Mean field predictions for interacting excitatory and inhibitory populations of spiking neurons

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Abstract

We extend a mean-field approach to the dynamics of populations of spiking neurons introduced in [8] to the multi-population case. In particular, in view of the phenomenological interest of balanced networks of excitatory and inhibitory neurons, we examine the case of two interacting populations of excitatory and inhibitory neurons, and compare theoretical predictions about the network relaxation times and spectral properties with simulation data.

1 Introduction

In [8] an approach was developed for a mean field treatment of time-dependent properties of the collective spiking activity, for an interacting population of integrate-and-fire (IF) neurons (for previous, and complementary approaches to the dynamic mean field theory of IF neurons see [2,1,4]). The theory incorporates the role of spike transmission delays, and of the finite number of neurons in the population. As a result, for asynchronous regimes of activity, we could compute the stability conditions, characterize the relaxation times of the transient response of the population, and calculate the power spectrum of its collective activity $\nu(t)$.

In the present work we extend the approach to the case of two interacting populations of excitatory and inhibitory IF neurons, list some theoretical predictions and compare those to simulation data.

2 Theory

Following [8], the network of IF neurons is described in the diffusion approximation via a Fokker-Planck (FP) equation for the probability density $p(v,t)$

of the membrane potential v of the excitatory and inhibitory neurons. The evolution of the $p(v,t)$ is driven by the (time-dependent) moments of the input current to the neurons, μ and σ^2, which depend both on 'external' spikes from outside the network, and on the recurrent activities ν^E and ν^I inside the network. In stationary condition, the static solution of the FP equation provide the 'fixed point', mean field solution which the network self-consistently sustain, up to finite-size fluctuations: the latter is equivalent to the solution (ν_0^E, ν_0^I) of the self-consistency equation $\nu^{E,I} = \Phi^{E,I}(\nu^E, \nu^I)$, where the single-neuron transfer function Φ is the inverse of the 'first passage time' to the emission threshold θ with input current (μ, σ^2), which in the network are in turn functions of the emission rates. By expanding the Fokker-Planck operator L onto the moving basis (see [6,5]) of its eigenfunctions $\phi_n(v,t)$ (with coefficients $a_n^{E,I}$), and taking into account the corresponding expansion for the emission rates $\nu^{E,I}$, it is possible to derive a closed (though infinite) system of equations that govern the time evolution of the population activities $\nu^{E,I}$ [8]. Given a fixed point (ν_0^E, ν_0^I), we can linearize the above equations, as follows:

$$\begin{cases} \dot{\mathbf{a}}^E(t) = \mathbf{\Lambda}^E\, \mathbf{a}^E(t) + \mathbf{c}^{EE}\, \dot{\nu}^E(t-\delta) + \mathbf{c}^{EI}\, \dot{\nu}^I(t-\delta) \\ \nu^E(t) = \nu^E(t-\delta)\, \partial_{\nu^E} \Phi^E + \nu^I(t-\delta)\, \partial_{\nu^I} \Phi^E + \mathbf{f}^E \cdot \mathbf{a}^E(t) \\ \dot{\mathbf{a}}^I(t) = \mathbf{\Lambda}^I\, \mathbf{a}^I(t) + \mathbf{c}^{IE}\, \dot{\nu}^E(t-\delta) + \mathbf{c}^{II}\, \dot{\nu}^I(t-\delta) \\ \nu^I(t) = \nu^E(t-\delta)\, \partial_{\nu^E} \Phi^I + \nu^I(t-\delta)\, \partial_{\nu^I} \Phi^I + \mathbf{f}^I \cdot \mathbf{a}^I(t) \end{cases}, \qquad (1)$$

Bold symbols denote vectors, whose components are the (infinite) modes of the spectral expansion; superscripts E and I refer to the excitatory and inhibitory populations, respectively. All quantities which do not depend on time are meant to be calculated at the fixed point defined by $(\nu^E = \nu_0^E, \nu^I = \nu_0^I)$ and $(\mathbf{a}^E = \mathbf{a}^I = 0)$, and $\mathbf{a}$ and ν are now the first-order perturbations with respect to the fixed point, stationary state. $f_n = -\sigma^2(v,t)/2\, \partial_v\, \phi_n(v,t)|_{v=\theta}$ $(n \neq 0)$; $c_n = \langle \partial_\nu\, \psi_n | \phi_0 \rangle$; ψ_n are the eigenfunctions of the adjoint operator L^+ and $\langle . | . \rangle$ is a suitable inner product. c_n are coupling terms, in that for uncoupled neurons μ and σ do not depend on the recurrent frequency ν, and $\partial_\nu\, \psi_n$ vanishes. $\mathbf{\Lambda}$ is the diagonal matrix of the common eigenvalues of L and L^+. For simplicity in Eq. (1) a single allowed synaptic delay δ appears. In [7] we examined the case of a non trivial delay distribution for a single neural population.

By taking the Laplace transforms $\nu^{E,I}(s)$ of $\nu^{E,I}$, and by studying their singularities in the complex plane, one can characterize the relaxation times of the transient network response to a perturbation (associated with the real part of the poles of $\nu^{E,I}(s)$), and its resonant frequencies (related to the imaginary part of the poles).

It turns out that a relevant distinction relates to the population being in a 'noise dominated' (ND), subthreshold regime, in which the fluctuating part of the input current to the neurons is essential in driving the neurons' firing, or in a 'signal dominated' (SD), suprathreshold regime, in which neurons fire in a quasi-deterministic way.

Analogously to the single population case, two families of poles emerge from the analysis: 'diffusion' poles, which reflect for each population the SD or ND nature of the neural dynamics, and whose imaginary parts are very close to the multiples of the population emission rate; 'transmission' poles, reflecting the effective spikes transmission delays δ, which are assumed to include the synaptic integration times. The transmission poles are spaced by $2\pi/\delta$ on the imaginary axis.

3 Simulation results

We present a few phenomenological implications of the above analysis by comparing the transient response (Fig. 2)and the spectral properties (Fig. 1) for a simulated network of excitatory and inhibitory IF neurons, and the theoretical predictions for the real and imaginary parts of the diffusion and transmission poles.

Data presented in the Figures are taken from a simulation of 7500 (excitatory + inhibitory) neurons with constant leakage ('linear neurons', see [3]); the parameters and the external rates are such that in stationary conditions the expected emission frequencies are $\nu_0^E = 7$ Hz for the excitatory neurons and $\nu_0^I = 20$ Hz for the inhibitory ones (mean field fixed point). Effective spike transmission delays are taken to be uniform across the network, with $1/\delta = 500$ Hz.

In Fig. 1 we show the power spectrum of the excitatory ($P_E(\omega)$, left) and inhibitory ($P_E(\omega)$, right) neurons in the coupled excitatory-inhibitory network, in stationary conditions. $P_E(\omega)$ exhibits a small and broad peak at $\omega/2\pi \simeq 7$ Hz, and a prominent, high peak at $\omega/2\pi \simeq 20$ (with its harmonics). The former peak is predicted to be at 7 Hz by the theory ('diffusion' peaks occur at multiples of the fixed point emission rate ν_0 of the population). The asymptotic value of $P_E(\omega)$ is very close to the value 0.0028, which equals the ratio ν_E/N_E between the average emission rate of excitatory neurons and their number, which is the variance of the expected finite-size white noise. The peak around 20 Hz is due to the interaction with the inhibitory population: both the excitatory and the inhibitory populations are in a SD regime. On the other hand, the C_{EE} connectivity was chosen to be very small, compared to the C_{II}, C_{IE} and C_{EI} (C_{XY} denotes the "from Y to X" connectivity); this, combined

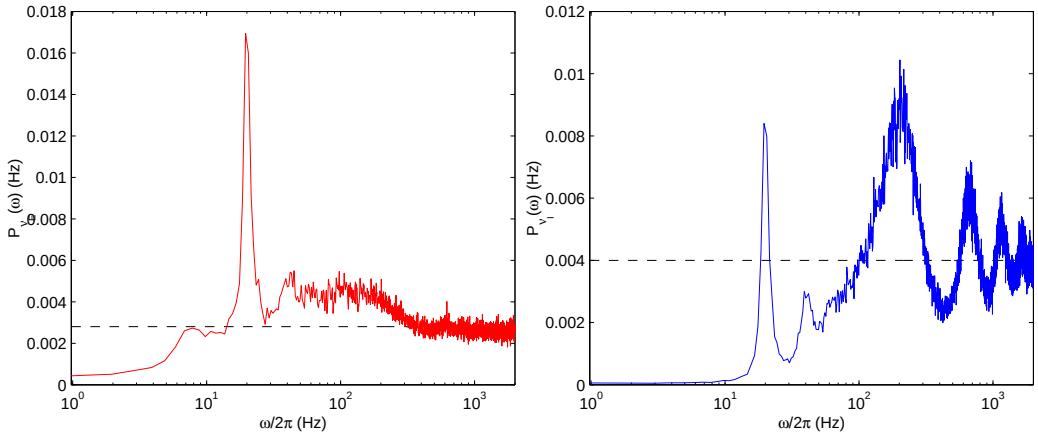

Fig. 1. Power spectral densities of the excitatory neurons (left panel) and inhibitory neurons (right panel) in the coupled excitatory-inhibitory network, in stationary conditions.

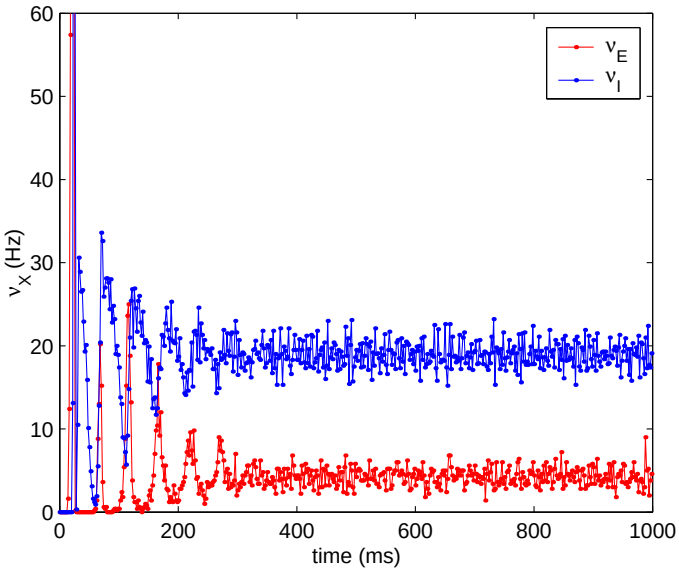

Fig. 2. Transient response of excitatory neurons (red) and inhibitory neurons (blue) in the coupled excitatory-inhibitory network. All neurons have resting membrane potential at $t = 0$, when a flux of external spikes to both the excitatory and inhibitory neurons is switched on.

with $\nu_E < \nu_I$, conspires to make the excitatory spectrum sensitive only to low-ω features of the inhibitory activity. Therefore, the inhibitory diffusion peak 'propagates' in $P_E(\omega)$, while the high-ω transmission peaks, expressing the global recurrent interactions in the network, are filtered in $P_E(\omega)$. The first transmission peak is predicted to be at $\omega = 230$ Hz, which is pretty close to the observed frequency of the first peak in $P_I(\omega)$.

For the chosen coupling and connectivity parameters, the real part of the transmission poles is predicted to be greater in module than the one of the

diffusion poles; the first inhibitory diffusion pole ($s^I_{d,1} = -7.6 + 126.7\,i\ s^{-1}$) has the smallest real part (in module). Since the reciprocal of the smallest real part essentially determines the relevant network's relaxation times, the characteristic time of 'forgetting' of the transient network response should be about $\tau_T = 1/|\mathrm{Re}(s^I_{d,1})| \simeq 131$ ms, such that by $3\tau_T \simeq 400$ ms the network should have lost memory of the initial conditions and relax to the stationary state. In Fig. 2 we show the response of the network to the sharp onset of an external excitatory input affecting all neurons; it is seen that the relaxation times well match the theoretical prediction. It is also seen that both the excitatory and the inhibitory populations show an oscillatory approach to the stationary state. This is consistent with the theoretical prediction of oscillatory transients whenever a population is in a SD regime (which is the case for the simulation shown); the frequency of the oscillation should be the reciprocal of the imaginary part of the inhibitory pole dominating the transient, $2\pi/\mathrm{Im}(s^I_{d,1}) \simeq 1/\nu^I_0 \simeq 50$ ms, which is well confirmed by simulation data.

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