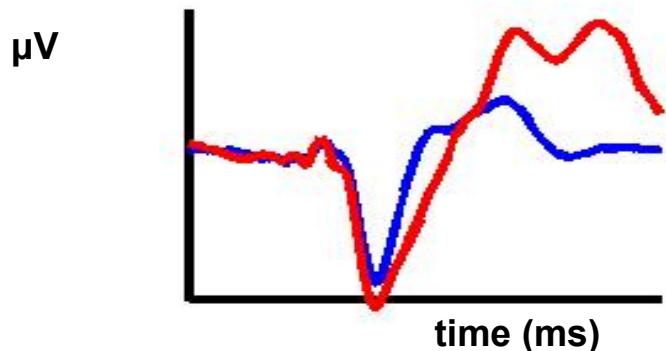
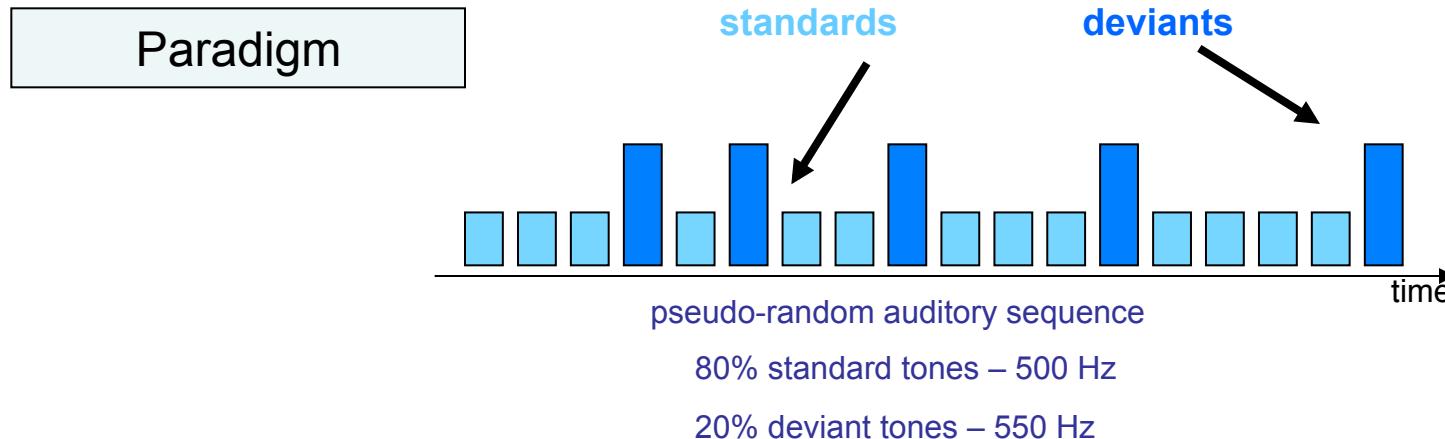
NMDA-mediated input to PFC from dCA1



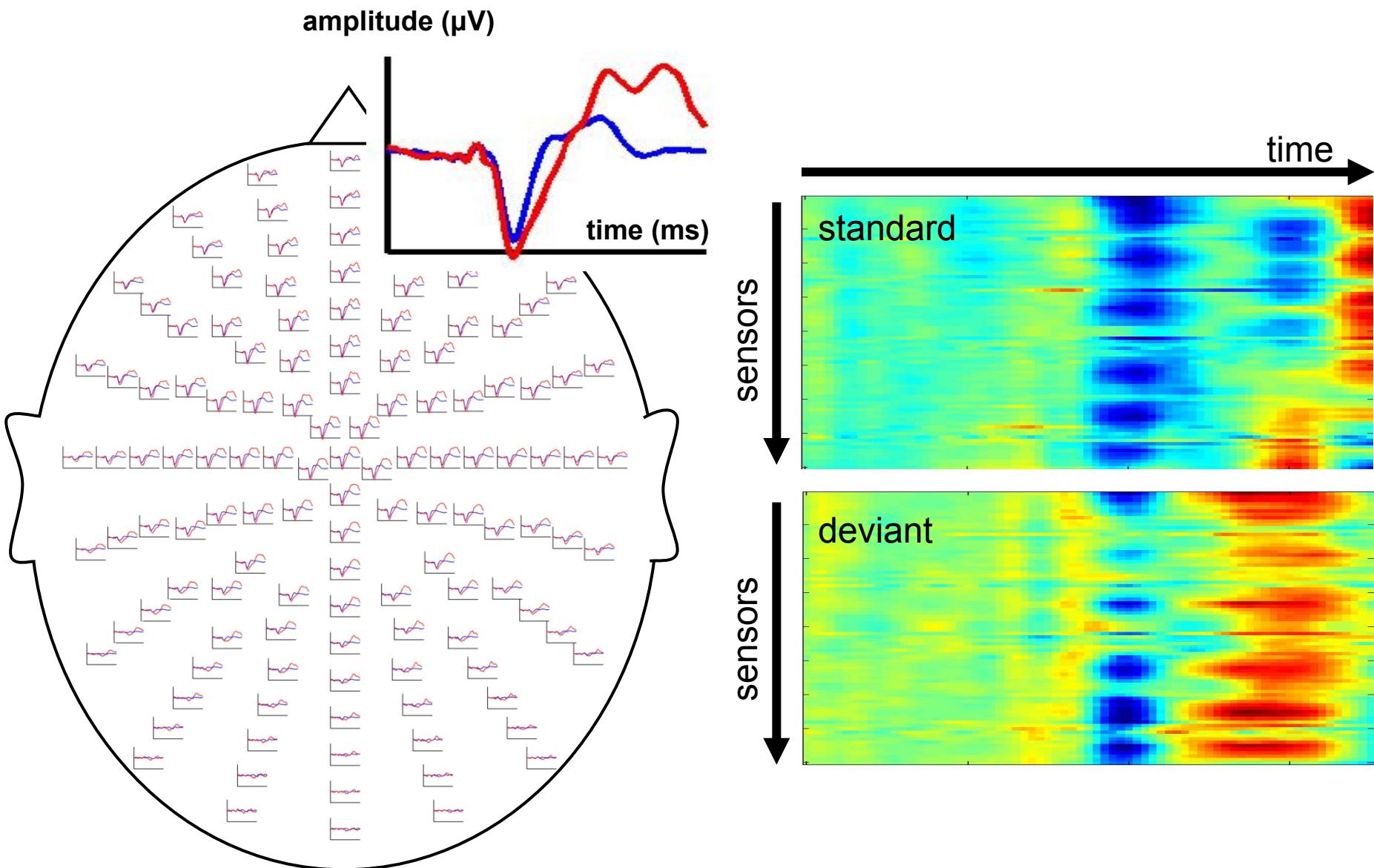
AMPA-mediated input to PFC from dCA1



# Mismatch negativity (MMN) Paradigm

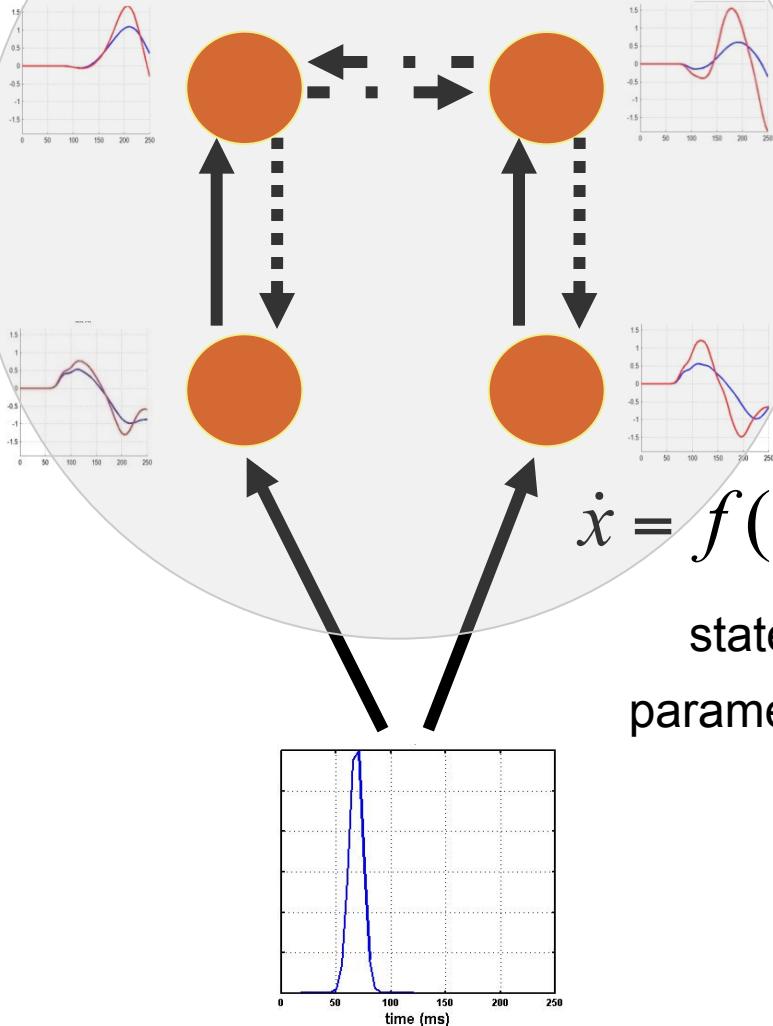


# Scalp Data

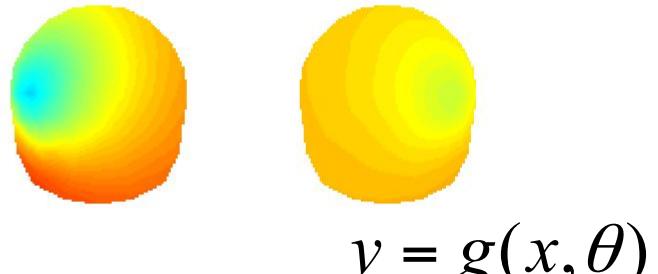


# DCM

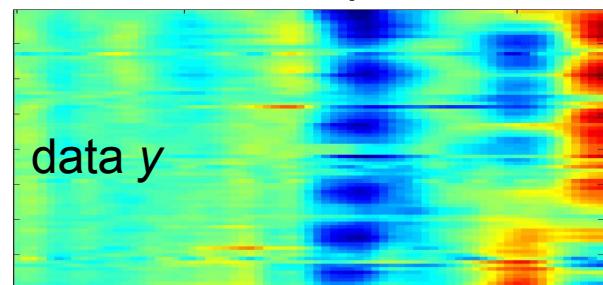
Source dynamics  $f$



Spatial forward model  $g$

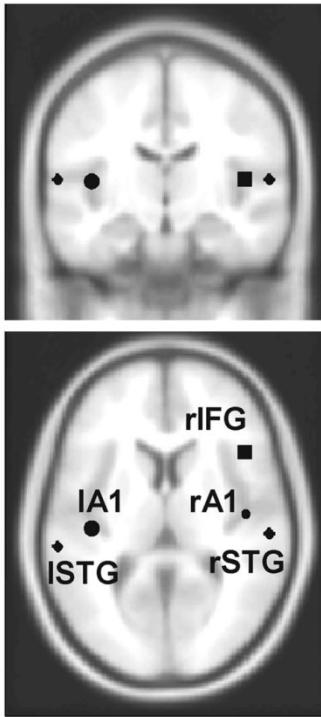


Evoked response



Input  $u$

# Sources



## MNI Coordinates from literature

**IA1** (-42 -22 7)

**rA1** (46 -14 8)

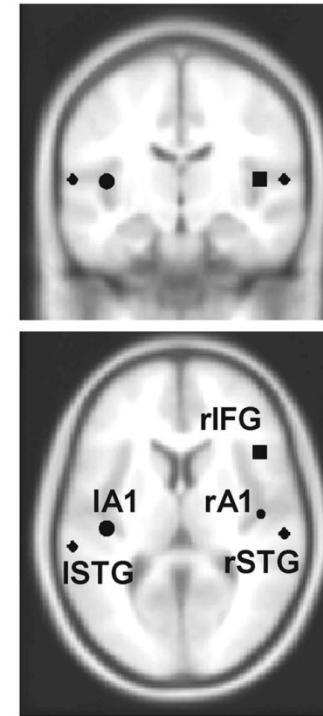
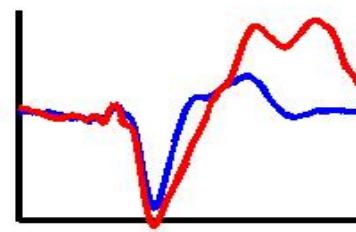
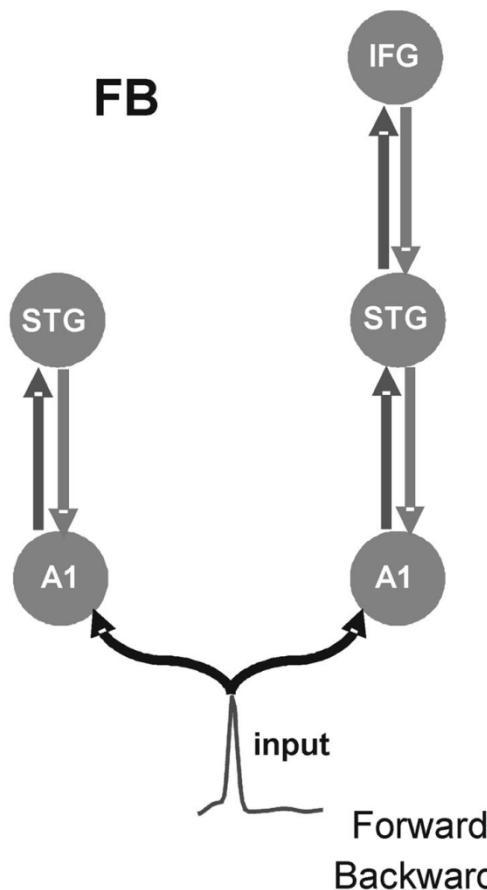
**ISTG** (-61 -32 8)

**rSTG** (59 -25 8)

**rIFG** (46 20 8)

# Model

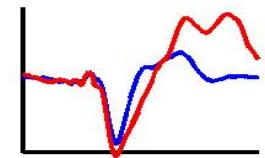
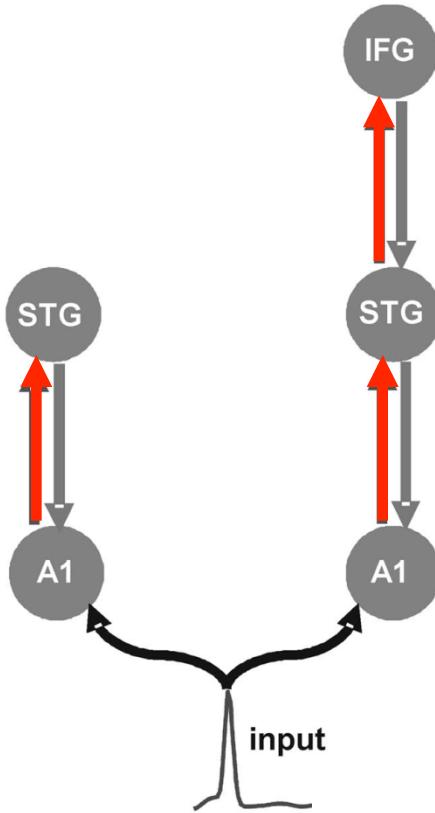
with backward connections



# Competing Hypotheses I

Do differences in standard and deviant evoked responses rely on forward connections

## Model 1



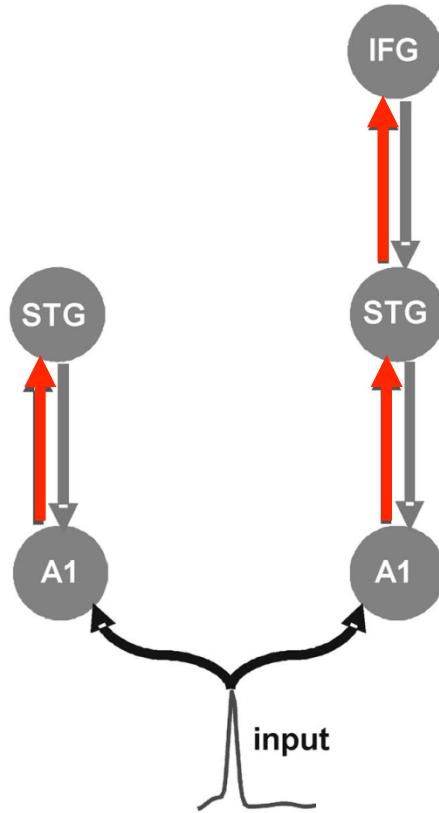
Connections = Baseline + Trial Specific

Garrido et al., PNAS, 2008

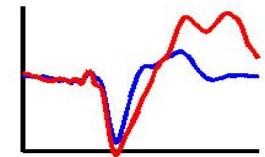
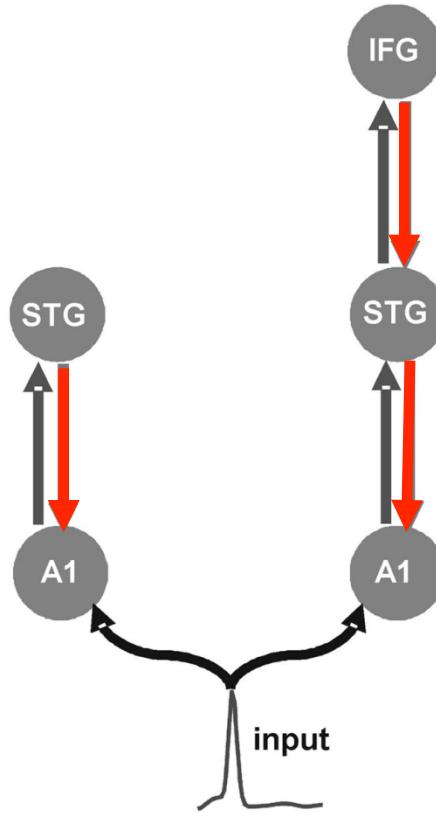
# Competing Hypotheses I

Do differences in standard and deviant evoked responses rely on forward connections or backward connections ?

Model 1



Model 2



Connections = Baseline + Trial Specific

Garrido et al., PNAS, 2008