Mean-field population dynamics of spiking neurons with random synaptic delays

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Abstract. We derive a dynamical equation for the spike emission rate $\nu(t)$ of a homogeneous population of Integrate-and-Fire neurons, in an “extended” mean-field approximation (i.e., taking into account both the mean and the variance of the afferent current). Conditions for stability and characteristic times of the population transient response are investigated, and both are shown to be naturally expressed in terms of single neuron current-to-rate transfer function. Finite-size effects are incorporated by a stochastic extension of the mean-field equations and the associated Fokker-Planck formalism, and their implications for the frequency response of the population activity is illustrated through the power spectral density of $\nu(t)$. The role of synaptic delays in spike transmission is studied for an arbitrary distribution of delays.

Dynamic mean-field equations for the population activity

In [7] we introduced a method to derive (via a population density approach, [6, 8]) the equatiuns which govern the time evolution of the population activity of an interacting ensemble of IF neurons in the extended mean field approximation (i.e., taking into account the instantaneous fluctuation of the afferent current). The general appraoch is also amenable to an approximate treatment, by which we could characterize the transient response and the power spectrum of the neural population in a specific context, elucidating a close and interesting relationship between the time course of the population activity $\nu(t)$ and the “transfer function” characterizing the static mean field properties of the system. The synaptic transmission delays turn out to play a major role in the above analysis.

In the present work we remove a somewhat unnatural constraint assumed in [7], that the synaptic delays are the same for all neurons, and we show how a distribution of synaptic delays can be easily embedded in the analysis, e briefly discuss some phenomenological implications.

In the diffusion approximation, the sub-threshold dynamics of the membrane depolarization $V$ of a general class of IF neurons is given by [11] $\dot{V} = f(V) + \mu(V, t) + \sigma(V, t) \Gamma(t)$, where the afferent current is described as a Gaussian white noise with mean $\mu(V, t)$ and variance $\sigma^2(V, t)$ and $f(V)$ is a leakage term.

In an ‘extended’ mean field approach [3,2] all neurons in a homogeneous population are driven by stochastic currents with the same mean and variance,
both depending on the recurrent $\nu$ and external $\nu_{\text{ext}}$ emission rates, equal for all neurons: $\mu = \mu(V, \nu, \nu_{\text{ext}})$ and $\sigma^2 = \sigma^2(V, \nu, \nu_{\text{ext}})$. In this approximation, the set of evolving $V$s is seen as a sample of independent realizations drawn from the p.d.f $p(v, t)$, governed by the Fokker-Planck equation:

$$\partial_t p(v, t) = \left[ L p(v, t) = -\partial_v \left[ f (v) + \mu(v, t) \right] + \frac{1}{2} \partial_v^2 \sigma^2(v, t) \right] p(v, t),$$

(1)

complemented by boundary conditions accounting for the realizations disappearing on the threshold and re-appearing at the reset potential $H [10, 1, 6, 4, 5]$. Since $L$ depends on $\mu$ and $\sigma^2$, it is an implicit function of the emission rate $\nu$. The latter in turn expresses the flux of realizations crossing the threshold (emitting spikes), or the fraction of neurons emitting spikes per unit time:

$$\nu(t) = -\frac{1}{2} \sigma^2 (v, t) \partial_v p(v, t)|_{v=0} .$$

(2)

It is convenient to expand $p(v, t)$ in Eqs. (1) and (2) into the complete set of eigenfunctions $\phi_n$ of $L [9, 6]$, $p(v, t) = \sum_n a_n(t) \phi_n(v, t)$. In stationary conditions $\nu$ is the inverse of the mean inter-spike interval, and it also equals the single neuron transfer function $\Phi(\mu, \sigma^2)$, given by (2), with $p(v, t) = \phi_0(v)$, the eigenfunction of $L$ with zero eigenvalue, stationary solution of Eq. (1). The time evolution of $p(v, t)$ is then described in terms of dynamical equations for the $a(t)$ (see e.g. [9, 6]); taking into account Eq. 2, allows us to write the equations governing the time evolution of $\nu(t)$ as (“emission rate equation”):

$$\begin{cases}
\dot{a}(t) = [ \Lambda (t - \delta) + C (t - \delta) \nu(t - \delta) ] a(t) + c(t - \delta) \nu(t - \delta) \\
\nu(t) = \Phi (t - \delta) + f (t - \delta) \cdot a(t)
\end{cases},$$

(3)

where $f_n$ is the contribution to the flux due to the mode $\phi_n (n \neq 0)$; $c_n = \langle \partial_v \psi_n | \phi_0 \rangle$, $C_{nm} = \langle \partial_v \psi_n | \phi_m \rangle$, $\psi_n$ are the eigenfunctions of the adjoint operator $L^+$ and $\langle ., . \rangle$ is a suitable inner product, $c_n$ and $C_{nm}$ are coupling terms, in that for uncoupled neurons $\mu$ and $\sigma$ do not depend on the recurrent frequency $\nu$, and $\partial_v \psi_n$ vanishes. $\Lambda$ is the diagonal matrix of the common eigenvalues of $L$ and $L^+$. For simplicity in Eq. 3 a single allowed synaptic delay $\delta$ appears. However, taking into account a distribution of delays is relevant both in order to relax a somewhat implausible assumption and because it might provide an effective treatment of non-instantaneous synaptic currents provoked by each spike (see [4]).

Going from a single $\delta$ to a distribution $\rho(\delta)$ amounts to substitute the convolution \( \nu(t - \delta) \rho(\delta) d\delta \) for \( \nu(t - \delta) \). We will show later the implications of a non-trivial $\rho(\delta)$.

**Stability, spectral analysis and transients**

The system (3) has fixed points $\nu_0$ given by the self-consistency equation $[3, 2]$

$a = 0$ and $\nu_0 = \Phi(\nu_0)$, whose stability we assess using a local analysis. To this end
we study the poles of the Laplace transform $\nu(s)$ of the linearized form of $\nu(t)$ in Eq. (3). The real and imaginary parts of these poles describe the characteristic times and the oscillatory properties of the collective activity $\nu(t)$. Fig. 1 shows a subset of poles of $\nu(s)$ for an inhibitory population of linear (constant leakage) IF neurons (‘LIF’) [5]. Such poles can be grouped in two classes. The first is related to the transmission delays (‘transmission poles’, circles in Fig. 1), appearing only in coupled networks, approximately given by:

$$s^{(t)}_n \simeq \frac{1}{\delta} \ln |\Phi'| + i \frac{n \pi}{\delta},$$

where $n$ is any odd (even) integer for inhibitory (excitatory) populations and $\Phi' = \partial_s \Phi|_{s = -\nu_0}$. The fixed point becomes unstable when $\text{Re}(s^{(t)}_n) > 0$, which happens exactly when

$$\Phi'(\nu_0) > 1,$$

for an excitatory population (as suggested in [3]). A sufficient condition for a weakly coupled inhibitory population to be stable is $\Phi'(\nu_0) > -1$. The “diffusion poles” $\{s^{(d)}_n\}$, (diamonds in Fig. 1) have negative real parts and do not contribute to the stability conditions. Far from instability $-1/\text{Re}(s^{(d)}_n)$ set the time scale of the transient of the network relaxation to the fixed point. For weak coupling, in a drift-dominated (supra-threshold) regime, $\{s^{(d)}_n\}$ are a perturbation of the eigenvalues $\lambda_n$ of $L$; for the longest time scale:

$$s^{(d)}_1 \simeq \lambda_1 \left( 1 + \frac{f_1 c_1}{1 - \Phi + \lambda_1 \delta} \right).$$

It is worth noting how, despite the fact that there is no obvious a priori relation between the single neuron properties and characteristic times, and the dynamics
of the collective activity, the single neuron transfer function $\Phi$ emerges in a leading role in determining both the stability of the system (via Eq. (3)), and the response times in Eq. (6).

Fig. 2, left shows how both the characteristic times of the transient population response to a stepwise change in the afferent current, and the frequency of the transient oscillations match well the predictions generated by the first diffusion poles.

The (stationary and transient) spectral content of $\nu(t)$ is embodied in the power spectrum $P(\omega)$, a proper treatment of which requires taking into account the effects of the finite number $N$ of neurons in the population under consideration. Besides incoherent fluctuations, e.g., due to quenched randomness in the neurons’ connectivity and/or external input, which are taken into account in the variance $\sigma^2$ of the afferent current entering the Fokker-Planck equation, two additional finite-size effects contribute: 1) For finite $N$, the number of spikes emitted per unit time is well described by a Poisson process with mean and variance $N\nu(t)$, such that the fraction $\nu_N$ of firing neurons is approximated by $\nu_N(t) = \nu(t) + \sqrt{\nu(t)/N \Gamma(t)}$, with $\Gamma$ a memoryless, white noise [4]. In the mean field treatment, the fluctuating $\nu_N$ enters the infinitesimal mean and variance of the afferent current, so that $\mu_N$ and $\sigma^2_N$ are now stochastic variables, thereby making the Fokker-Planck operator itself $N$-dependent and stochastic ($L_N$). 2) While the above transition $L \rightarrow L_N$ still describes the time evolution of an infinite number of realizations, though driven by $N$-dependent fluctuating currents, the finite size of the neurons sample has to be explicitly taken into account in the boundary condition expressing the conservation of the total number of realizations crossing the threshold and re-appearing at the reset potential. The latter effect was not considered in previous treatments of finite-size effects. The combined finite-$N$ effects result in the following modified FP equation

$$\partial_t p(v, t) = L_N p(v, t) + \delta(v - H) \frac{\nu(t - \tau_0)}{N} \Gamma(t - \tau_0)$$

(7)

In the framework of the local analysis, the resulting expression for the power spectral density of the population activity is:

$$P(\omega) = \frac{\left| 1 + f \cdot (i \omega \mathbf{I} - \mathbf{A})^{-1} \psi e^{-i \omega \tau_0} \right|^2}{\left| e^{i \omega \delta} - \Phi \rho(i \omega) - i f \cdot (i \omega \mathbf{I} - \mathbf{A})^{-1} c \omega \rho(i \omega) \right|^2} \frac{\nu_0}{N}.$$  

(8)

$\rho(i \omega)$ is the Fourier transform of the delays distribution centered around the mean $\delta$; the elements of $\psi$ are the eigenfunctions of $L^+$, evaluated at $H$, and the $\psi$-dependent term accounts for the finite-$N$ effects on the boundary. We remark that: 1) the asymptotic $P(\omega)$ oscillates around the white-noise flat spectrum $\nu_0/N$; 2) $P(\omega)$ has resonant peaks centered in the imaginary parts of the poles $s_n$ (the zeros of the denominator of Eq. 8). Those peaks disappear for uncoupled neurons, since in that case $\Phi' = 0 = c$; 3) the numerator of Eq. 8 modulates the spectrum and introduces peaks corresponding to the imaginary part of the
eigenvalues of $L$. We conjectured, and checked by explicit calculation for the LIF neuron, [7] that the eigenvalues are real for noise-dominated regimes, and complex conjugates pairs for drift-dominated regimes, in which case the imaginary part approximates the stationary mean emission rate $\nu_0$. The finite-$N$ effects on the boundary $H$ reflects itself in low-$\omega$ peaks in $P(\omega)$, approximately centered in multiples of $\nu_0$. We stress that the latter peaks are present even for uncoupled neurons, and they are inherently related to the finite number of neurons in the population. In Fig. 2, right, the predicted power spectrum is compared to

![Figure 2](image-url)

**Fig. 2.** Left: Transient response to a step change in the afferent current of a population of inhibitory neurons in a drift-dominated regime: Simulations vs theory. Starting from an asynchronous stationary state with $\nu = 0.2Hz$, an instantaneous increase of the external current, thereafter kept constant, drives the activity towards a new stable state with $\nu = 20Hz$. The solid black line is the mean of the activity from 10 simulations of a coupled network (5000 inhibitory LIF neurons). The thick gray line is the theoretical prediction, obtained from the first 4 pairs of diffusion poles. Right: Power spectrum of the activity of a population of inhibitory neurons in a stationary, drift-dominated regime: Simulations vs theory. The solid black line is the power spectrum from a 60 seconds simulation; the thick gray line is the theoretical prediction; the dashed line is the power spectrum of the white noise with variance $\nu_0/N$, being $\nu_0 = 20Hz$ and $N = 5000$.

the estimate from simulation for the stationary population activity of the same network as in Fig. 2, left; it is seen that the agreement is remarkably good. For the network of Fig. 2 all the spikes are propagated with the same delay; for a distribution of delays, it can be seen from Eq. 8 that 1) the absolute value of the real part of the transmission poles is an increasing function of the width $\Delta$ of $\rho(\delta)$; in other words, for given network parameters the transmission poles are driven far away from the imaginary axis as $\Delta$ increases, so that a distribution of delays improves the network stability. 2) as $\Delta$ grows, the power spectrum $P(\omega)$ gets more and more smoothed, and only the lowest frequency peaks survive for high enough $\Delta$. As an illustration, Fig. 3 reports the theoretical predictions vs simulations for a uniform $\rho(\delta)$ (left) and the smoothing effect predicted by the theory depending on the width $\Delta$ of $\rho(\delta)$ (right).
Fig. 3. Non-trivial distribution of synaptic delays. Left: $P(\omega)$ for 2000 coupled inhibitory neurons in a 4Hz noise-dominated regime: simulation (gray) vs theory (black). $\rho(\delta)$ is uniform in [8, 10]ms. Right: For the same network, the theoretical prediction for a single $\delta = 10$ms (thin line) is compared to $\rho(\delta)$ uniform in [7, 13]ms (thick line).

References