Coexistence of fast and slow gamma oscillations in one population of inhibitory spiking networks

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Introduction

Oscillations with different rhythms are widespread in brain circuits. Neuronal gamma oscillations crucially depending on the presence of interneurons have been observed in several brain structures. Specifically, in the region CA1 of the hippocampus experimental evidence indicates the presence of multiple Gamma rhythms: fast (60-110 Hz) and slow(30-50 Hz) Gamma events. Here we consider balanced sparse inhibitory networks of quadratic integrate-and-fire (QIF) neurons, whose dynamics can be rewritten at a mean field (MF) level in terms of few macroscopic observables [1, 2]. In the MF we observe the coexistence of foci and oscillatory dynamics in proximity of sub-critical Hopf bifurcations. In the network dynamics we observe the coexistence of collective oscillations (COs) in the slow and fast Gamma band emerging within the same neuronal population. The slow and fast gamma rhythms are generated via two different mechanisms: the slow gamma COs are due to the microscopic irregular dynamics, characteristic of the balanced dynamics, which turns the damped oscillations towards the MF focus in sustained COs [2]; the fast gamma COs are instead related to the oscillatory branch emerging via the sub-critical Hopf bifurcation from the asynchronous state. Furthermore, to make a closer contact with the experimental observations [3, 4], we consider the modulation of the gamma rhythms induced by a theta forcing. We observe phase-phase coupling between the fast and slow gamma oscillations and the theta forcing, with the slow gamma occurring earlier during the theta cycle.

Network of QIF inhibitory neurons

We consider a balanced network of N QIF neurons described by the following equations:

$$\tau_m \dot{v}_i(t) = I_0\sqrt{K} + v_i^2(t) - \frac{J_0\tau_m}{\sqrt{K}} y_i(t) \quad \textbf{(1a)}$$

$$\tau_d \dot{y}_i(t) = -y_i(t) + \sum_j \epsilon_{ij}\delta(t - t_j(m)) \ , \quad \textbf{(1b)}$$

where $I_0\sqrt{K}$ is the DC current, τ_d synaptic time and τ_m the membrane time constant. The synaptic field y_i is the linear super-position of all the IPSPs $s(t) = \exp(-t/\tau_d)$ received by the neuron i from its pre-synaptic neurons in the past, ϵ_{ij} is the adjacency matrix of the network and $k_i = \sum_j \epsilon_{ij}$ is the in-degree of neuron i. We consider sparse networks with in-degrees k_i extracted from a Lorentzian distribution with median K and HWHM $\Delta_k = \Delta_0\sqrt{K}$.

Mean field model for a sparse network

For this sparse network we derived an effective mean-field (MF) by employing recently developed reduction techniques for QIF networks [1, 5]. In particular, for the balanced network we have that each neuron i is subject to an average inhibitory synaptic current of amplitude $g_0 k_i Y/(\sqrt{K})$ proportional to its in-degree k_i. Therefore, by interpreting the quenched connectivity disorder as random synaptic couplings we can consider the neurons as fully coupled, but with couplings distributed as a Lorentzian of median $\bar{g} = -J_0\sqrt{K}$ and HWHM $\Gamma = J_0\Delta_k$ [2]. The MF dynamics is then

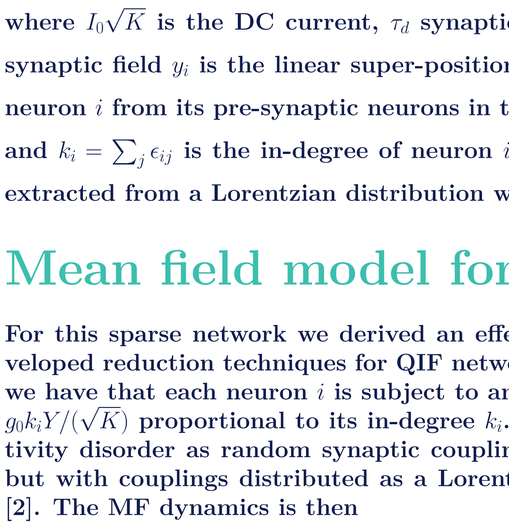
$$\tau_m \dot{R} = \frac{\Delta_0 J_0}{\pi} Y + 2RV \quad \textbf{(2a)}$$

$$\tau_m \dot{V} = V^2 + \sqrt{K}(I_0 - J_0\tau_m Y) - (\pi\tau_m R)^2 \quad \textbf{(2b)}$$

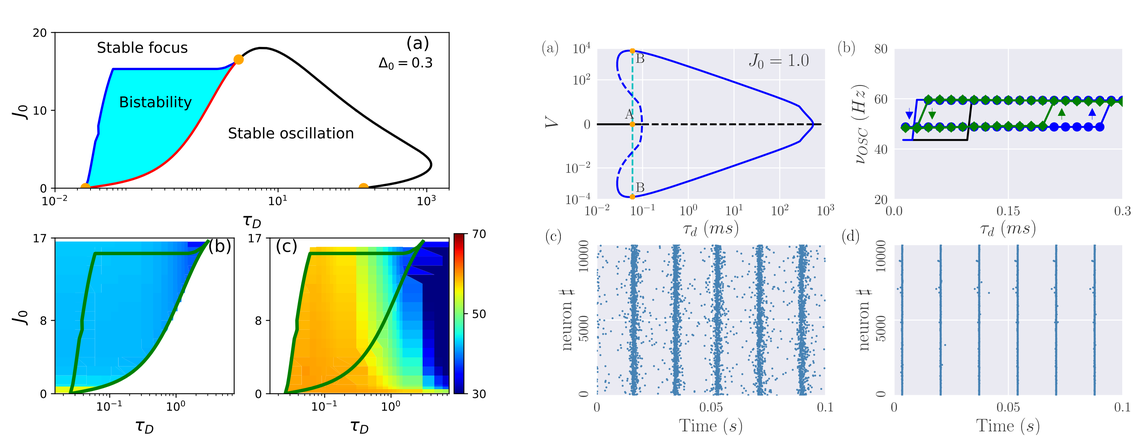
$$\tau_d \dot{Y} = -Y + R; \quad \textbf{(2c)}$$

R is the firing rate, V the average membrane potential and Y the average synaptic field.

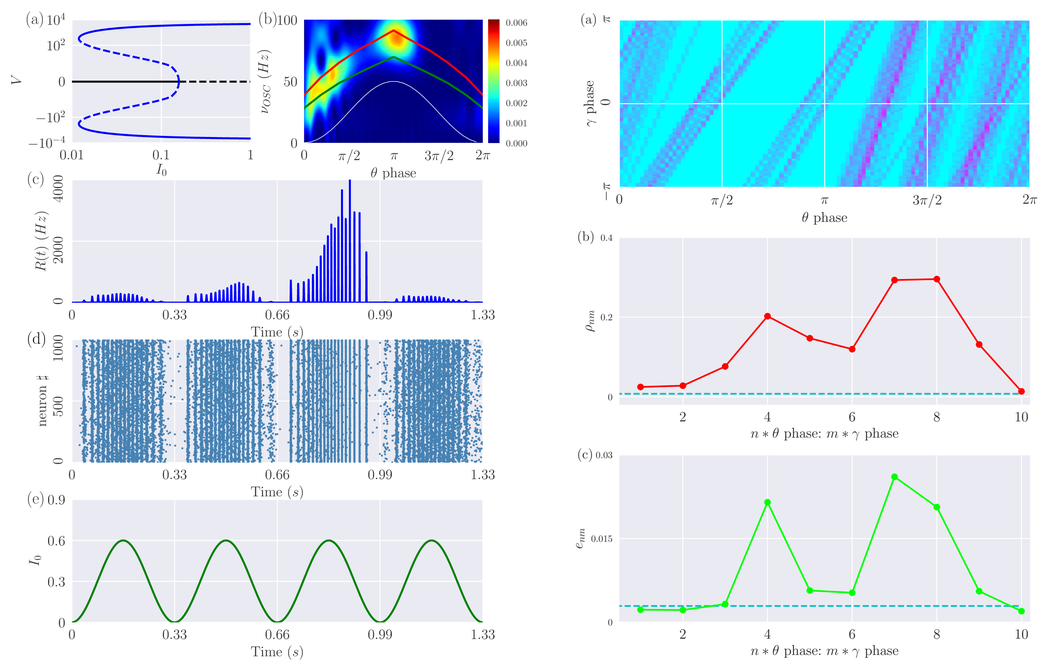
Simulation results and MF results



Collective variables V (a), R (b) and Y (c) versus time, obtained from simulations of the spiking network **(1)** (blue circles) as well as from the MF formulation **(2)** (black line). In (d) the corresponding raster plot is displayed, revealing clear COs with frequency $\nu_{OSC} \simeq 24$ Hz. Dynamics of the network of $N = 10000$ neurons with median in-degree $K = 1000$ and $\Delta_0 = 0.3$. $I_0 = 0.25$, $J_0 = 1.0$ and $\tau_d = 15$ ms.



Left Figures: (a) Hopf boundaries for the MF **(2)** [5]: region of coexistence of stable foci and oscillations shown in blue. The frequency of the damped oscillations ν_D towards the focus are reported in (b), while the oscillation frequencies ν_O of the limit cycles in (c). Right Figures: sub-critcal Hopf bifurcation. Coexistence of an asynchronous and a synchronous state in the MF, in the network these states correspond to two coexisting COs with different frequencies (b) and dynamics as shown by the raster plots (c-d).



Left Figures: Fast and slow gamma oscillations entrainment with the theta phase. (a) Bifurcation diagram reporting the extrema of V as a function of I_0 displaying stable (solid line) and unstable solutions (dashed lines) for foci (black) and limit cycles (blue). (b-e) Results of the simulation of the spiking network with forcing $I_0(t) = I_\theta[1 - cos(2\pi\nu_\theta t)]$ (e). (b) Spectrogram of the COs versus the phase of the theta forcing, in the same panel are shown ν_D (green line), ν_O (red line) and I_0 (white line). Right Figures: Phase-phase coupling $n : m$ between theta and gamma oscillations. (a) Bidimensional histogram for the $(\theta(t), \gamma(t))$-phases; (b) Kuramoto order parameter ρ_{nm} and (c) normalized entropy e_{nm} for the phase difference $\Delta_{nm}(t)$. Parameters are the same as in left figure, apart for $\nu_\theta = 10$ Hz.

Conclusions

- We derived an effective MF for a balanced spiking inhibitory QIF network with finite synaptic decay.
- In the MF we observe the coexistence of a stable focus with a stable oscillatory state
- In the network, due to the dynamical balance we observe two collective oscillatory states corresponding to slow and fast Gamma rhythms in a single inhibitory population [6].
- Our results on theta locking and on the origin of fast and slow gamma oscillations can also be interpreted in terms of experimental findings reported for freely behaving rats [3, 4].

References

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