Next generation neural mass models

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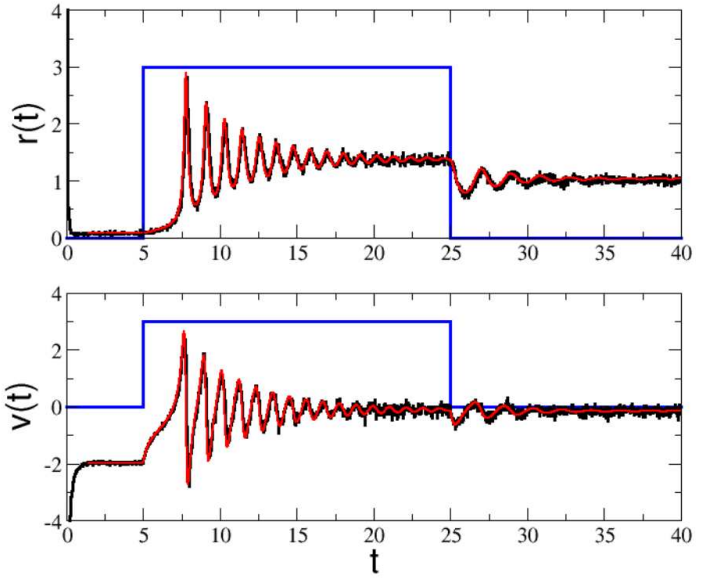
Plan of the Talk



- The Quadratic Integrate and Fire (QIF) Neuron
- Derivation of the Neural Mass Model (MPR) (Montbrió, Pazó, Roxin, PRX, 2015)
- Applications of the Neural Mass Model to Interacting Populations (2020)
  - Cross frequency coupling
  - Theta-nested gamma oscillations
  - Synaptic-based working memory
- Extension of the neural mass to fluctuation driven population dynamics (2021)
  - Neural Networks with Background Noise
  - Neural Networks with Random Sparse Synaptic Couplings

Quadratic Integrate and Fire Neuron

This a very simple model, which can mimick realistic neural dynamics

$$\frac{dV}{dt} = V^2 + \eta$$

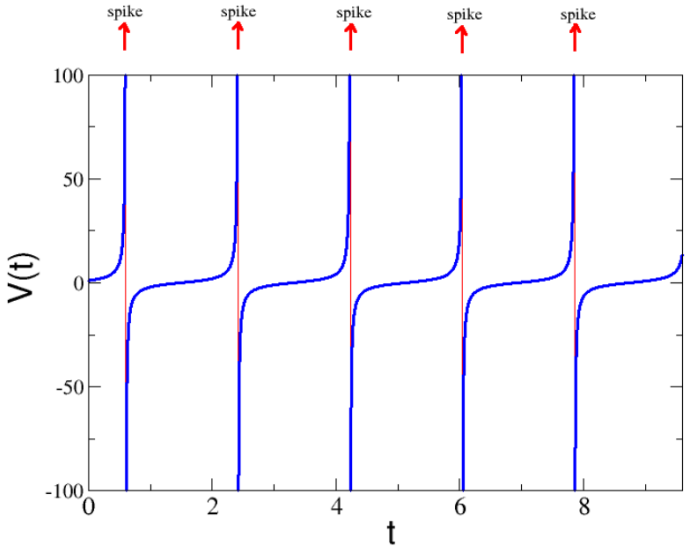
- V membrane potential with threshold V_{th} and reset V_r
- η neural excitabilty
  - $\eta > 0$ Tonic Neuron
  - $\eta \leq 0$ Excitable Neuron

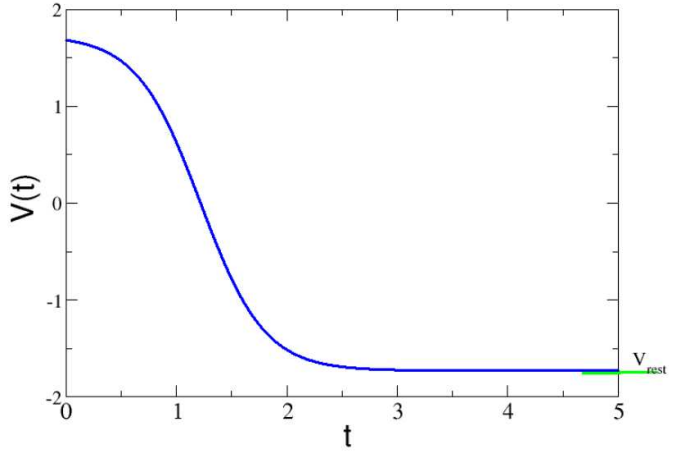
The model has been developed to reproduce parabolic bursting in neurons of the Aplysia abdominal ganglion (when sinusoidally forced) and low firing neurons (Class I).

Ermentrout and Kopell, (1986) SIAM Journal on Applied Mathematics

Latham et al. (2000) Journal of Neurophysiology

Quadratic Integrate and Fire Neuron





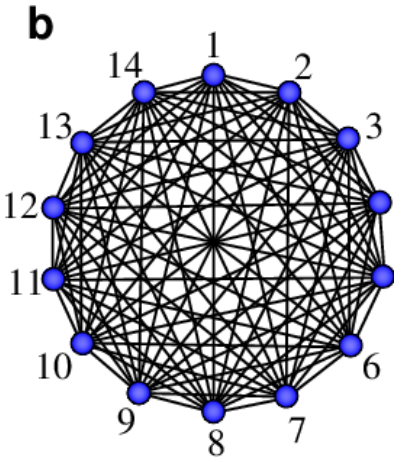
Tonic neuron $\eta > 0$

- The firing period is $T_{firing} = \frac{\pi}{\sqrt{\eta}}$

Excitable neuron $\eta \leq 0$

- If $V(t_0) \leq \sqrt{|\eta|}$ - Subthreshold Dynamics $V \to V_{rest} = -\sqrt{|\eta|}$
- If $V(t_0) > \sqrt{|\eta|}$ emission of a spike followed by relaxation to V_{rest}

Population of QIF neurons



The evolution of the membrane potentials of N globally coupled heterogeneous neurons can be written as

$$\frac{dV_j}{dt} = V_j^2 + \eta_j + I_e(t) + Jr(t)$$

where

- η_j is the excitability of neuron j
- $I_e(t)$ is some external current
- J is the synaptic coupling – $J > 0$ ($J < 0$) excitatory (inhibitory) neurons
-

$$r(t) = \frac{1}{N} \sum_j \sum_{k(j)} \delta(t - t_{k(j)}) = \frac{1}{N} \sum_j r_j(t)$$

is the average firing rate due to all the post-synaptic potentials (δ-spikes) emitted by all neurons in the network

Neural Mass Model

In the limit $N \to \infty$ we can describe the population of N neurons in term of a probability density function (PDF) $\rho(V|\eta, t)$ of their membrane potentials

Continuous formulation

$\rho(V|\eta, t)dV$ = fraction of neurons with membrane potentials between V and $V + dV$, with excitability η, at time t

The excitabilities $\eta_j \Rightarrow$ are continuous random variable distributed according to a PDF $g(\eta)$

Since the number of neurons should be conserved in time, the PDF ρ satisfies the following

Continuity equation $$\partial_t \rho + \partial_V [(V^2 + \eta + Jr + I_e)\rho] = 0$$

The stationary solution ρ_0 (for constant I_e) is given by

$$\rho_0(V|\eta) \propto \frac{1}{V^2 + \eta + Jr + I_e}$$

this is a Lorentzian (or Cauchy) Distribution $L(x) = \frac{\Delta}{\pi(x^2+\Delta^2)}$

Neural Mass Model

Lorentzian Ansatz

MPR assumed that also for non-stationary case the distribution is Lorentzian at any time

$$\rho(V|\eta,t) = \frac{1}{\pi}\frac{x(\eta,t)}{[V-y(\eta,t)]^2 + x(\eta,t)^2}$$

where the median $y(\eta,t)$ and the HWHM $x(\eta,t)$ correspond to the

Mean Membrane Potential

$$v(\eta,t) = y(\eta,t)$$

for the neurons with excitability η

Firing Rate

$$r(\eta,t) = \frac{x(\eta,t)}{\pi}$$

for the neurons with excitability η

Neural Mass Model

Therefore within the Lorentzian Ansatz the continuity equation can be rewritten as follows for the complex variable $w(\eta, t) = \pi r(\eta, t) + iv(\eta, t)$

$$\partial_t w(\eta, t) = i[-w^2(\eta, t) + \eta + Js + I]$$

This is a neural mass equation describing the dynamics of a population of neurons with the same excitability η in terms of their firing rate $r(\eta, t)$ and their mean membrane potential $v(\eta, t)$.

However for a heterogeneous population one has still an extremely large number of equations corresponding to all the possible values of the excitability η_i

How can we reduce the system to a low dimensional one ?

Neural Mass Model

Systems with random heterogeneities have been treated exactly in statistical mechanics by assuming a Lorentzian distribution for the heterogeneities E. Yakubovich, Soviet Physics JETP. 1969

$$g(\eta) = \frac{1}{\pi} \frac{\Delta}{(\eta - \bar{\eta})^2 + \Delta^2}$$

This allows to estimate exactly the average mean membrane potential and firing rate

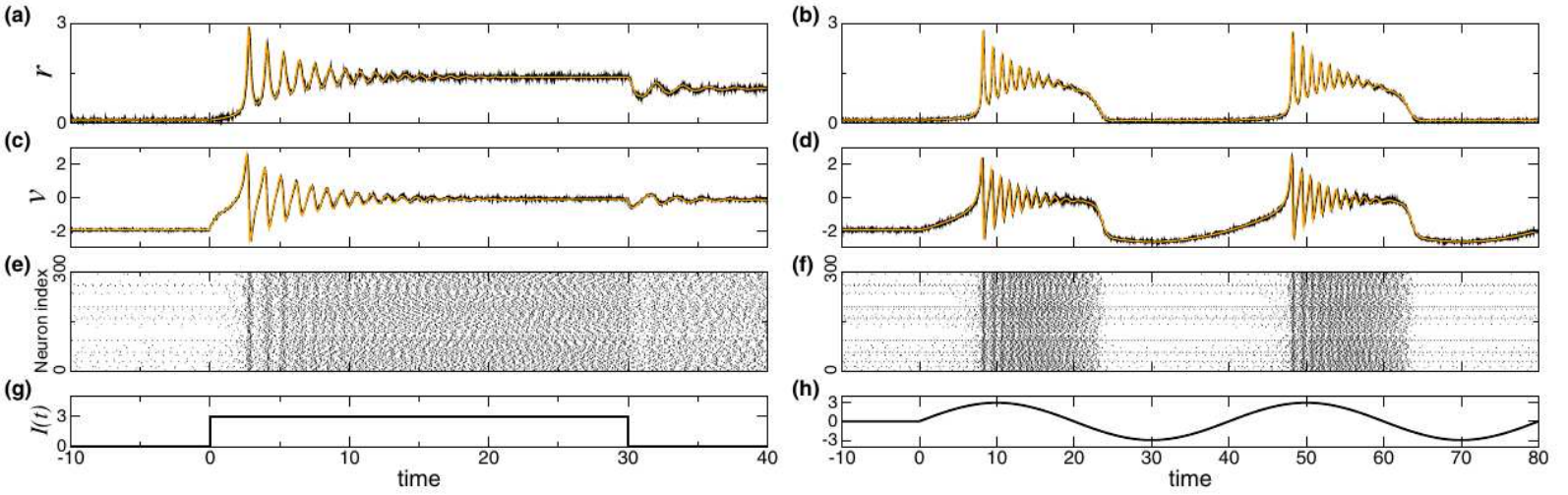
$$v(t) = \int_{-\infty}^{+\infty} v(\eta, t) g(\eta) d\eta \qquad r(t) = \int_{-\infty}^{+\infty} v(\eta, t) g(\eta) d\eta$$

2-dimensional Neural Mass Model

$$\dot{r} = \frac{\Delta}{\pi} + 2rv \qquad \dot{v} = v^2 + \bar{\eta} + Jr + I(t) - \pi^2 r^2$$

[Montbrió, Pazó, Roxin, Phys. Rev. X (2015)]

MPR Model



2-dimensional Neural Mass Model

$$\dot{r} = \frac{\Delta}{\pi} + 2rv \qquad \dot{v} = v^2 + \bar{\eta} + Jr + I(t) - \pi^2 r^2$$

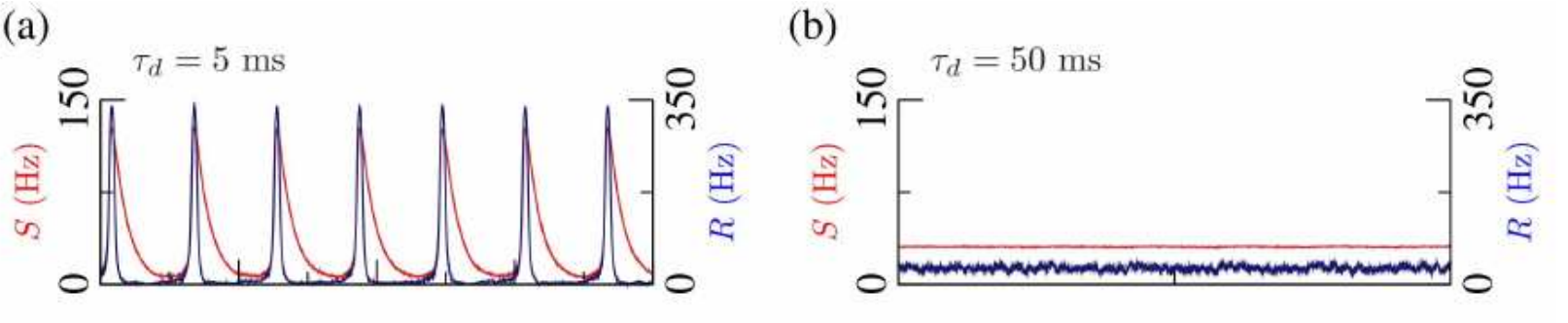
This Neural Mass Model describes exactly the dynamics of the Network done of N QIF neurons

[Montbrió, Pazó, Roxin, Phys. Rev. X (2015)]

γ Oscillations in Inhibitory Networks

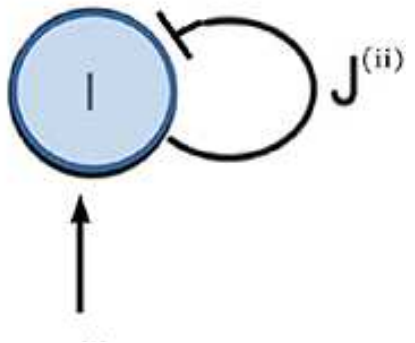
Fast γ oscillations emerge in recurrently coupled inhibitory networks

- An external excitatory drive tends to synchronize the inter-neuronst that fire together
- The firing of the inhibitory neurons leads to their silencing on a timescale dictated by the duration τ_d of the post-synaptic potentials S
- This mechanism generates collective oscillations in the network
  [Whittington, Traub, Jefferys, Nature. 1995]



If $\tau_d >> \tau_m$, the synaptic time scale is longer that that the membrane time scale no oscillations, the recurrent input appears as an average inhibitory current
[DeValle, Roxin, Montbrió, PLOS Comp Biol (2017)]

γ Oscillations in Inhibitory Networks

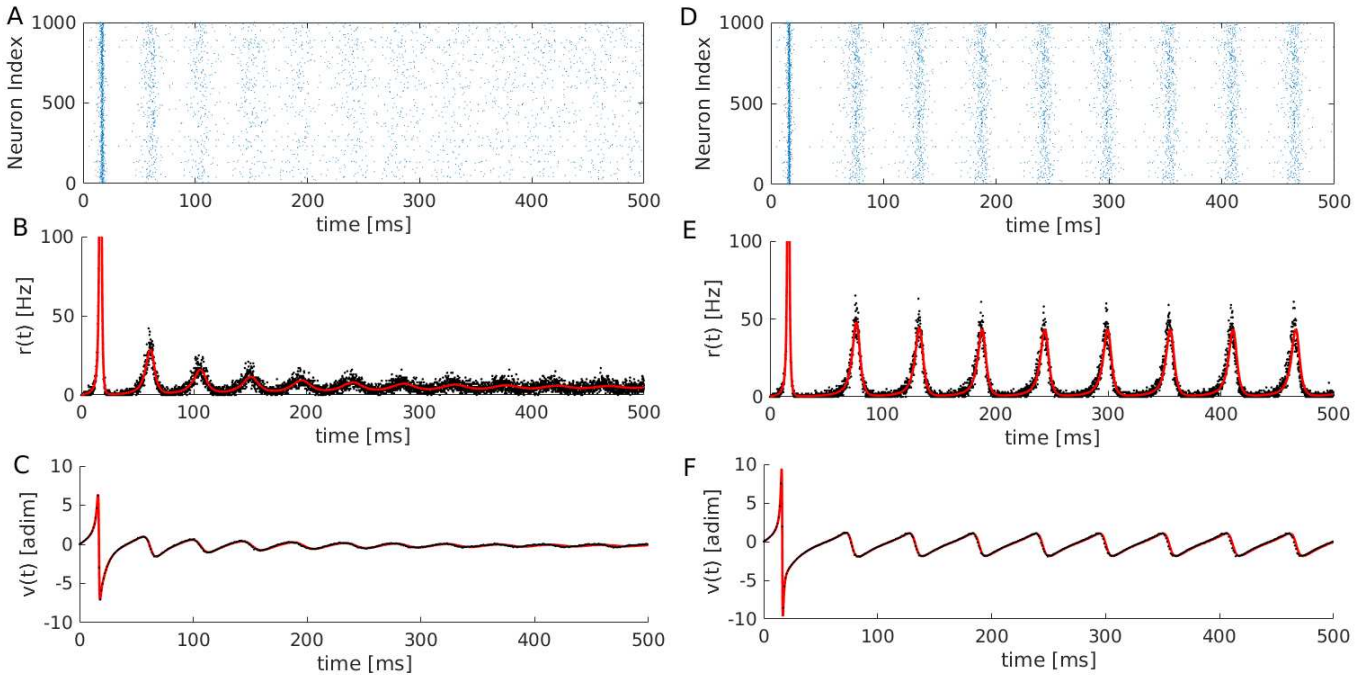


$$\tau_m \dot{r} = \frac{\Delta}{\tau_m \pi} + 2rv$$

$$\tau_m \dot{v} = v^2 + \bar{\eta} - \tau_m J^{(ii)} S + I^{(i)} - (\pi \tau_m r)^2$$

$$\tau_d \dot{S} = -S + r$$

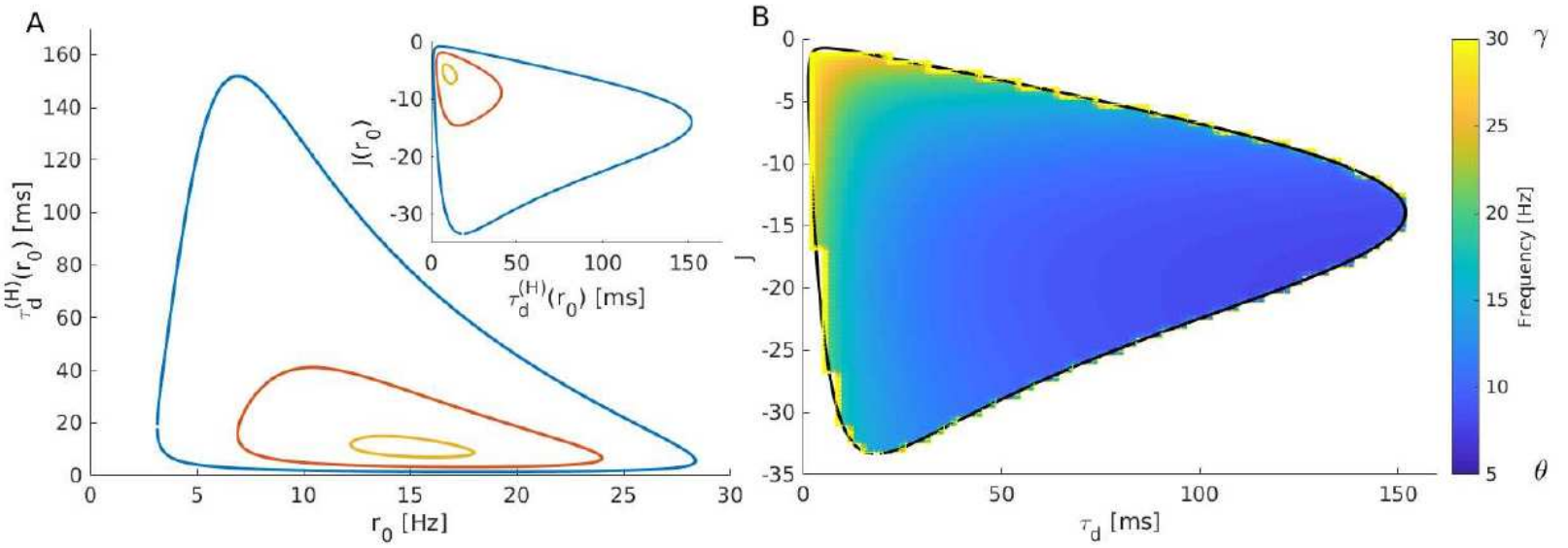
super-critical Hopf-Bifurcation to COs



Ceni, Olmi, AT, Angulo Garcia, Chaos (2020)

γ Oscillations in Inhibitory Networks

- Oscillations are sustained within a frequency range ($\nu \simeq 5 - 30Hz$) thanks to
  - finite synaptic time τ_d
  - self-inhibitory action of neurons



- For increasing heterogeneity ($\Delta = 0.01$, $\Delta = 0.05$, $\Delta = 0.1$, $\Delta = 0.14$) the observation of collective oscillations requires finer tuning of the parameters
- Frequency decreases for increasing τ_d and J

γ Oscillations in Inhibitory Networks

The heuristic firing rate models (e.g. Wilson-Cowan model)

$$\begin{aligned} \tau_m \dot{r} &= -r + F(-\tau_m J^{(ii)} S + I^{(i)}) \\ \tau_d \dot{S} &= -S + r \end{aligned}$$

do not display Collective Oscillations without introducing an effective delay which takes somehow in account for the sub-threshold dynamics

- Firing rate models do not encompass Subthreshold Dynamics of the Membrane potential
- The membrane potential dynamics is fundamental to have synchronization effects
- The MPR model reproduces network dynamics in this case, Wilson-Cowan not

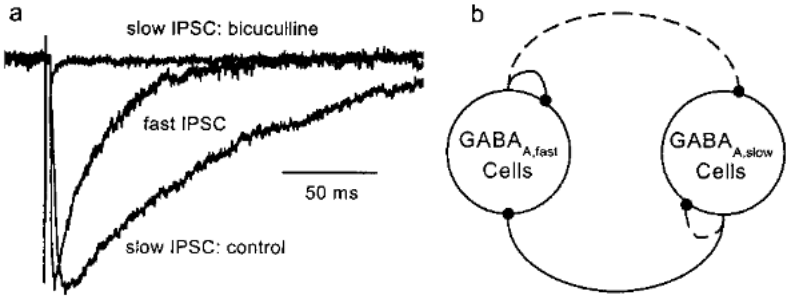
[DeValle, Roxin, Montbrió, PLOS Comp Biol (2017)]

θ-γ Cross Frequency Coupling

One of the most present cross-frequency coupling in the brain is the interaction between slow θ-rhytms (5-10 Hz) and fast γ-oscillations (20-100 Hz)

Which is the origin of this CFC ?

White, Banks, Pearce, and Kopell, PNAS 2000 proposed that θ-γ CFC emerges due to the interaction of two inhibitory populations with different kinetic properties



Two classes of interneurons identified in the Hippocampus (CA1)

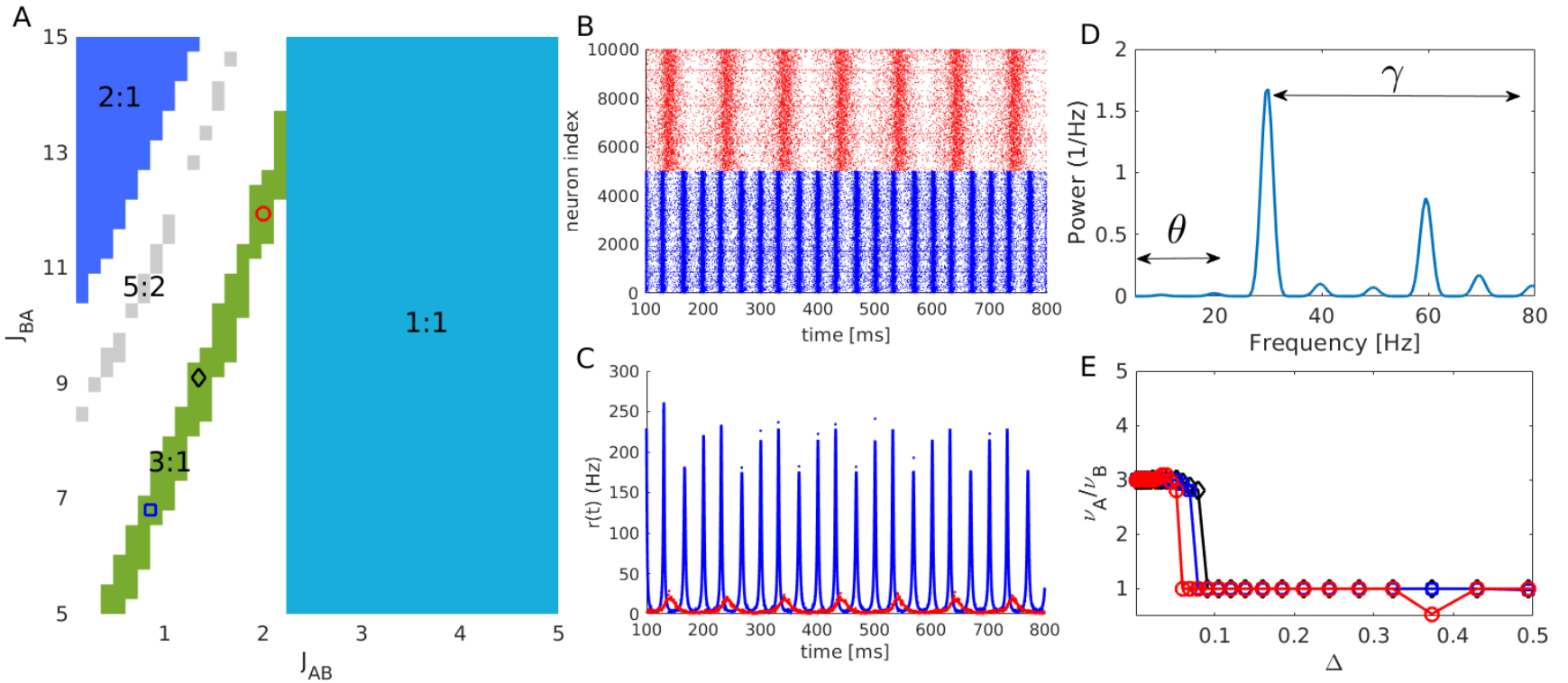
- GABA$_{A,fast}$ $\tau_d \simeq 9$ ms
- GABA$_{A,slow}$ $\tau_d \simeq 50$ ms

May we observe this phenomenon for two coupled neural masses A and B with exponential synapses with different kinetics ?

- A slow inhibitory population A with self-coupling $J_{AA} = -2$,
- A fast inhibitory population B with self-coupling $J_{BB} = -11$
- Cross-couplings J_{AB} (A $\rightarrow$ B) and J_{BA} ($B \rightarrow A$)

θ-γ Cross Frequency Coupling

- Fast population: $\tau_{A,d} = 9$ ms - Slow population: $\tau_{B,d} = 50$ ms
- $3:1$ phase-locking mode: $\theta - \gamma$ coupling

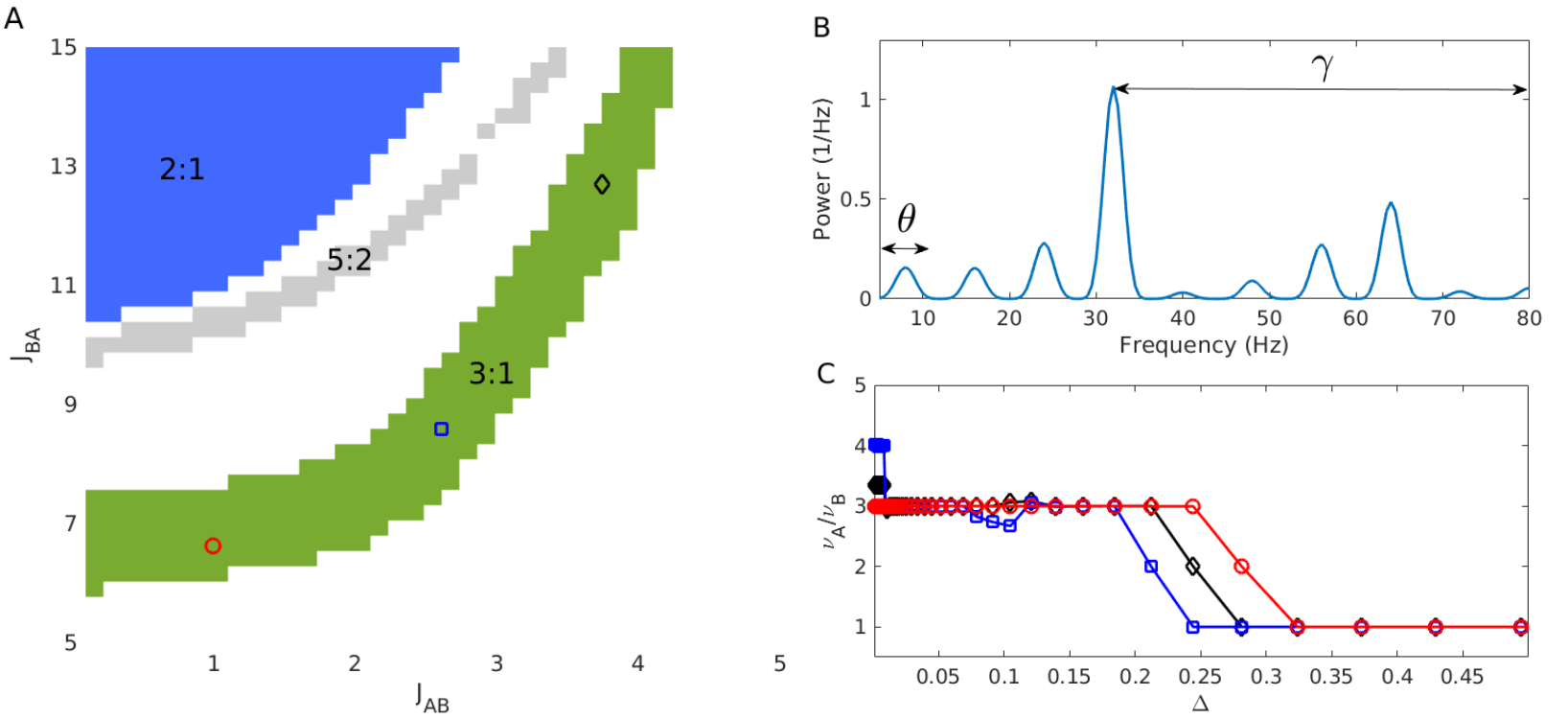


$\Rightarrow$ For large values of J_{AB} and disorder Δ, the $3:1$ locked mode is lost

[Ceni, Olmi, AT, Angulo Garcia, Chaos (2020)]

θ-γ Cross Frequency Coupling

- External θ-forcing on the slow population $\theta \simeq 10$ Hz
- Fast population: $\tau_{A,d} = 9$ ms - Slow population: $\tau_{B,d} = 50$ ms



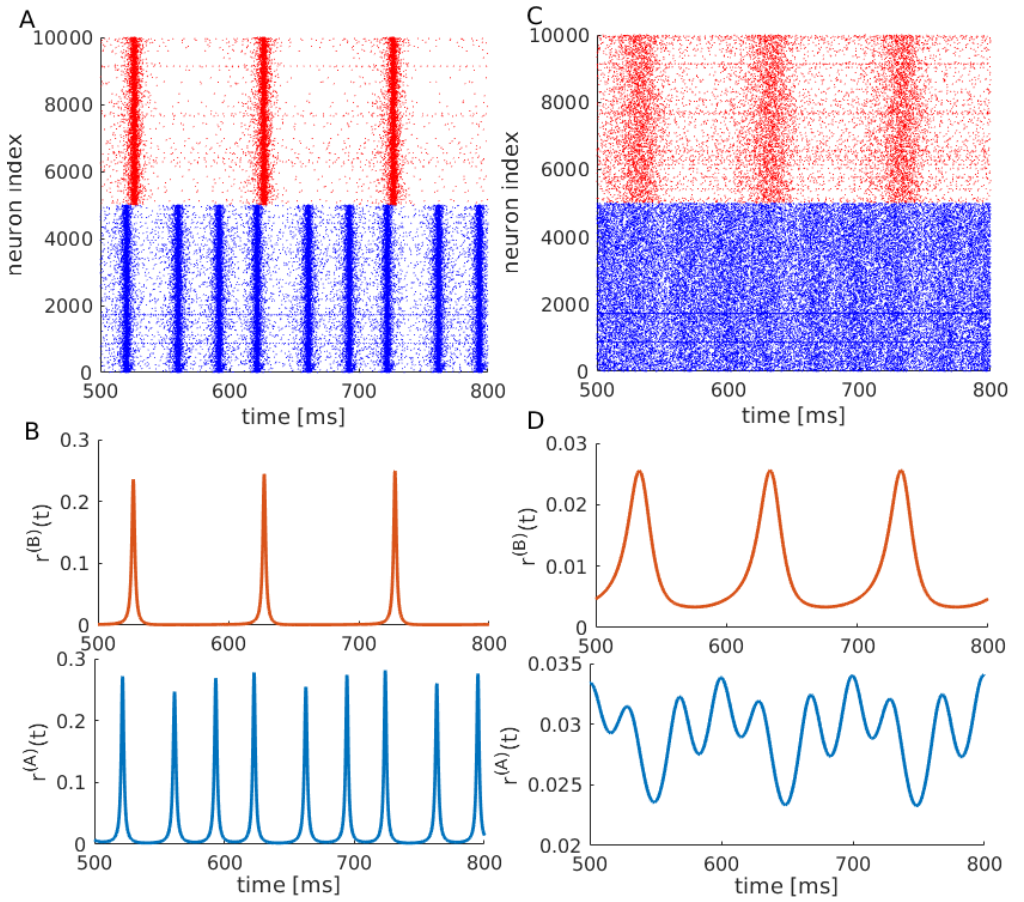
$\Rightarrow$ Power in the θ band is increased

$\Rightarrow$ Adding the (slow) modulation to the slow population increases the amount of disorder (Δ) that can sustain the $\theta - \gamma$ coupling

[Ceni, Olmi, AT, Angulo Garcia, Chaos (2020)]

θ-γ Cross Frequency Coupling

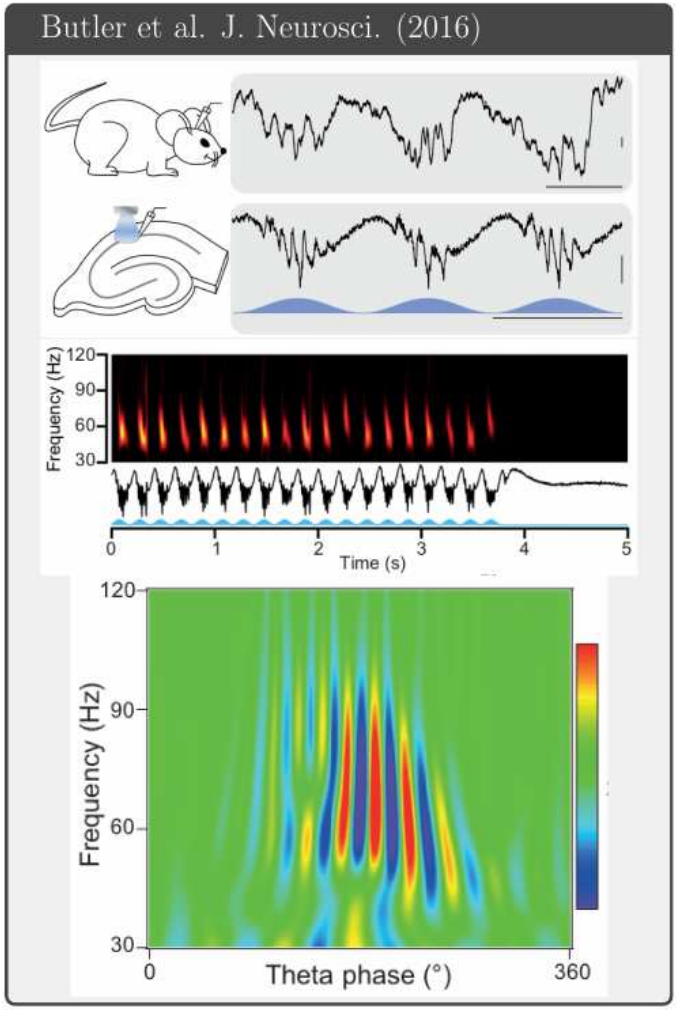
$$I_B = I_0 \sin(2\pi 10 t)$$



- $\Delta = 0.05$
  - Phase-phase CFC: collective oscillations of the slow population an of the fast one are locked in phase
- $\Delta = 0.2$
  - Phase-amplitude CFC: the slow population modulates the amplitude of the oscillations of the fast one

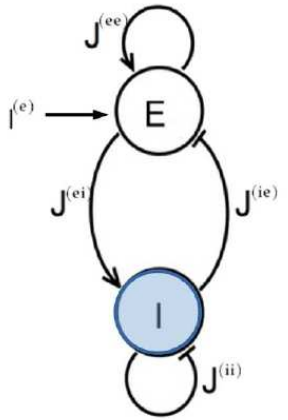
θ Nested γ Oscillations

Several optogenetic experiments performed in different areas of the hippocampus and entorhinal cortex suggest that a θ frequency drive is sufficient to induce in vitro θ-γ CFCs



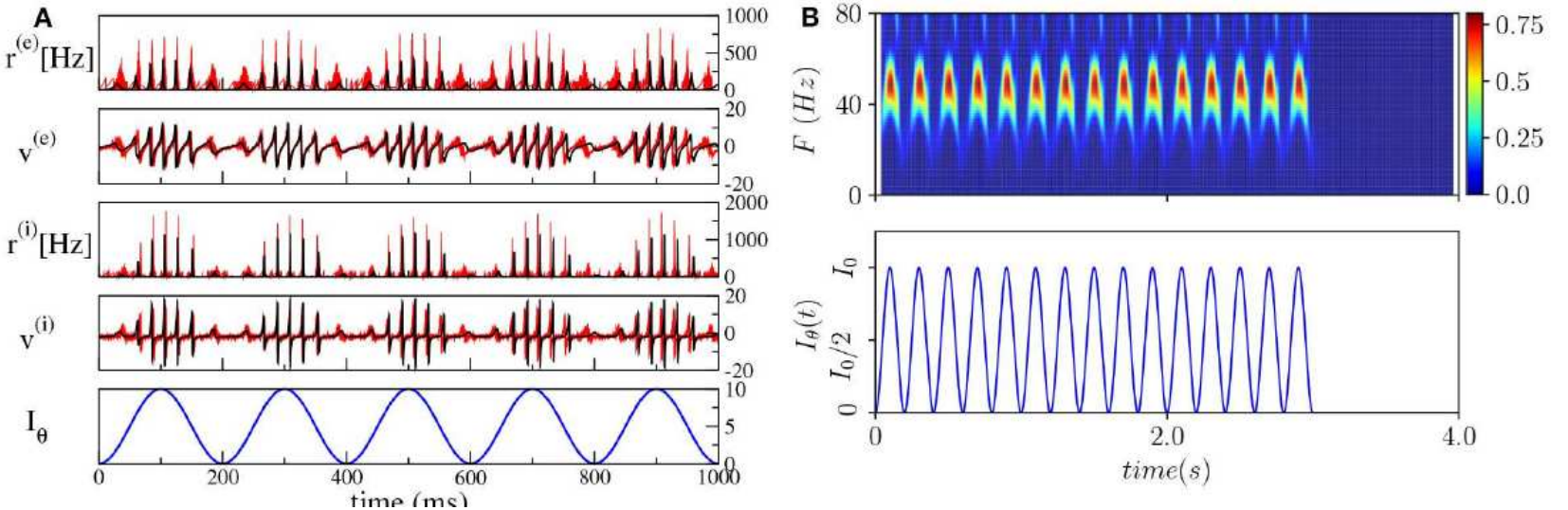
- Butler et al. (2016) stimulated CA1 pyramidal neurons with an excitatory drive $I_\theta = I_0 \sin(\nu_\theta t)$ with $\nu_\theta \in [1:10]$ Hz in vitro
- They show that this is sufficient to generate intrinsic CA1 γ oscillations in vitro similar properties to in vivo CA1 γ oscillations.
- They suggest that the mechanism for the generation of the oscillations is of the pyramidal - interneuron gamma (PING) type

θ Nested γ Oscillations



Excitatory-Inhibitory Neural Masses

- We consider two coupled populations : an excitatory and an inhibitory plus an external periodic forcing on the excitatory population
- We observe θ nested γ oscillations as in the experiments

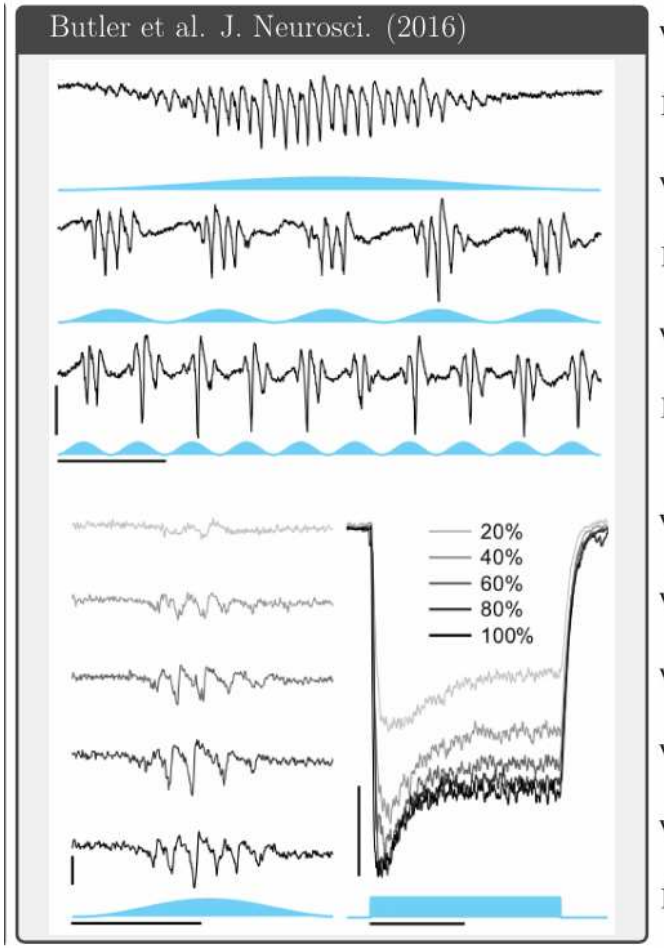


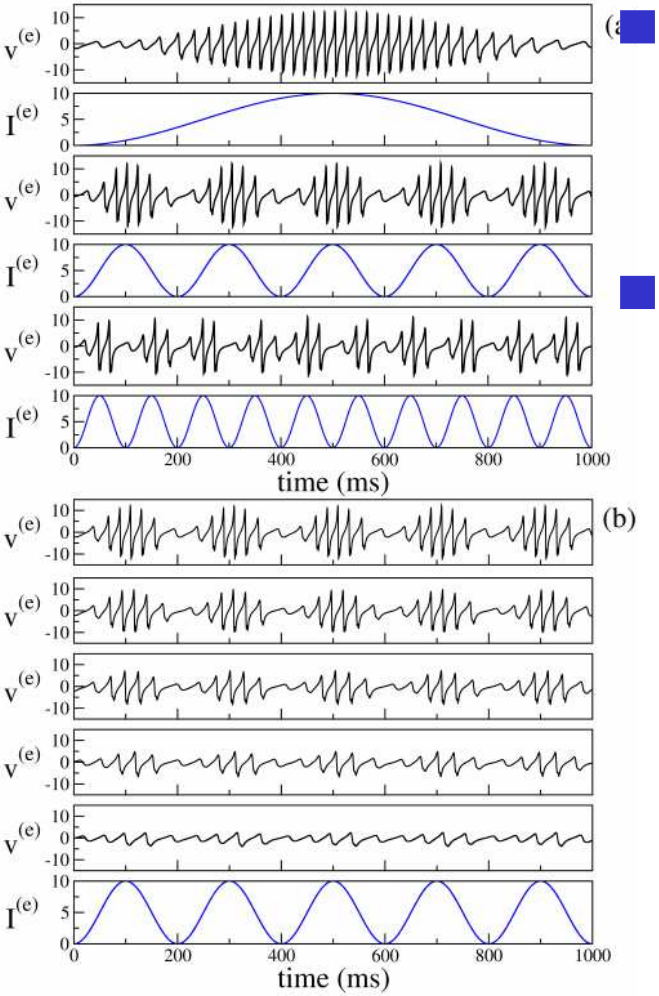
[Segneri, Bi, Olmi, AT Front. Comp. Neuroscience (2020)]

θ Nested γ Oscillations

Comparisons with the experiments

$$I^{(e)}(t) = I_0 \sin(\nu_\theta t)$$

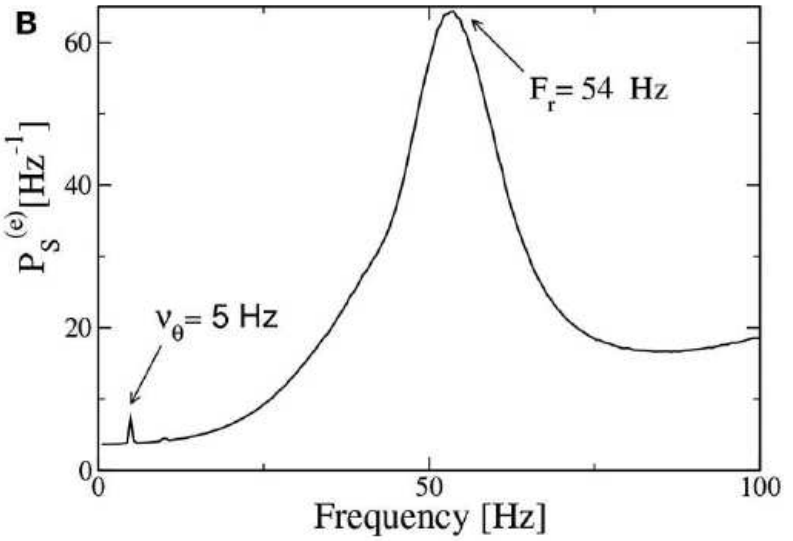




- nested γ oscillation are observable in the whole θ-range $\nu_\theta = [1:10]$ Hz
- For increasing I_0 the amplitude of the γ-oscillations increases

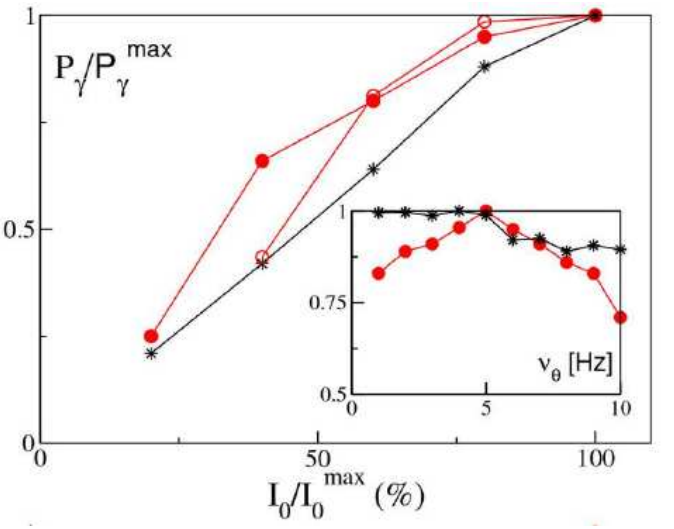
θ Nested γ Oscillations

Power Spectra



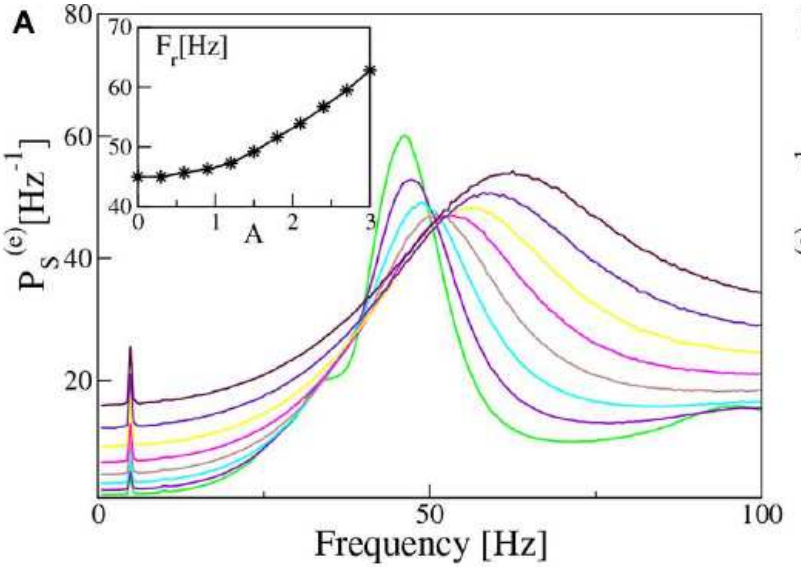
- ν_θ : forcing frequency
- F_r : response frequency
- I_0 : forcing amplitude
- P_γ : power under the main γ-peak

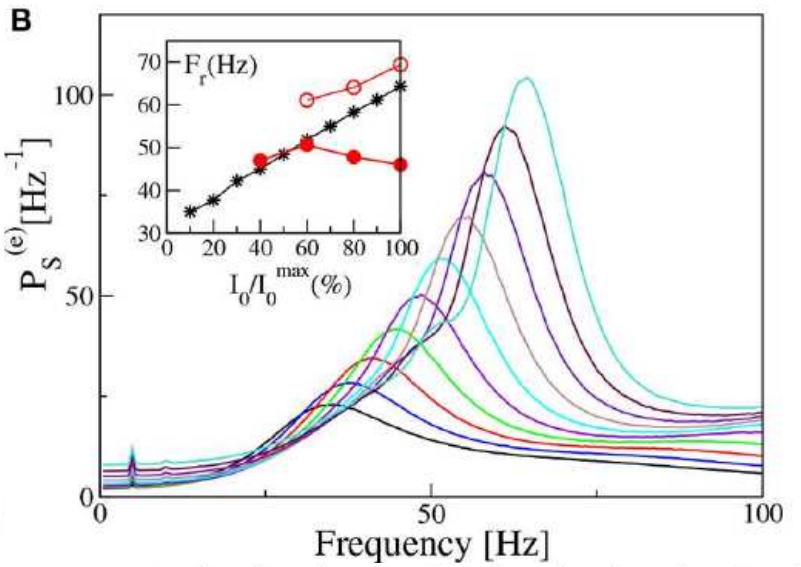
- Black stars : simulations
- Red cicles experiments:
  - Filled: Butler et al., J. Neurosci. (2016)
  - Empty: Butler et al., Eur. J. Neuroscience (2018)



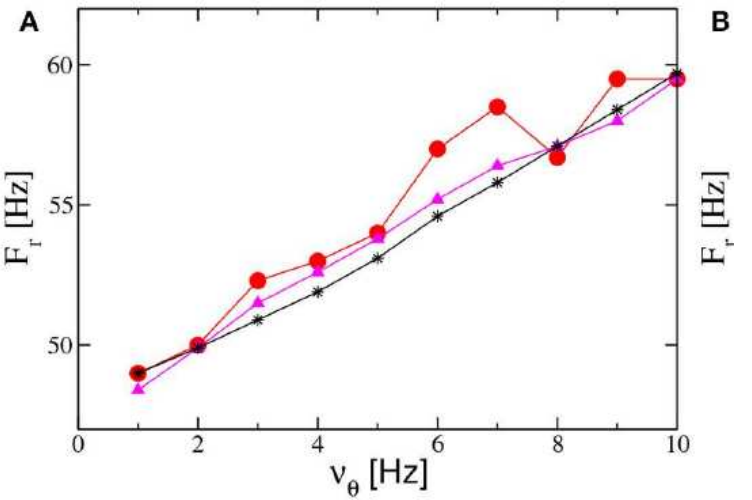
θ Nested γ Oscillations

Power Spectra





- F_r grows with I_0 (as in experiments) and with the noise amplitude
- In the experiments F_r grows with ν_θ : in the simulation this happens only by increasing at the same time the θ-power



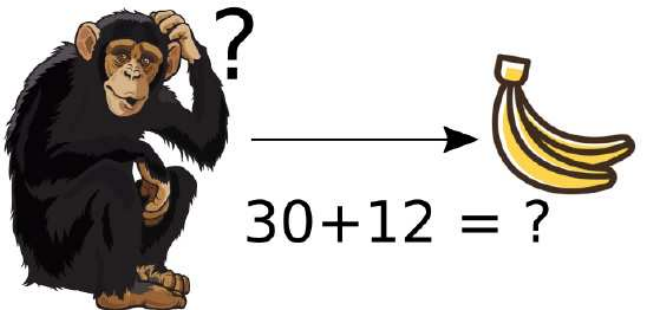
[Segneri, Bi, Olmi, AT Front. Comp. Neuroscience (2020)]

Synaptic-Based Working Memory

What is working memory (WM) ?

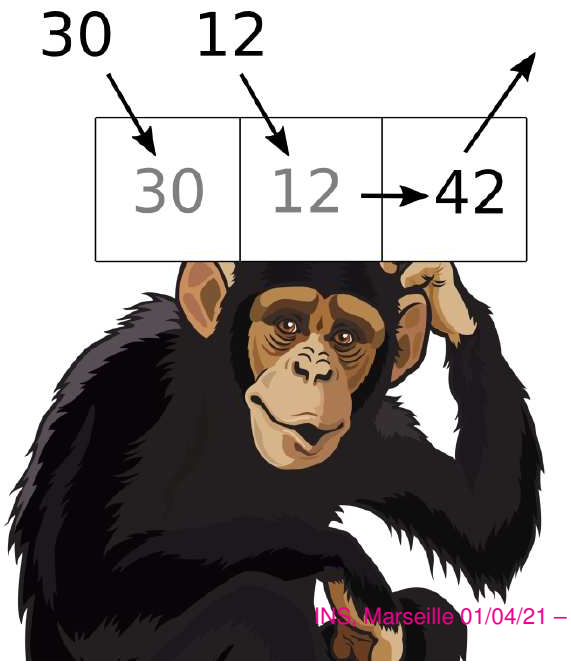
Working memory is a cognitive function fundamental for goal directed behaviours

- solve a task
- achieve the desired goal



Information can be rapidly

- stored & maintained
- processed
- rehearsed

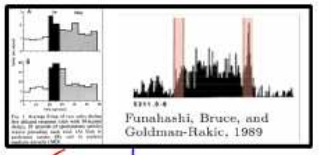


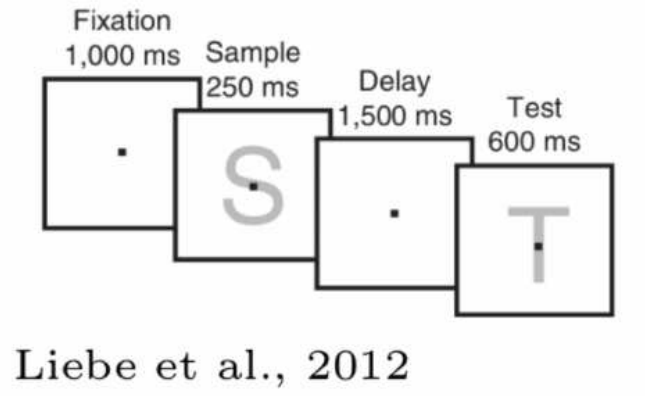
Synaptic-Based Working Memory

How to measure WM related activities ?

Train a subject (animal or human) to solve a task requiring short-term storage of information

- Perform electrophysiological measurements:
  - Electrodes: spike trains or local field potentials (LFPs)
  - Electroencephalography (EEG): Event Related Potentials (ERPs)
- Delayed response paradigm:
  - Present a sample
  - Remove the sample for a delay period
  - Test if new samples match the initial one





Liebe et al., 2012

Synaptic-Based Working Memory

Early experimental results

Fuster & Alexander (1971):

- Single unit recording in monkeys in the prefrontal cortex (PFC)
- Sample presentation (cue) evokes increased firing
- Enhanced activity persists in delay period

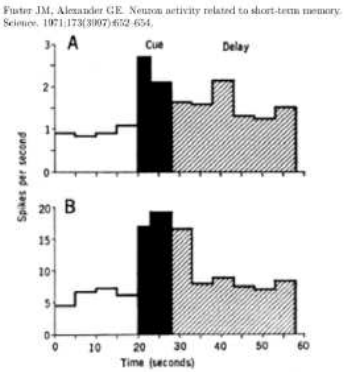
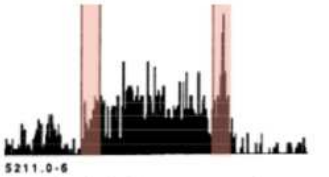


Fig. 1. Average firing of two units during five delayed response trials with 30-second delays, 20 seconds of spontaneous activity record preceding each trial. (A) Unit in prefrontal cortex; (B) unit in nucleus medialis dorsalis (MD).

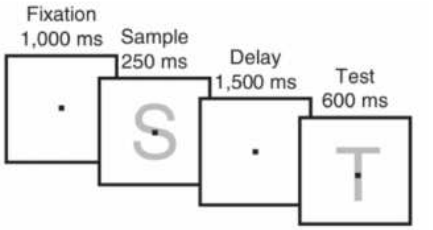


Funahashi, Bruce, and Goldman-Rakic, 1989

Information is stored in WM via persistent spiking

Criticisms to the Persistent State Paradigm

- High metabolic cost
- Data processing artifacts (neural spiking averaged over time and across trials)
- Absence of delayed activity in some experiments



Liebe et al., 2012

Short-Term Synaptic Plasticity (STP)

A pioneering study revealed that the interactions among pyramidal neurons in the PFC display synaptic facilitation lasting hundreds of milliseconds [Wang et al., Nature Neuroscience (2006)] :

Synaptic Plasticity could be relevant for WM

Short-term synaptic plasticity can be mimicked with a model developed by Tsodyks, et al. (1998) based on 2 synaptic variable :

depression $x(t)$ and facilitation $u(t)$

The post-synaptic potentials delivered by neuron i are given in this model by:

$$Jx_i(t)u_i(t)$$

- $x_i(t)$ is the fraction of still available resources after neurotransmitter depletion
- u_i is the the fraction of available resources x_i ready for use : $u_i x_i$

Short-Term Synaptic Plasticity (STP)

The evolution in time of these 2 synaptic variables is given by

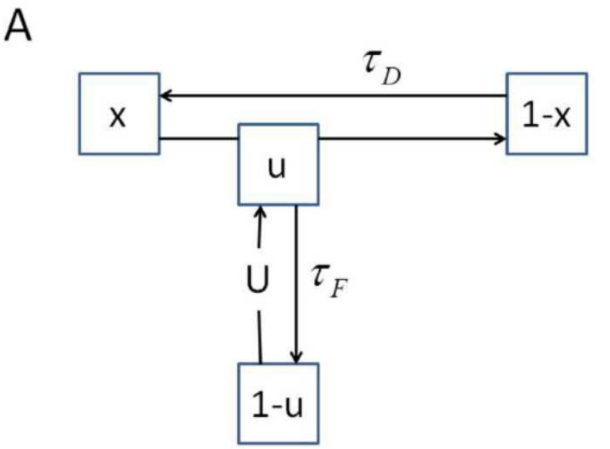
$$\frac{dx_i}{dt} = \frac{1 - x_i}{\tau_d} - u_i x_i \delta(t - t_k^{(i)}) \qquad \frac{du_i}{dt} = \frac{U - u_i}{\tau_f} + U(1 - u_i)\delta(t - t_k^{(i)})$$

Following a spike emission,

- u_i increases due to spike-induced calcium influx to the presynaptic terminal
- after which a fraction u_i of available resources x_i is consumed to produce the post-synaptic current

Between spikes,

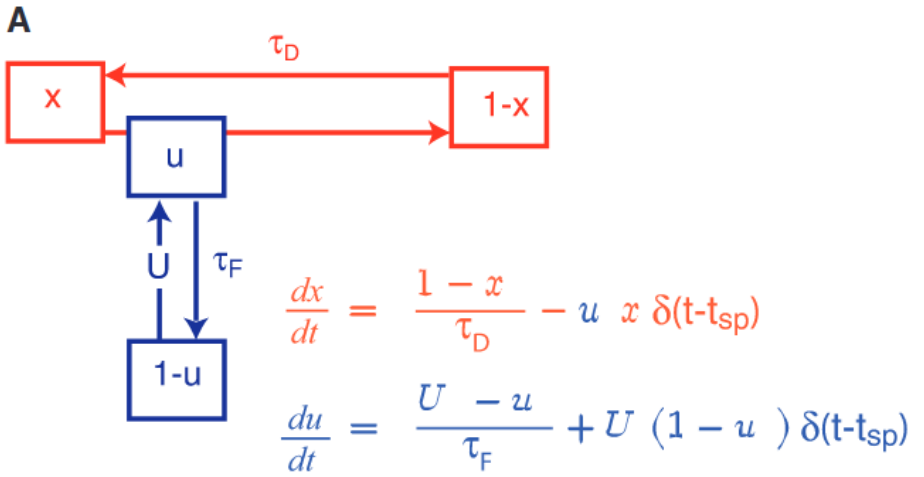
- u_i decays back to U with time constant τ_f
- x_i recovers to one with time constant τ_d

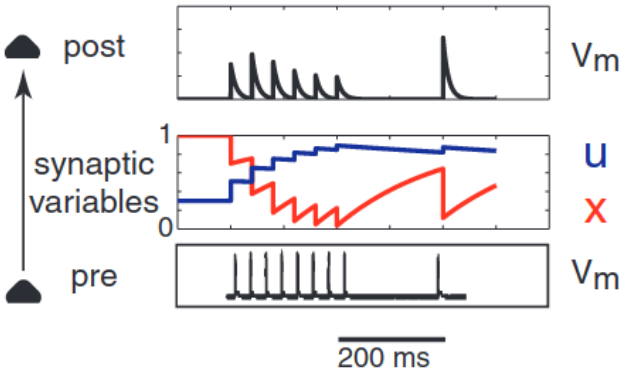


[Tsodyks, Pawelzik, Markram (1998) Neural Computation]

Short-Term Synaptic Plasticity (STP)

Facilitation Dominated Synapse $\tau_f = 1500$ ms $>> \tau_d = 200$ ms





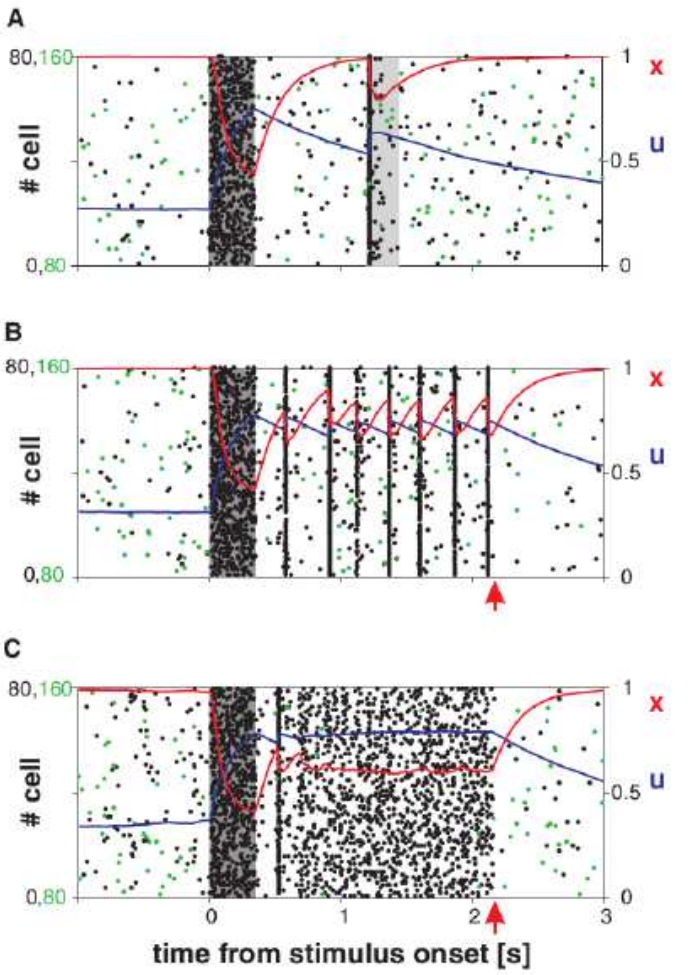
[Tsodyks, Pawelzik, Markram (1998) Neural Computation]

Synaptic Theory of Working Memory

A new paradigm

- A spiking network model for WM with synaptic depression x and facilitation u
- Cue presentation triggers: depression x & facilitation u
- Depression: Triggers population bursts needed to refresh memory
- Facilitation: Silent WM maintenance & selectivity for unspecific stimuli

[Mongillo, Barak, and Tsodyks, Science (2008)]



Synaptic Theory of Working Memory

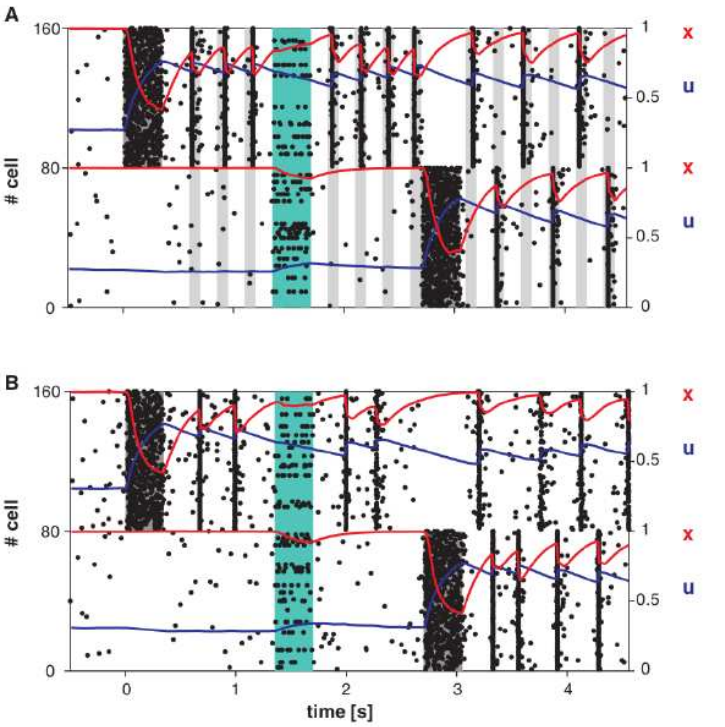
Benefits of Synaptic Theory of Working Memory

- WM is maintained in absence of spiking
- Population bursts allow interference free storage of more memory items
- Information is stored at a population level

We search for a neural mass model for WM:

- Based on short-term synaptic plasticity (STP)
- Able to exhibit spike synchrony
- Capable to give acces to experimental measures (EEGs,LFPs,ERPs)

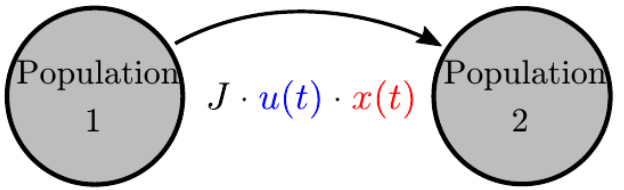
IDEA : extend the MPR model by including STP



Synaptic-Based Working Memory

Neural Mass with Synaptic Depression and Facilitation

Short-term synaptic plasticity (STP):



- Depression[4] : $\dot{x} = \frac{1-x}{\tau_\mathrm{d}} - uxr$
- Facilitation[4]: $\dot{u} = \frac{U-u}{\tau_\mathrm{f}} + U(1-u)r$

On population level: macroscopic STP

Neural mass

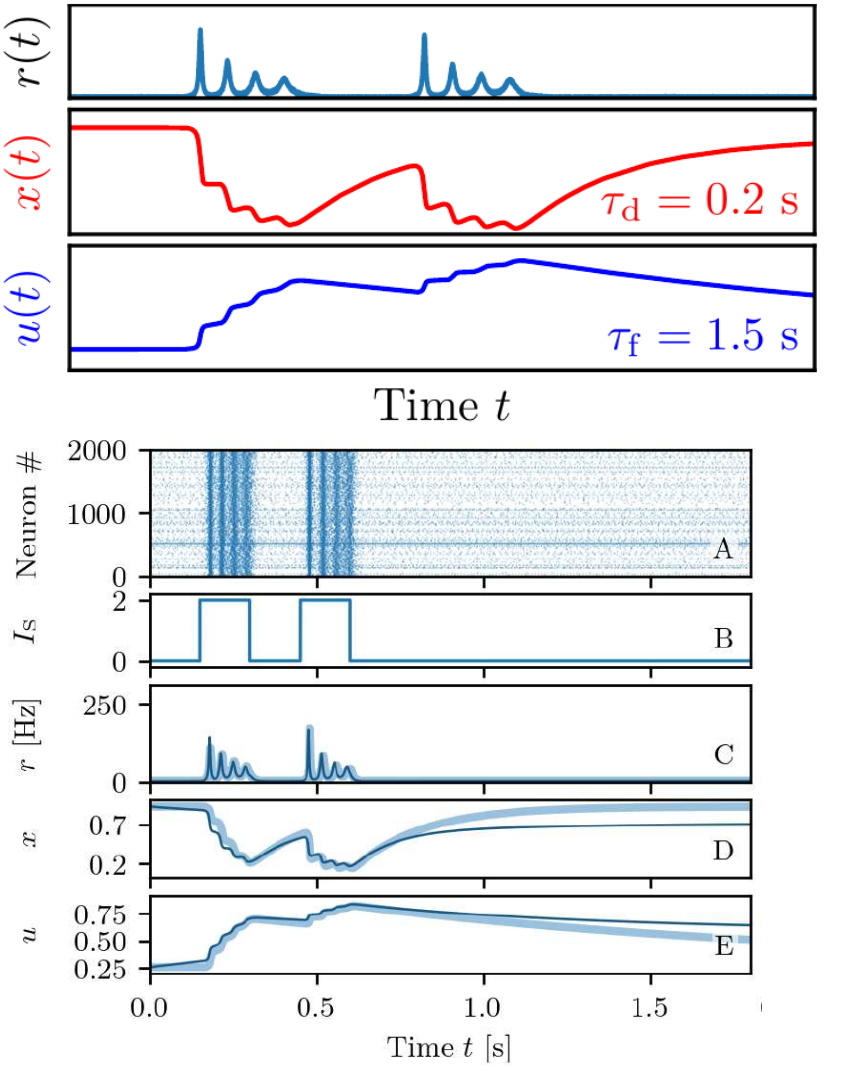
4 equations:

$$\tau_\mathrm{m}\dot{r} = \frac{\Delta}{\pi\tau_\mathrm{m}} + 2rv$$

$$\tau_\mathrm{m}\dot{v} = v^2 - (\pi\tau_\mathrm{m}r)^2 + Jux\tau_\mathrm{m}r + \bar{\eta}$$

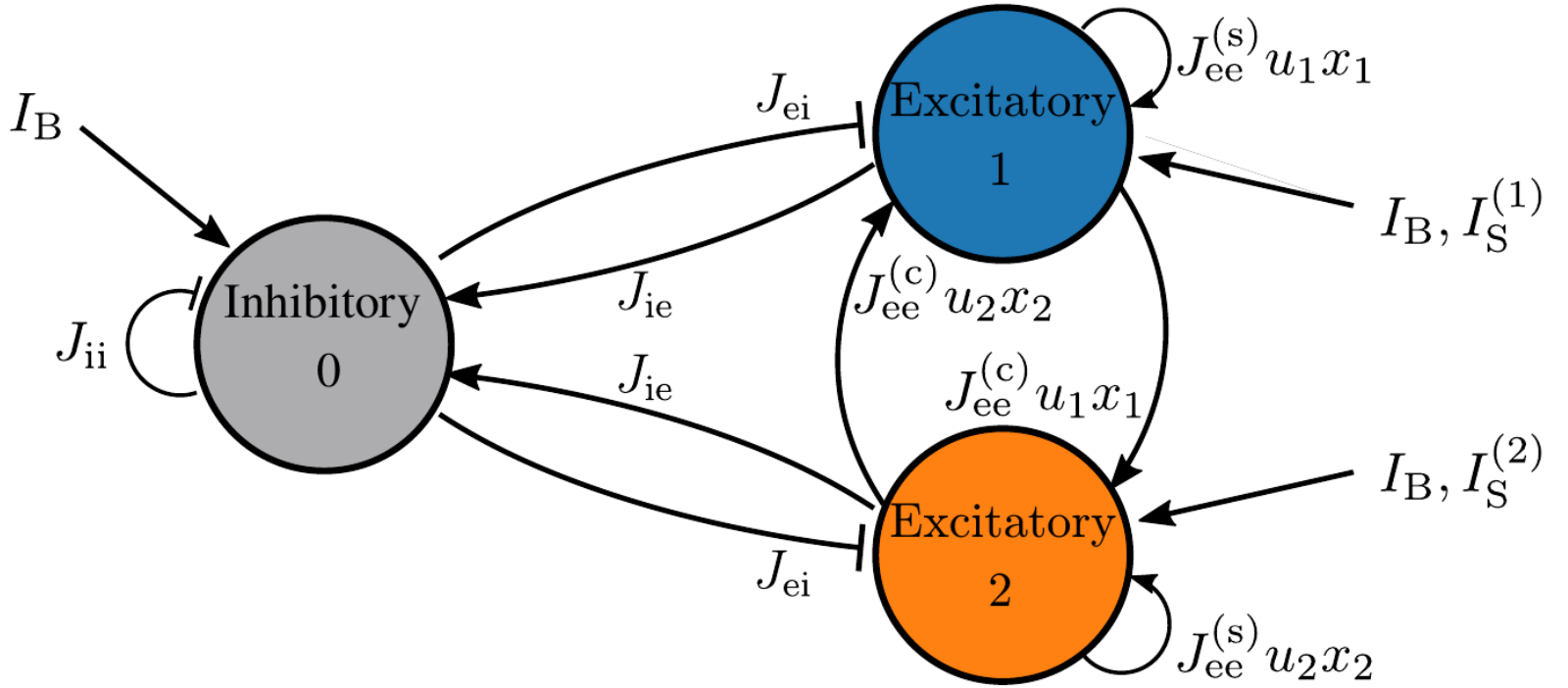
$$\dot{x} = \frac{1-x}{\tau_\mathrm{d}} - uxr$$

$$\dot{u} = \frac{U-u}{\tau_\mathrm{f}} + U(1-u)r$$



Synaptic-Based Working Memory

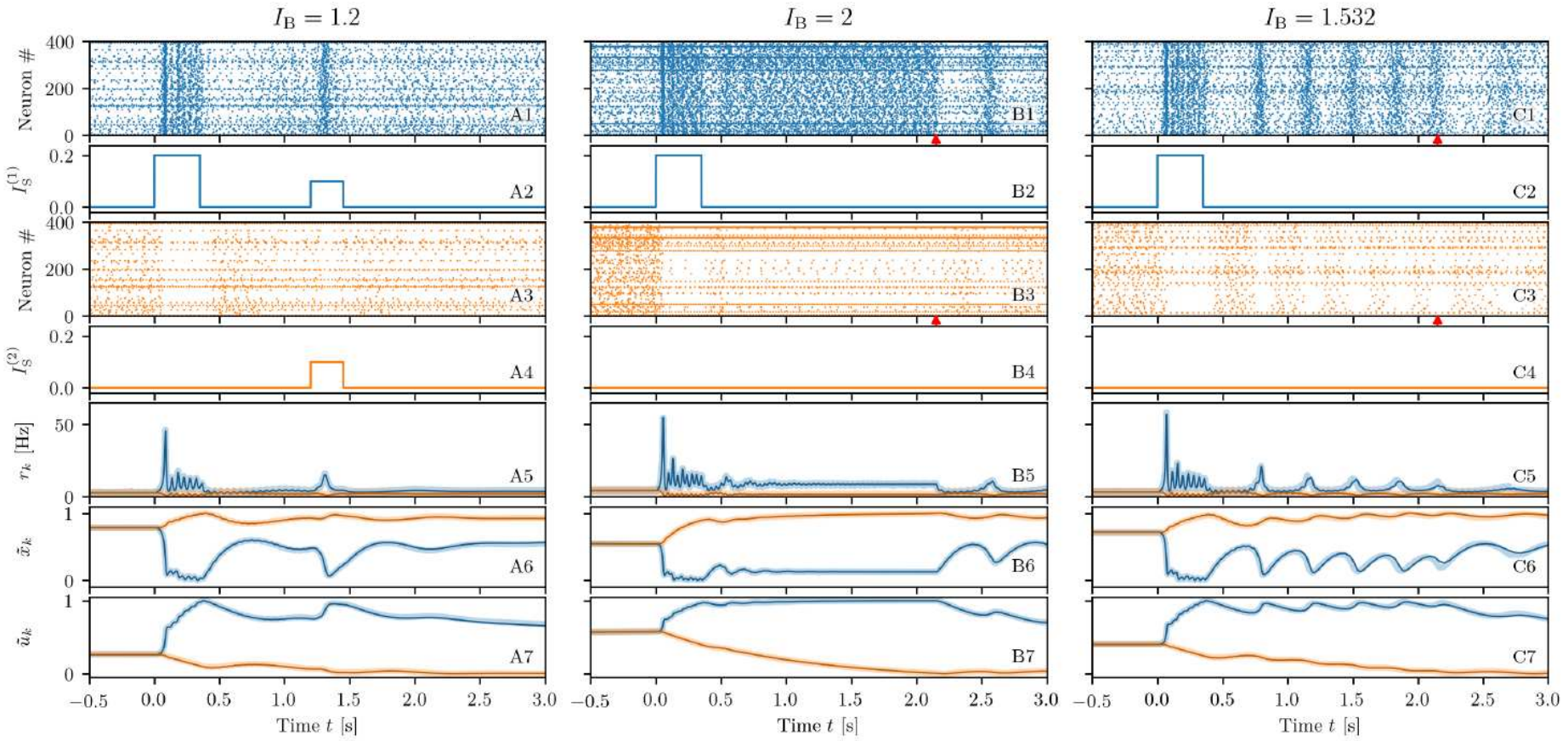
Minimal Architecture to Load up to Two Items in the WM



- Each Item is Loaded in an Excitatory Population
- The Inhibitory Pool avoids Abnormal synchronization
- 3 Neural mass Models ===> 10 Degrees of Freedom
- Comparison with networks with $N = 600,000$ neurons

Synaptic-Based Working Memory

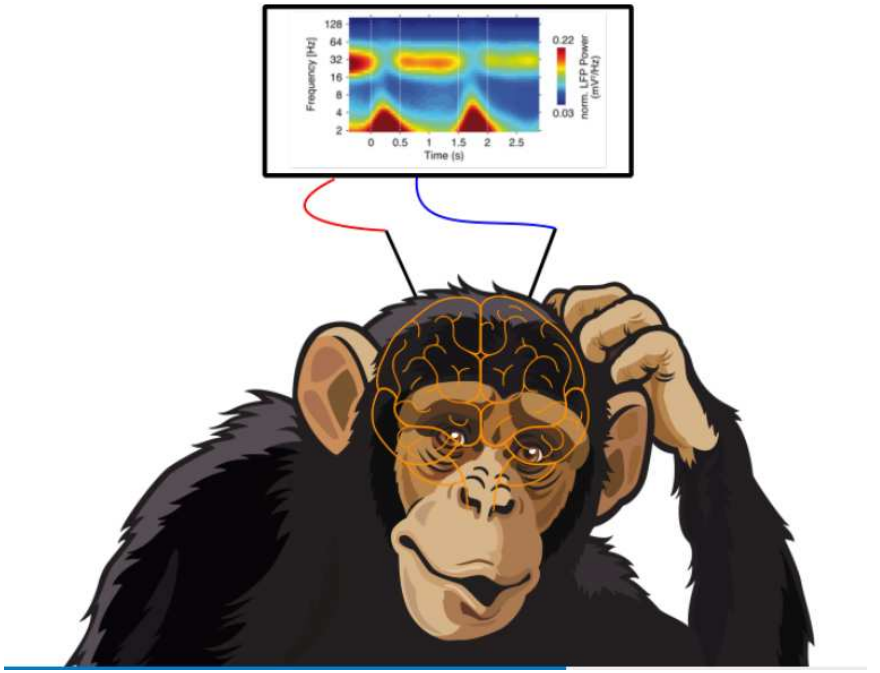
Loading of One Item in the WM



[Taher, Olmi, AT PLOS Comp Biol (2020)]

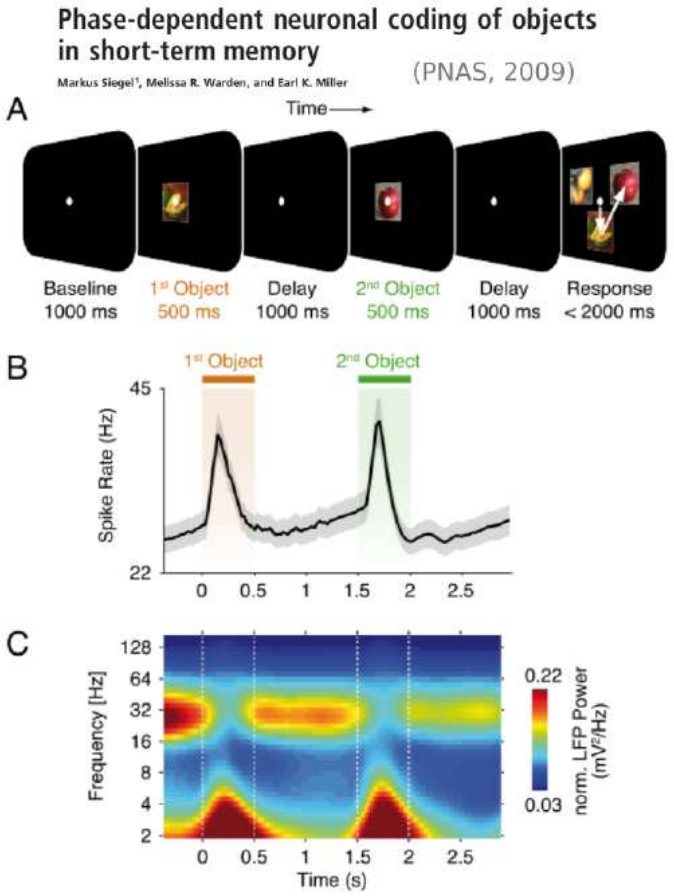
Comparison with Experimental Data

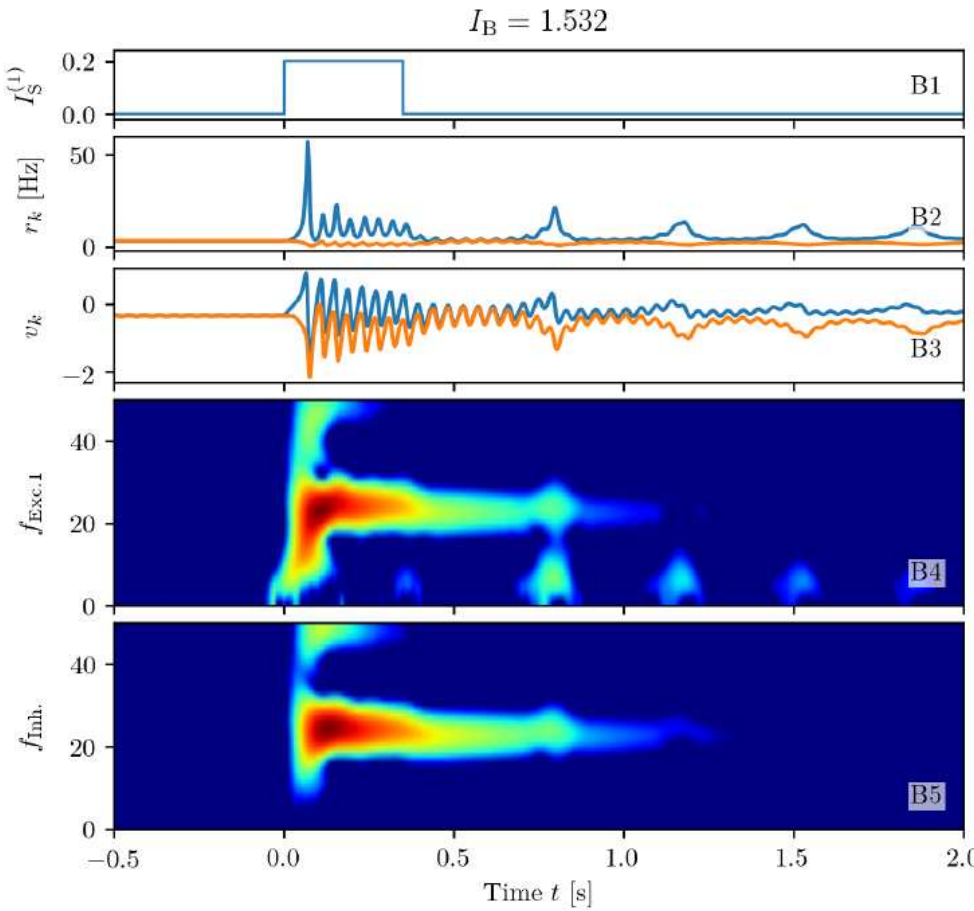
The novelty of this neural mass is that we have at disposal the mean membrane potential V and we can estimate the Power Spectra (the Spectrogram) and compare with experimental measurements at a macroscopic scale (LFPs, EEGs, ERPs)



LFP measurements in prefrontal cortex of monkeys during WM tasks

LFPs in Monkeys





[Taher, Olmi, AT PLOS Comp Biol (2020)]

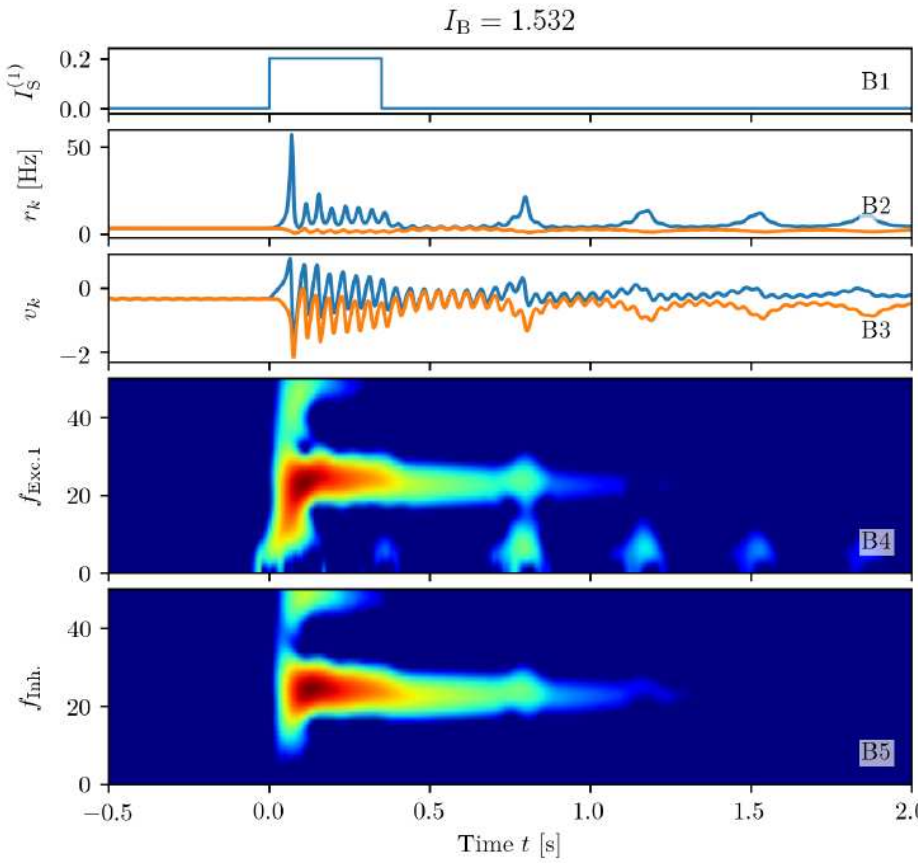
LFPs in Monkeys

Simulation resembles experimental results:

- δ-band (2 Hz - 4 Hz) activity locked to stimulus onset
- Sustained β-band (12 Hz - 25 Hz) activity

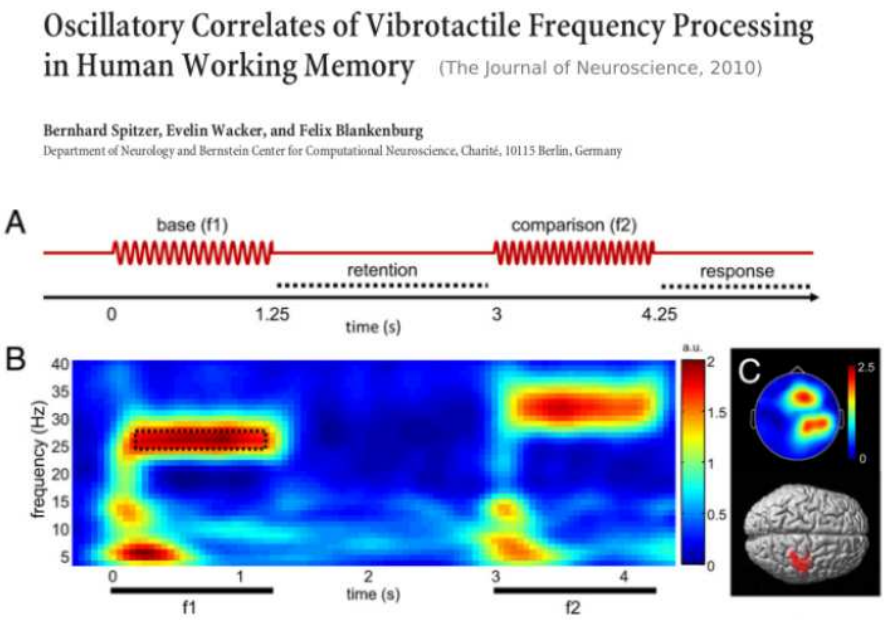
Beyond the experiment

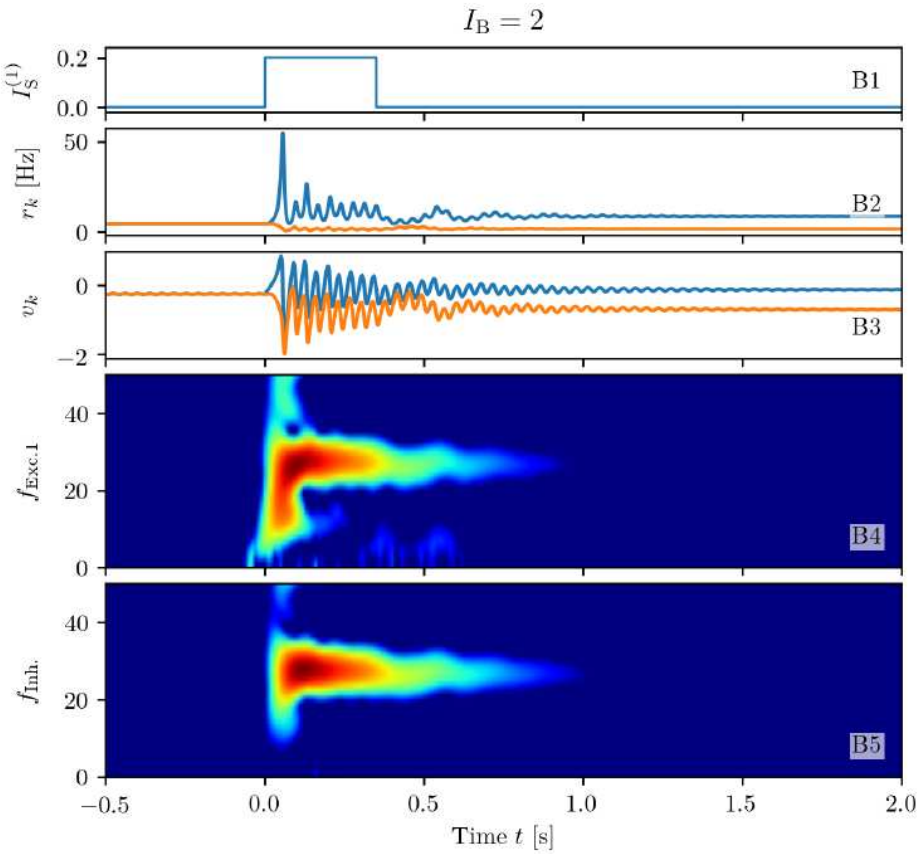
- Encoding via PING like mechanism in β-band
- Excitatory population bursts generate δ-band signal



[Taher, Olmi, AT PLOS Comp Biol (2020)]

EEGs in Humans





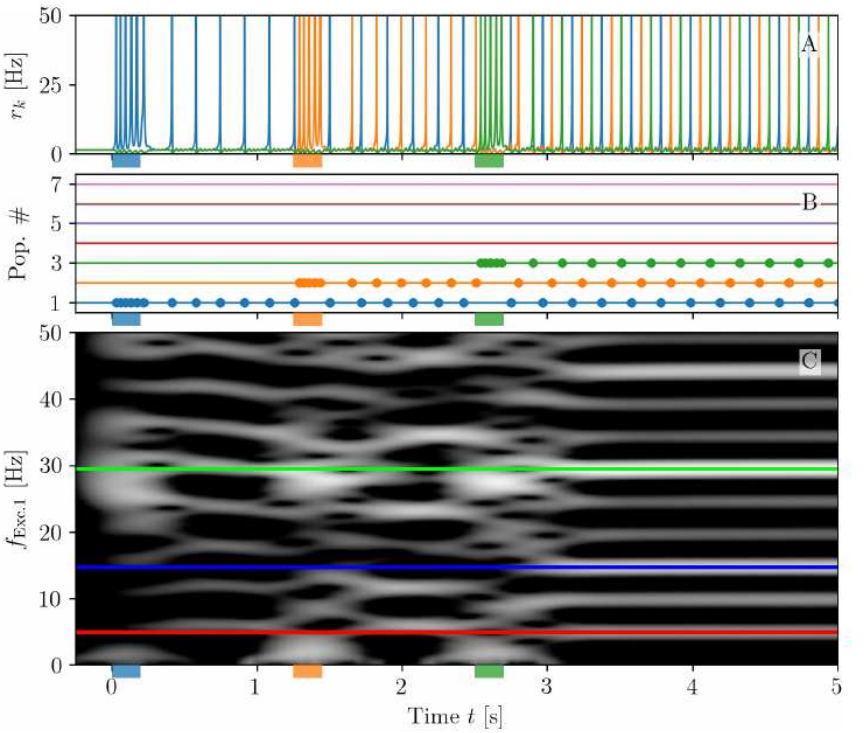
[Taher, Olmi, AT PLOS Comp Biol (2020)]

Multi-item Loading in WM

Multiple items can be loaded in an architecture with 7 excitatory populations and 1 inhibitory pool

Three Item Loading in WM

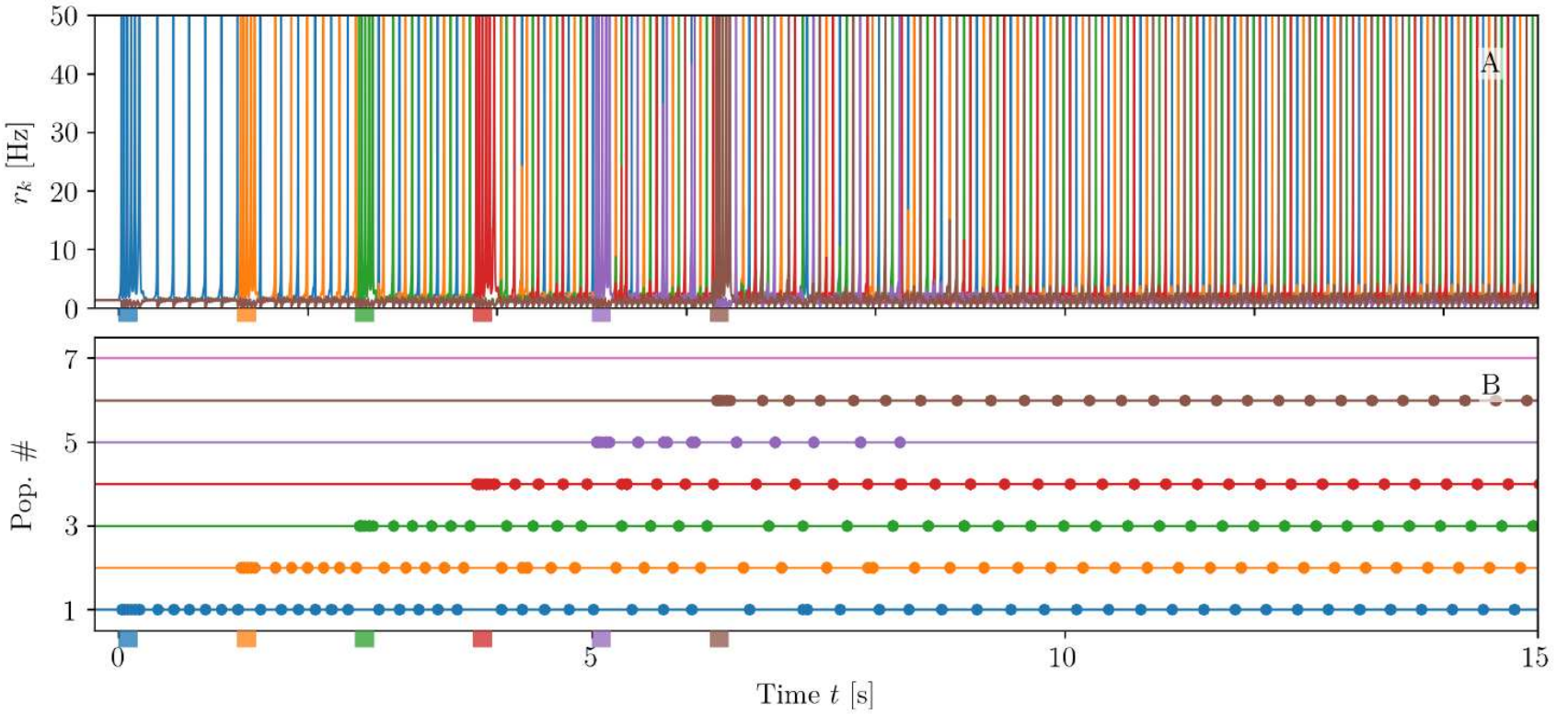
- Activity in the δ-band upon item presentation
- Fundamental memory cycle frequency f_{cycle}
- Frequency of burst emission f_{burst}
- Resonance with the β-band sub-thresold oscillations



[Taher, Olmi, AT PLOS Comp Biol (2020)]

Working Memory Capacity

A series of studies have investigated the working memory capacity and indicated as maximal number of stored items $N_c \simeq 3 - 5$

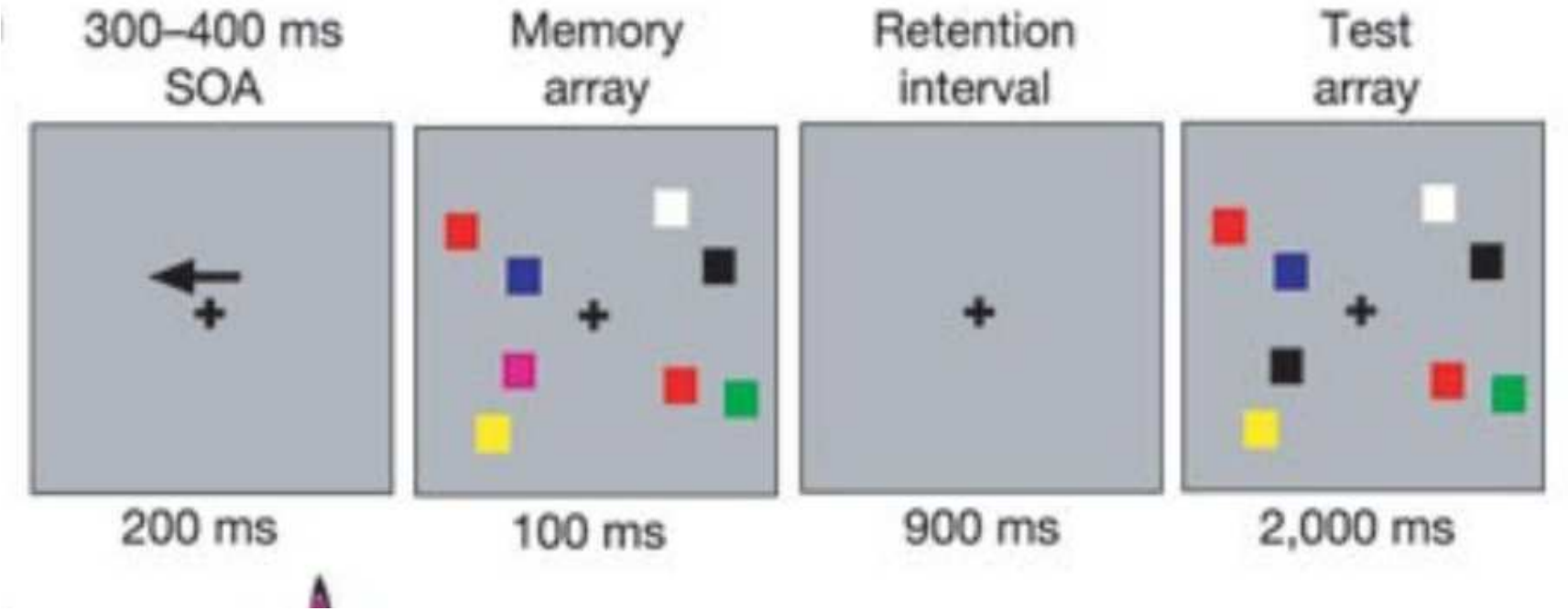


[Cowan N, Behavioral and brain sciences (2001); Cowan N, Current directions in psychological science (2010)]

Working Memory Capacity

How to measure the WM Capacity from neurophysiological data ?

Vogel et al. (Nature 2004, Nature 2005) introduced a measure of the WM capacity on humans based on event-related potentials (ERPs) from adults performing a visual memory task.



The task consists in memorizing an array of N_L colored squares

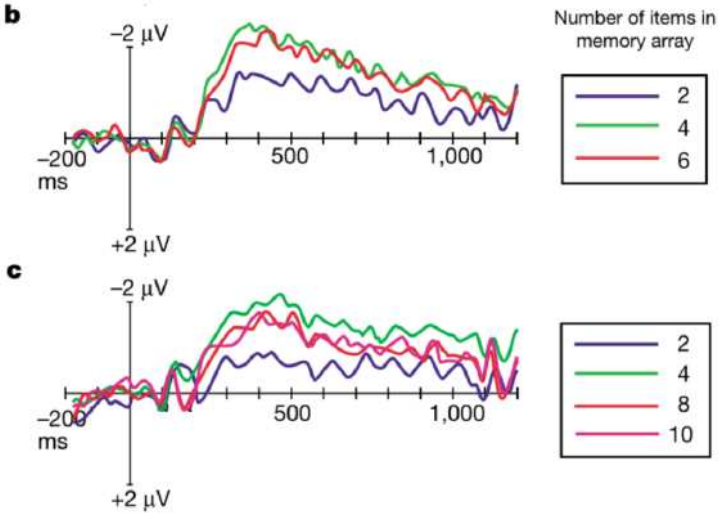
Working Memory Capacity

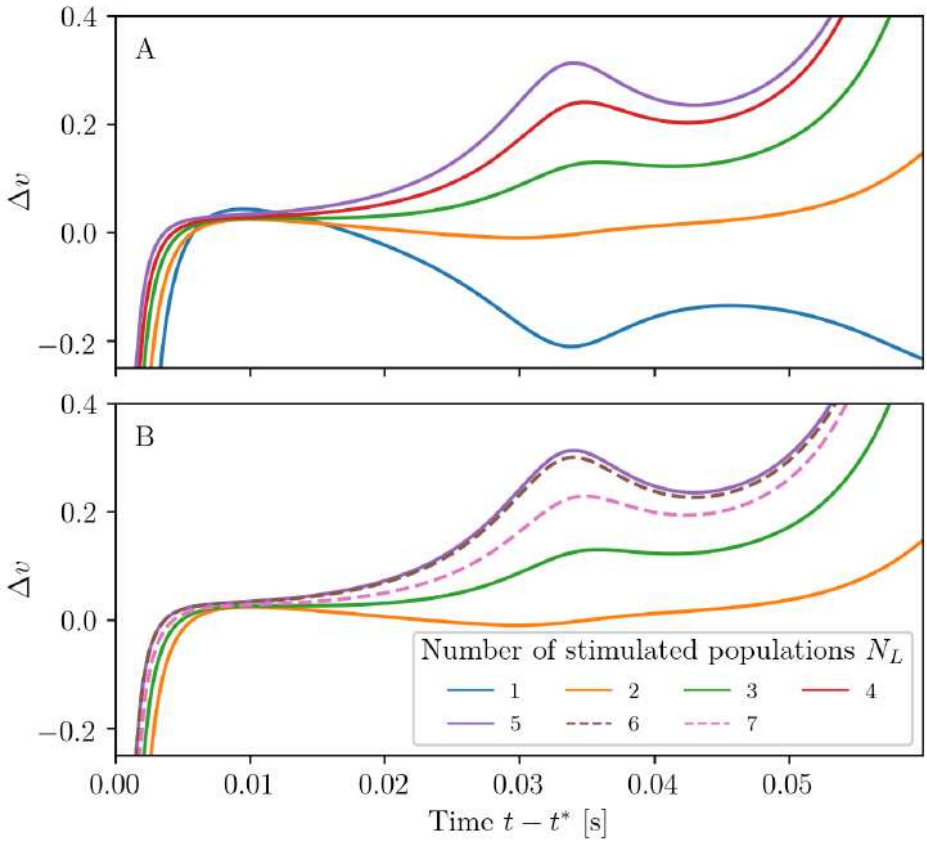
The mean membrane potential can be employed, analogously to the ERP in the experiments, as a proxy to measure the memory load and capacity

Neural activity predicts individual differences in visual working memory capacity (Nature, 2004)

Edward K. Vogel & Maro G. Machizawa

Department of Psychology, University of Oregon, Eugene, Oregon 97403-1227, USA





Neural Mass for Fluctuation Driven Populations

- The MPR Neural Mass (PRX,2015) reproduces the dynamics of one heterogeneous population of globally coupled neurons
- The MPR model can be extended to two or more interacting populations
- The MPR model can be extended to the whole connectome: [V. Jirsa, S. Olmi, G. Rabuffo, et al, bioRxiv 2 preprints (2021)]
- The MPR model has been extended to encompass delay, gap junctions, short-term plasticity, asymmetric spike forms, conductance based neurons etc

However so far this Neural Mass always concerns globally coupled neurons without noise sources, a non realistic representations of neural systems, which always present:

- background noise
- random distribution of the synaptic connections

May we develop a Neural Mass encompassing quenched and dynamical disorder sources ?

YES WE DID !

Neural Mass for Fluctuation Driven Populations

- The MPR model is based on the assumption that the distribution of the membrane potentials is Lorentzian (LD),
- The presence of dynamical disorder modifies the LD, which is now distorted
- The LD cannot be expanded in regular cumulants or moments, they all diverge

We have introduced an expasion of the LD in pseudo-cumulants, to treat distorted LD

This allow to derive a neural mass encompassing different sources of noise

- A low dimensional mean-field model reproducing the dynamics of spiking QIF neurons
  - subject to background noise
  - and/or arranged in sparse random network

[Goldobin, diVolo, AT, "A reduction methodology for fluctuation driven population dynamics", preprint (2020)]

The Model

For a heterogeneous population of QIF neurons subject to different noise sources we can derive a 4 dimensional neural mass model

$$\dot{r} = \frac{\Delta_\eta + \Delta_J r + p_2}{\pi} + 2rv$$

$$\dot{v} = I_0 + \eta_0 + J_0 r - \pi^2 r^2 + v^2 + q_2$$

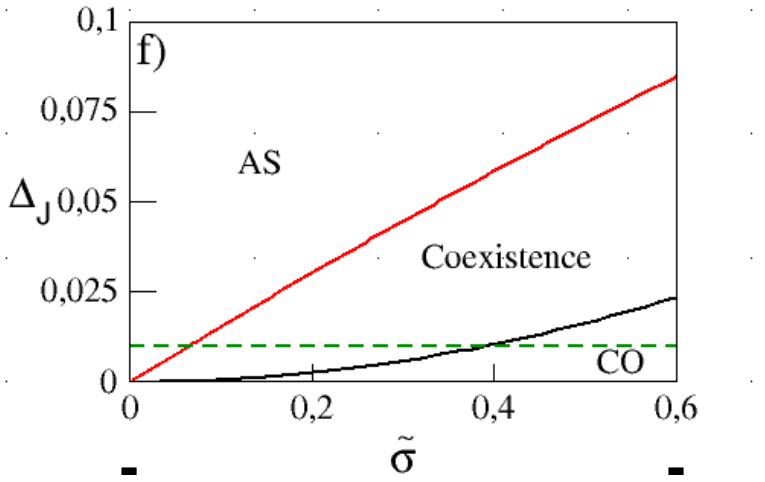
$$\dot{q}_2 = 2\mathcal{N}_R + 4(q_2 v - \pi p_2 r)$$

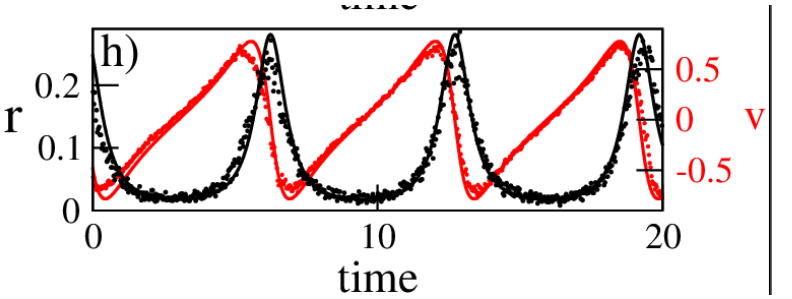
$$\dot{p}_2 = 2\mathcal{N}_I + 4(\pi q_2 r + p_2 v)$$

- Four Macroscopic Variables :
  - Mean Membrane Potential and Firing Rate (v, r)
  - Two Dynamical Variables for the LD Distortions (q_2, p_2)
- Quenched heterogeneities
  - Excitabilities : LD with (η_0, Δ_η)
  - Synaptic Couplings : LD with (J_0, Δ_J)
- Noise Sources are encompassed in the terms $(\mathcal{N}_R, \mathcal{N}_I)$

Background Noise

Globally coupled inhibitory network of QIF neurons each subject to independent additive Gaussian noise of variance σ^2 : $(\mathcal{N}_R = \sigma^2, \mathcal{N}_I = 0)$



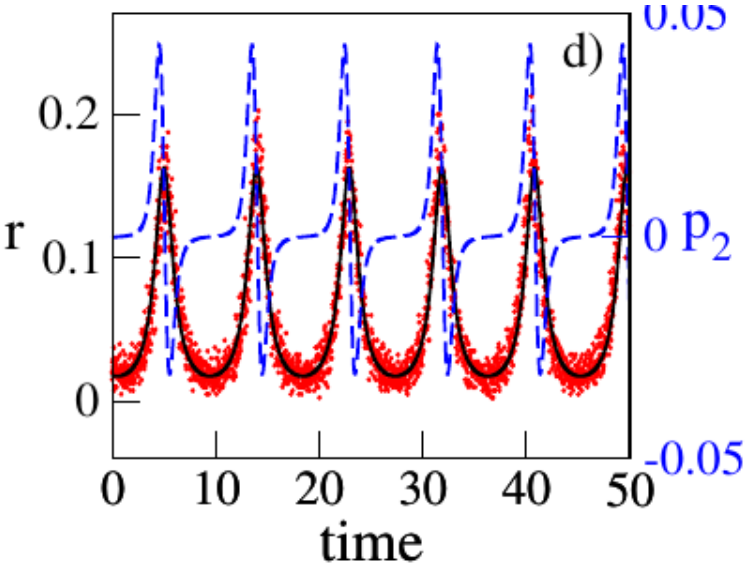


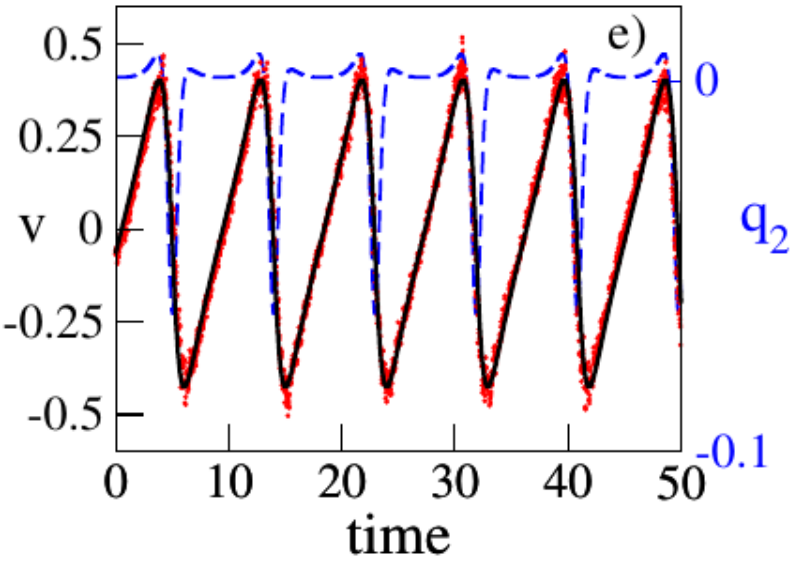
- the bifurcation diagram reveals Asynchronous States (ASs), Collective Oscillations (COs) and regions of coexistence of ASs and COs
- the neural mass results are in agreement with the network simulations
- the MPR model cannot capture even qualitatively these regimes displayed by the noisy spiking network
- Noise induced COs in absence of any synaptic or delay time scale

[Goldobin, diVolo, AT, bioRxiv (2020)]

Sparse Networks

- Random network with in-degrees k_j distributed as a LD, with median K and HWHM $\Delta_K = \Delta_0 K$.
- We assume that each neuron receive k_j independent Poissonian spike trains with rate r
- This amounts to set $(\mathcal{N}_R = (J_0^2 r)/(2K), \mathcal{N}_I = -\Delta_0 \mathcal{N}_R)$





Summary

The Next Generation Neural Masses of MPR open a complete new perspective for realistic simulations of heterogenous spiking networks

- The Neural Masses reproduce with high fidelity the network dynamics:
  - not only the firing rate but also the sub-threshold membrane potential dynamics
  - synchronization and de-synchronization phenomena
- The Neural Masses reproduces relevant neuroscience phenomena:
  - θ-γ cross coupling in the hippocampus and other areas
  - fast and slow γ oscillations in the hippocampus
  - working memory processes
- the neural mass MPR can be extended to capture fluctuation driven phenomena present in realistic brain circuits

[Ott, & Antonsen Chaos (2008)] 753 citations

Collaborators

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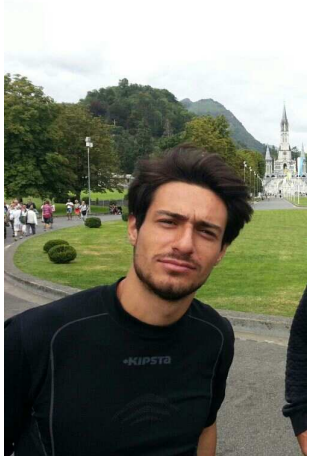
Andrea Ceni



Marco Segneri

Honjie Bi

Matteo di Volo

Denis Goldobin

Fundings & Publications

- M. di Volo, AT, "Transition from asynchronous to oscillatory dynamics in balanced spiking networks with instantaneous synapses", Phys. Rev. Lett. (2018)
- H. Bi, M. Segneri, M. di Volo, AT "Coexistence of fast and slow gamma oscillations in one population of inhibitory spiking neurons", Physical Review Research (2020)
- A.Ceni, S. Olmi, AT, D. Angulo Garcia, "Cross frequency coupling in next generation inhibitory neural mass models", Chaos (2020)
- M. Segneri, H.Bi, S. Olmi, AT, "Theta-nested gamma oscillations in next generation neural mass models", Frontiers in Computational Neuroscience (2020)
- H. Taher, AT, S. Olmi, "Exact neural mass model for synaptic-based working memory", PLOS Computational Biology (2020)
- D. Goldobin, M diVolo, AT, "A reduction methodology for fluctuation driven population dynamics", bioRxiv (2020)