Stochastic population dynamics of spiking neurons

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1 Introduction

We will review in this chapter some developments in the use of the theory of stochastic processes and nonlinear dynamics in the study of large scale dynamical models of interacting spiking neurons. Without aiming at a full coverage of the subject, we will review the basic principles underlying the approach, provide a very brief (and probably biased) selection of key theoretical results in the last decade, and give a short account of our own work on the subject.

Undertaking to summarize the way in which a large set of interacting neurons can be usefully described as a stochastic dynamical system in biologically motivated modelling, the question present itself as to why on earth the brain, exhibiting clear signs of a very sophisticated organization in its ability to perform perception and processing tasks, should be approached as a stochastic system in the first place.

David H. Hubel remarked, in a nice popular science book [1], that anybody claiming that the brain can be described as a random system must have never looked at a real brain. In fact, the intriguing possibility, surfacing now and then in the physicists' approach to the problem, is that the structured aspects of the brain activity are, at least in part, 'mesoscopic' emerging dynamical properties of a largely 'random', underlying intricate web of neurons and synapses. With *random* we allude here to the combination of several effects, ranging from the microscopic noise in the single synaptic transmission events, to the irregular pattern of synaptic connectivity among neurons in a population. Most notably, the messages the neurons exchange in the stereotyped language of temporal sequences of spikes, bear an impressive resemblance to samples of stochastic point processes.

If, then, one takes the view that the spike traffic can be usefully described as being generated by suitable stochastic processes, how can one recover the stimulus dependence of neural activity, and whatever features attach a functional meaning to the activity of neurons? The latter should appear as modulations of the statistical quantities describing the sequences of spiking events in a population of neurons,

such that for example the time- dependent average, variance, or correlation of the spike trains will turn out to express computationally relevant features of the collective neural dynamics.

In cortical conditions, if we are allowed to speak about a 'typical' neuron, even its 'rest' condition will in fact be a very noisy one: an ongoing synaptic bombardment due to the *spontaneous activity* of the surrounding neural population. Even though the spike emission rate is very low in spontaneous activity (few spikes per second), the typical neuron has very many afferents (thousands of them), which sum to several tens of thousand spikes per second received in 'rest' conditions [2].

On top of this deafening background noise *signals* have to find their way, solving a hard signal-to-noise ratio problem, the signal being whatever modifications in the neural activities are evoked by a stimulation or a computation, and the noise is the unspecific spontaneous activity.

A possible solution is to make signals detectable by extra spikes coming to our neuron, from a *selective* set of cells belonging to a sensory or processing pathway: either making the selective emission rate overwhelmingly high, or having many neurons selectively increase their frequency. Experimental evidence on the selective emission rates in the cortex tends to rule out the former solution.

One way to implement the latter, population-rate based, solution to the signal-to-noise ratio problem relies on the ability of the noise to modulate the sensitivity of the neural system to small changes in the average rate of afferent spikes. For example, a population of neurons having their membrane potential fluctuating just below threshold ([3, 4]) due to background noise can very quickly react to small changes in the average input current. Another example, which received recently much attention, is the *stochastic resonance* scenario [5].

Another rate-based approach, pursued with considerable success in the last 15 years or so, is to have stimulus-induced, highly nonlinear dynamics of the collective state of an interacting population of neurons with intense feedback. The population can signal significant changes in the statistics of neurons' input by jumping from the global, unspecific spontaneous state to a selective collective state. Also in this case, the noise can act as a key dynamical ingredient, i.e. favoring multistability.

The above rate-based scenarios do not require, or entail, the establishment of any special temporal structure in the spike trains travelling in the population; the time dependent average emission rate, the oldest and simplest neural correlate of a stimulation in the electrophysiology tradition, would still be an essentially complete dynamical description of the system.

Alternatively, the strategy could be that of relying on the precise timing of 'meaningful' spikes, having them count because they are detected to be correlated in time, in the face of their relatively small number. Such a possibility cannot

be ruled out, and it is in fact both a major line of research, and an hotly debated issue; on the other hand, even when experimental findings seem in favor of this hypothesis, it is still to be figured out in details how the putative synchronization taking place in one set of neurons could be efficiently 'read' by downstream neural populations.

With a caution about the above mentioned, and well known, persisting debate on the ultimate nature of the neural code, in this brief review we conform to the view that the average spike emission rate in a neural population completely characterizes the system, and will set aside from now on questions of principle as for the biological motivations and plausibility, and will concentrate on techniques and results in the probabilistic approach to the dynamics of ensembles of neurons.

The main steps involved in the construction of such an approach are essentially the following: i) Define the specific single neuron model (different types of leakage currents, adaptation etc.); ii) Characterize the single neuron dynamics of the chosen model for stochastic input currents: typically (under conditions to be specified) Gaussian, white (or moderately colored) noise. The usual quantities of interest are the average *first passage time* (see later), the statistical distribution of the time intervals between spikes, the equilibrium distribution of the neuron's membrane potential, in a stable asynchronous state; iii) Extend the stochastic formulation to a population of interacting neurons, making the statistics of the neurons' afferent currents dependent on the average activity of those same neurons; iv) Work out the stationary properties in the interacting case; v) Compute the corrections due to the finite number of neurons in the population; vi) Devise approximate methods for dealing with the transient behavior of the system (at least for not too large departures from the reference stable asynchronous state, where a linearization will be possible); vii) Characterize the frequency response properties (the power spectrum) of the neural population; viii) Venture to move, with the same equipment, to the open sea of dynamical states far from stable asynchronous conditions or small perturbations of them, such as collective oscillations, *i.e.* limit cycles of the system dynamics, and other attractors.

2 Stochastic single neuron dynamics

In the following we will adopt as the single neuron model the Integrate-and-fire (IF) model, most widely used workhorse in neural modelling; we shall neither review the motivations for the IF model, nor shall we discuss its many variations and their biological plausibility (for a thorough discussion see for example [6, 7, 8]). We will simply state its general form, and most of the results that follow will in fact be quite general in the IF-neuron framework.

The generic IF neuron is point-like and described only by the value of its membrane potential V at time t:

$$\dot{V} = f(v) + I(V,t)/C \tag{1}$$

where C is the capacitance of the neuron's membrane (which will be omitted in the following, assuming the current to be measured in units of voltage/time). Two choices for the leakage term $f(v)$ correspond to the *Leaky* and *Linear* IF neuron

$$f(v) = \begin{cases} -\frac{v}{\tau} & \text{leaky IF} \\ -\beta & \text{linear IF} \end{cases} \tag{2}$$

The first is the most frequently version of IF neuron, while the second (see [9]) is a version of the Gerstein and Mandelbrot linear model [10], in which a key ingredient is introduced: a lower bound on the V values, acting as a reflecting barrier for the corresponding stochastic process (see below). The latter IF model was found to be convenient for electronic VLSI implementations [11, 12].

The driving current in Eq. (1) is

$$I(V,t) = I(t) = \sum_k J_k \delta(t - t_k - \delta_k), \tag{3}$$

and is assumed in general to be a stochastic spike train, each contributing a PSP (post-synaptic potential) J_k (the jump in V induced by a single spike). Such a random sequence of 'weighted' instantaneous events is sometimes called a *marked*, stochastic point process.

δ_k is the time that the spike emitted at time t_k takes to reach the target neuron.

The afferent spikes composing the input current $I(t)$ will be in general emitted by different neurons: the point process I will then be the superposition of several point processes.

A key assumption in what follows is that the spikes impinging on the target neuron are statistically independent events. As we will discuss later, such assumption is not as severe as it might appear at first sight, since for spike rates and connectivity typical of cortical conditions the above independence assumption will be found to be reasonable even for a network of interacting neurons with feedback.

For a broad class of point processes, a useful theorem guarantees that the superposition of such processes is a memoryless process. Indeed, a limit theorem in the theory of queues due to Çinlar [13] (also attributed to Grigelionis [14]) assures, in loose terms, the following: the composite point process obtained by a superposition of sufficiently 'sparse' and mutually independent, but otherwise arbitrary, point processes approaches a Poisson process as the number of component processes tends to infinity.

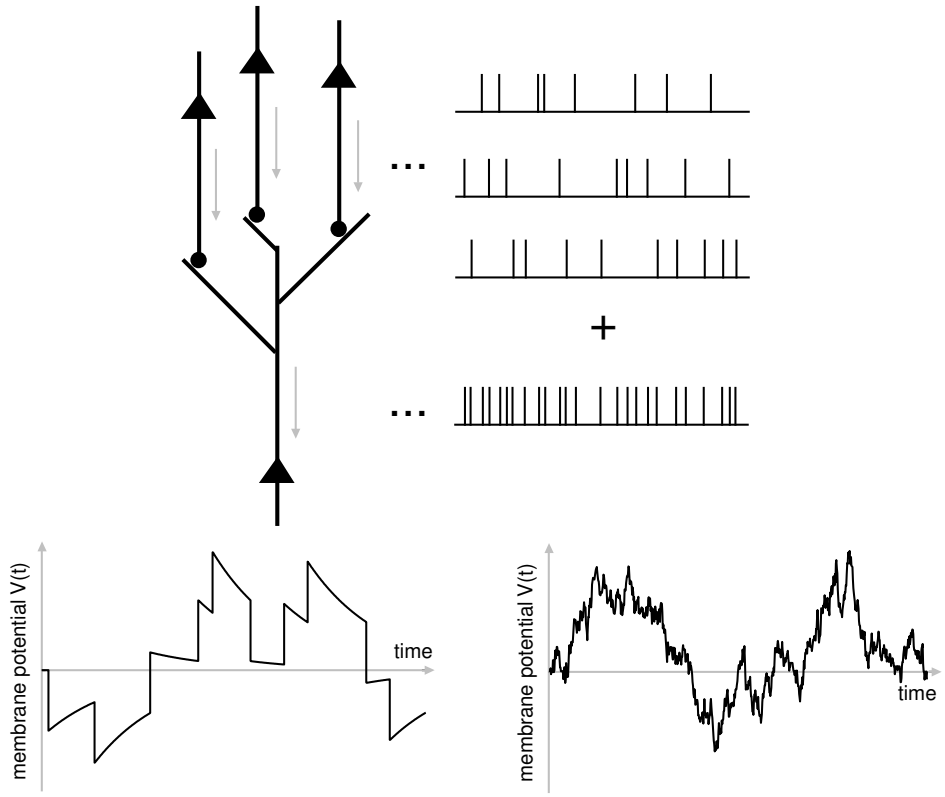

Figure 1: Schematic illustration of the neuron's afferent current as the superposition of stochastic point processes and the limit to a diffusion process. Top left diagram: schematic drawing of a target neuron with the synaptic contacts on its dendritic tree; top right: illustration of the stochastic point processes of the spikes received from each pre-synaptic neuron, and the superposition of all of them, resulting in a nearly Poisson point process; Bottom left plot: sample trajectory of the neuron's membrane potential V, with instantaneous (upward or downward) jumps upon reception of each (excitatory or inhibitory) spike, and the deterministic (exponential) decay between spikes (Stein process); Bottom right plot: sample trajectory of the neuron's membrane potential V in diffusion limit, for a leaky integrate-and-fire neuron model driven by a white noise with same infinitesimal mean and variance as the Poisson process driving the Stein process in the left plot.

Therefore, under the above hypotheses, the process $I(t)$ is a Poisson process and, as such, it is δ-correlated.

Figure 1 illustrates the neuron's input as a superposition of independent point processes: the left drawing in the top part of the figure is a schematic representation of the target neuron, with its dendritic arborization, through which afferent spikes come from different neurons; the corresponding superposition process is sketched on the right. The bottom, left panel provides an example time evolution of the membrane potential V, as it results from the instantaneous (upward and downward) jumps induced by incoming (excitatory and inhibitory) spikes, and the deterministic leakage term between spikes.

At this point Eq. 1 which governs the evolution of V is a stochastic equation (called 'Stein process' for the leaky IF neuron, see [15]), to which we can associate a probability density function (p.d.f.) $p(v,t)$ for V such that $p(v,t)dv$ is the probability that $V(t) \in (v, v+dv)$. $p(v,t)$, as usual, can be thought of as built up by collecting the infinite 'histories' (*realizations*) corresponding to the infinite instances of the random current $I(t)$: $p(v,t)dv$ is then the fraction of realizations that are in $(v, v+dv)$ at time t.

A complete description of the stochastic process V will then be provided by an equation for the time evolution of $p(v,t)$, to which we now turn. In the following we illustrate the general argument for the case of the leaky IF neuron, and we assume for notational simplicity $\delta_k = 0$, $J_k = J\,\forall k$ (only excitatory spikes) and $\tau = 1$.

In a small time interval dt the *transition probability* for v is

$$W(v, t+dt|w,t) = (1-\nu\,dt)\,\delta(v - w\,\mathrm{e}^{-dt}) + \nu\,dt\,\delta(v - (w\,\mathrm{e}^{-dt} + J)), \quad (4)$$

the sum of the (Poisson) probabilities of the alternative events of just having decayed in the absence of spikes in dt, or having undergone an instantaneous jump J because of a spike received in dt (ν is the average rate of the Poisson events).

The law of composition of probabilities, combined with the memoryless nature of the process give rise to the Chapman-Kolmogorov equation for the conditional p.d.f of v:

$$p(v, t+dt) = \int dw\, W(v, t+dt|w,t)\, p(w,t) \quad (5)$$

which, upon substitution of Eq. (4), and taking into account the properties of the δ function gives:

$$p(v, t+dt) = (1-\nu\,dt)\,\mathrm{e}^{dt} p(v\,\mathrm{e}^{dt}, t) + \nu\,dt\,\mathrm{e}^{dt}\,p(\mathrm{e}^{dt}\,(v-J), t) \quad (6)$$

from which, assuming dt small and expanding to first order accordingly, one gets:

$$\partial_t p(v,t) = \partial_v(vp(v,t)) + \nu[p(v-J,t) - p(v,t)] \quad (7)$$

This is a *master equation* expressing the continuity condition on the flow in the phase space of the system: the first term on the r.h.s. is the 'Liouville' flow related to the deterministic part of the equation of motion, while the second term accounts for the balance due to spikes bringing the realizations out and into the interval $(v, v+dv)$ (being negligible the probability of multiple jumps in dt). Eq. (7) is easily generalized to the case of excitatory and inhibitory spikes, and different allowed PSPs, and provides the sought evolution equation for the p.d.f of the membrane potential v.

2.1 The diffusion approximation

It is often convenient, and justified, to take one further step, to transform the above master equation in a *Fokker-Planck*, diffusion equation. The needed element is essentially a small-J assumption. Indeed, if one performs a Taylor expansion of Eq. (7), the following *Kramers-Moyal expansion* results:

$$\partial_t p(v,t) = \sum_{k=1}^{\infty} \frac{(-1)^k}{k!} D_k(v,t)\, \partial_v^{(k)} p(v,t) \tag{8}$$

where

$$D_k(v,t) = \lim_{dt\to 0} \frac{1}{dt} \int dx\, x^k\, p(v+x, t+dt) = \begin{cases} -v + \nu\, J & k=1 \\ \nu\, J^k & k>1 \end{cases} \tag{9}$$

are the infinitesimal moments of the membrane potential V; the p.d.f. of v constrained to be $p(v,t) = \delta(v)$. It can be proved that for the Kramers-Moyal expansion either all the terms with $k > 2$ vanish (in which case we have the *diffusion* process we will be interested in) or all the terms are non-zero (Pawula Theorem).

It can also be proved that, provided both excitatory and inhibitory spikes are present in the input spike train, a limit process can defined, with the spike rates tending to infinity and the EPSP/IPSP (Excitatory/Inhibitory PSP) going to zero in such a way that the first two coefficients D_1 and D_2 of the Kramers-Moyal expansion are non-zero and all the other vanish.

The limit process turns the marked point process $I(t)$ into a Gaussian white noise with infinitesimal mean μ (the drift) and variance σ^2 given by D_1 and D_2: essentially, the number of impinging spikes, each one contributing a very small change in v compared to the spike emission threshold, is assumed to be large even in infinitesimal time intervals, such that the central limit theorem applies. For practical purposes, as far as the spike emission properties of the neuron are concerned, even when the limit procedure leading to the diffusion approximation cannot be rigorously carried out, the same approximation still provides a good description of the neuron's stochastic dynamics (see later).

The above diffusion approximation gives rise to the Fokker-Planck equation:

$$\partial_t\, p(v,t) = L\, p(v,t), \tag{10}$$

where L is the Fokker-Planck operator

$$L(v,t) = -\partial_v(-v + \mu(v,t)) + \frac{1}{2}\,\partial_v^2\sigma^2(v,t)\,, \tag{11}$$

generating the evolution of the p.d.f. of V, which now follows, instead of Eq. (1), the the well known *Langevin equation* for the brownian motion (a sample trajectory is illustrated in the bottom right panel in Fig.1):

$$\dot{V} = f(V) + \mu(V,t) + \sigma^2(V,t)\,\Gamma(t) \tag{12}$$

where $\Gamma(t)$ is a δ-correlated white noise with zero mean and unitary variance.

Eq. (10) can be written as a continuity equation for the probability flow

$$\partial_t p(v,t) = -\partial_v S_p(v,t) \tag{13}$$

where

$$S_p(v,t) = (-v + \mu(v,t))\, p(v,t) - \frac{1}{2}\,\partial_v[\sigma^2(v,t)p(v,t)]\,. \tag{14}$$

The Fokker-Planck equation must be complemented by the appropriate boundary conditions. For the IF neuron, the boundary conditions have to account for the emission of the spike (which is not generated by the dynamics Eq. (12) of the system, but introduced as an *ad hoc* condition), the possibility of a lower bound $v_{\min}$ on the v values, and the enforcement of the normalization of the p.d.f $p(v,t)$.

The first condition is expressed by the fact that any realization of the stochastic process $v(t)$ which happens to reach the threshold θ for spike emission 'disappears', i.e. θ acts as an *absorbing barrier*. The spike emission rate, the fraction of realizations crossing θ per unit time, is

$$\nu(t) = S_p(\theta,t) = -1/2\,\sigma^2\,\partial_v\, p(v,t)|_\theta\,. \tag{15}$$

To understand which condition is appropriate for such a boundary, we remind that for a diffusion process, given any value $v(t) \in [v_{\min}, \theta]$ taken by the process at time t, v will be lifted by the gaussian noise with probability 1 in the subsequent infinitesimal time interval; the p.d.f. can nonetheless be non-zero in general because of the compensating probability flow bringing realizations in v; this compensation fails at θ, since by definition there is no flux of realizations coming from above. Therefore it must be

$$p(\theta,t) = 0\,. \tag{16}$$

Next, it must be taken into account that just after spiking v is assumed to be brought to the reset potential H (and stay there for the absolute refractory period τ_0); each realization of the process v crossing the threshold θ is rewound in H, and there is no loss of realizations, *i.e.* the p.d.f. $p(v,t)$ stays correctly normalized at any time. Therefore, a condition must hold for the conservation of the flux S_p, such that the outgoing flux in θ is exactly compensated for by a (delayed) extra influx at H:

$$\nu(t-\tau_0) = S_p(\theta, t-\tau_0) = S_p(H^+, t) - S_p(H^-, t)\,. \tag{17}$$

In what follows, we will neglect for simplicity the absolute refractory period τ_0.

Finally, a reflecting barrier $v_{\min}$ (possibly $v_{\min} \to -\infty$) implies that there is no flux crossing $v_{\min}$:

$$S_p(v_{\min}, t) = 0\,. \tag{18}$$

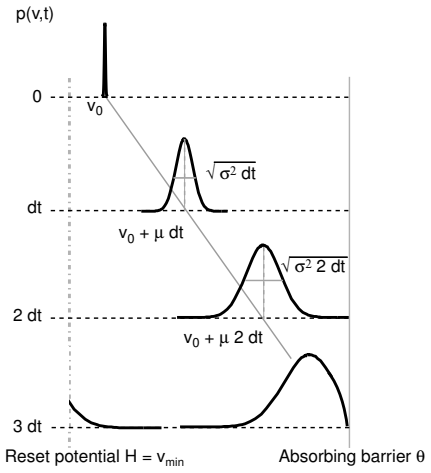

Figure 2: Evolution of the p.d.f. of V starting from a point-like initial condition for an IF neuron whose input current is a gaussian white noise with large drift and small variance.

The evolution of the $p(v,t)$ is schematically illustrated in Fig. 2: $p(v,t) = \delta(v - v_0)$. $H = v_{\min}$ in this case. The gaussian input current, with positive drift, drives $p(v,t)$ towards θ, while spreading it; the center of the gaussian and its standard deviation evolve as $v_0 + \mu\, t$ and $\sqrt{\sigma^2\, t}$, respectively. When the bulk of $p(v,t)$ reaches θ, the p.d.f. is in general much deformed with respect to a gaussian, to fulfil the boundary condition $p(\theta,t) = 0$ (its slope in θ determines the

instantaneous spike emission rate according to Eq. (15)). The outgoing flux $\nu(t)$ re-enters H; the process continues, tending to the equilibrium distribution, solution of $\partial_t\, p(v,t) = 0$. $p(v,t)$ vanishes for $v < v_{\min}$ because of the reflecting barrier condition.

2.2 Statistical properties of the neuron's firing

2.2.1 The ISI distribution

Assuming $V(0) = H$ as the initial condition for the evolution of the membrane potential for a generic IF neuron, we can establish a link between the spike emission rate $\nu(t)$ and the p.d.f. $g(t)$ of the Inter-Spike Intervals (ISI) in stationary conditions ($\partial_t\, \mu = 0$ and $\partial_t\, \sigma^2 = 0$); the latter is the same as the p.d.f. of the time needed to the membrane potential to cross for the first time the emission threshold starting from H (the *first-passage time* [16]).

$$\Pr\{\text{ISI} \in (t, t+dt)\} \equiv g(t)\, dt \tag{19}$$

Following standard steps in the theory of renewal point processes [17] we define the probability $g_n(t)\, dt$ that the n-th spike is emitted in $(t, t+dt)$, after one is emitted at $t = 0$. Under the renewal hypothesis, that the intervals are statistically independent, the following convolution recursion rule holds:

$$\begin{aligned} g_1(t) &= g(t) \\ g_n(t) &= \int_0^t g(t-t')\, g_{n-1}(t')\, dt \end{aligned}$$

From the $g_n(t)$ one can derive the *renewal intensity function*, which is nothing but the spike emission rate:

$$\begin{aligned} \nu(t) &\equiv \sum_{n=1}^{\infty} g_n(t) \\ &= g(t) + \sum_{n=2}^{\infty} \int_0^t g_{n-1}(t')\, g(t-t')\, dt \\ &= g(t) + \int_0^t \sum_{n=1}^{\infty} g_n(t-\tau)\, g(\tau)\, d\tau \end{aligned} \tag{20}$$

$$\nu(t) = g(t) + \int_0^t \nu(t-\tau)\, g(\tau)\, d\tau \tag{21}$$

The Laplace transform $\nu(s)$ of $\nu(t)$ has then a simple form:

$$\nu(s) = \frac{g(s)}{1 - g(s)} \tag{22}$$

where $g(s)$ is the Laplace transform of the ISI's p.d.f.. Conversely if we know $\nu(t)$ it is possible to compute $g(t)$ inverting its Laplace transform:

$$g(s) = \frac{\nu(s)}{1 + \nu(s)}\,. \tag{23}$$

For a renewal point process it can be shown that the auto-correlation function of the spike train is equivalent for positive time lag to $\nu(t)$.

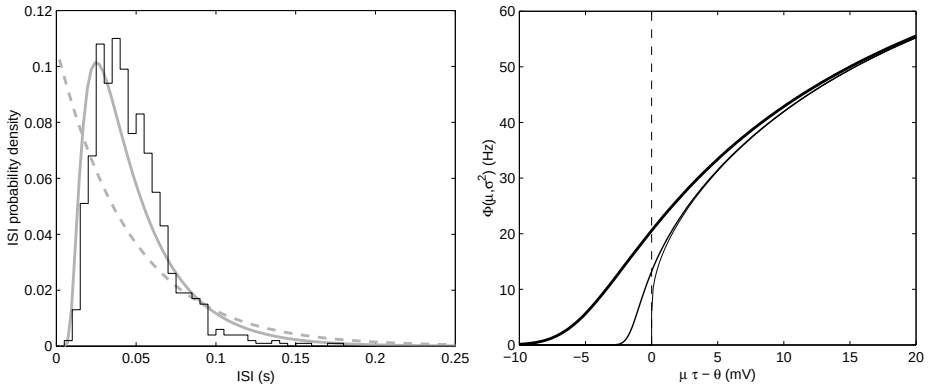

Figure 3: ISI distribution and current-to-rate transfer function for the IF neuron. In the left panel we illustrate the distribution of the ISI for an IF operating in a regime dominated by fluctuations. Solid line: histogram of the ISI sampled from a simulation; dotted line: theoretical prediction for the ISI distribution (numerical inversion of the Laplace transform of the ISI distribution of Eq. (23); dotted line: the ISI exponential distribution for a Poisson process with the same mean emission rate. In the right panel the current-to-rate transfer function $\Phi(\mu, \sigma^2)$ *vs* μ is plotted for different values of σ^2. The thicker the line, the higher σ^2.

Starting from this it is straightforward to derive an expression for the asymptotic emission frequency of the neuron $\lim_{t\to\infty} \nu(t) \equiv \nu_0$. Expanding in Taylor's series $g(s)$ it can be written:

$$g(s) = g(0) + s\, g'(0) + s^2 \sum_{n=2}^{\infty} \frac{g^{(n)}(0)}{n!} s^{n-2} \tag{24}$$

and since $g(s) = \int_0^\infty g(t)\ \exp(-s\,t)\,dt$ is also the moment generating function

$$g^{(n)}(0) = \langle(-t)^n\rangle = \int_0^\infty (-t)^n\, g(t)\, dt \tag{25}$$

we have

$$\begin{aligned} g(0) &= 1 \\ g'(0) &= -\langle t\rangle \end{aligned}$$

and

$$\nu(s) = \frac{g(s)}{s\left(\langle t\rangle - \sum_{n=1}^\infty \frac{\left\langle(-t)^{(n+1)}\right\rangle}{(n+1)!}s^n\right)} \tag{26}$$

from which we can clearly distinguish a pole at $s = 0$ representing a constant non vanishing contribution to the emission rate $\nu(t)$, corresponding to ν_0. Inverting the Laplace transform, for a time long enough to forget the initial condition ($V(0) = H$) one has

$$\nu_0 = \frac{g(0)}{\langle t\rangle} = \frac{1}{\langle t\rangle} \tag{27}$$

which is the well known relationship between the asymptotic mean emission frequency ν_0 and the average first-passage time $\langle t\rangle$.

2.2.2 The current-to-rate transfer function $\Phi(\mu, \sigma^2)$

It is relevant to derive the above asymptotic mean emission rate as a function of the parameters defining the afferent current, μ and σ^2: *the current-to-rate transfer function* $\Phi(\mu, \sigma^2)$. To accomplish this we follow [9]. Starting from the expression for the p.d.f. of v in stationary conditions ($p_0(v)$)

$$-\partial_v[(f(v) + \mu(v))\, p_0(v)] + \frac{1}{2}\,\partial_v^2[\sigma^2(v)\, p_0(v)] = 0 \tag{28}$$

and imposing the boundary conditions for the flux conservation, and for the absorbing and reflecting barriers, we can express $p_0(v)$ as a function of μ and σ^2 and the unknown asymptotic emission frequency ν_0, which can be explicitly computed by imposing the normalization condition and solving the integral equation

$$1 = \int_{v_{min}}^\theta p_0(v, \nu_0, \mu, \sigma^2)\, dv \tag{29}$$

such that

$$\nu_0 = \Phi(\mu, \sigma^2)\,. \tag{30}$$

It is important to fully realize, however, that such expected equivalence between single neuron properties like $\Phi(\mu, \sigma^2) = 1/\langle t \rangle$ and the asymptotic emission rate ν_0 derived from a population density approach, holds because of course the statistics in the 'time' and the one in the 'population' are equivalent in stationary conditions, a kind of ergodic statement. On the other hand, in non stationary conditions the population approach provides a very general framework, in which the dynamics can be accurately described on arbitrarily small time scales, which the time-based approach does not allow.

3 The population dynamics

Starting from the reduction of the neural dynamics to a stochastic process discussed in the previous section, we completely characterize the system behavior through a population formulation in which neurons loose their identity, and everything is described in terms of a probability flow. The shift in perspective is in several ways analogous to the one leading the physicist from the description of the random velocity of a molecule in a gas, alternating constancy between collisions and sharp changes upon hitting other molecules or a wall, and the formulation of the problem in terms of the probability density function of the velocity at time t, defined over the relevant statistical ensemble.

Much as in the case of the molecules in a gas, all neurons having $V \in (v, v + dv)$ at time t are indistinguishable; at time $t + dt$ they will be " anonymously" propagated by the Chapman-Kolmogorov kernel. Indeed, the totally "anonymous" character of the re-distribution of the realizations is a consequence of the memoryless nature of the driving noise.

Bringing the above treatment into the domain of interacting populations of IF neurons involves incorporating into the formalism the recurrent part of the neurons' afferent current due to the activity of all the neurons in the population. To the extent that one can still consider in the coupled network the firing of different neurons as independent events (see also the discussion at the beginning of Section3.3), the afferent current will still be described as a Gaussian white noise[1]. The radically new ingredient is that, as we will see in the present Section, the infinitesimal moments of the afferent current now recursively depend on the average emission rate they

[1]We remark that the diffusion approximation, and the Fokker-Planck formalism, can be extended to non-instantaneous synaptic currents, in which case the diffusion problem gets an additional dimension, and the noise driving the dynamics of v is no longer white. This colored noise case has been treated under some approximations in [18, 19] at the level of the single neuron dynamics, and the frequency response of the neuron have been shown to strongly depend on the memory of the driving noise.

determine, and then $\nu(t)$ becomes the only variable characterizing the state of the system and the effective vehicle of interaction among neurons in the population.

3.1 The mean-field approximation

Different neurons can have afferent currents with different statistics. Furthermore the single neuron features, like the leakage term, may differ from cell to cell. In what follows we will consider "homogenous" populations of neurons: Cells in the same population have the same single neuron properties, and share the same connectivity (average number of synaptic connections). Under such conditions, in the limit of a large number N of neurons, the moments of the afferent current to a neuron are the same for all the cells [2], and are expressed as functions of $\nu(t)$ [4], now interpreted as the emission rate of pre-synaptic neurons

$$\begin{array}{rcl} \mu(v,t) & = & \mu[v,\nu(t)] \\ \sigma^2(v,t) & = & \sigma^2[v,\nu(t)] \end{array} .$$

To the extent that the neuron is a typical member of its population, its instantaneous emission rate will be the same (up to delays) as the ν driving it, and closing this loop makes the Fokker-Planck equation (10) non-linear, because the infinitesimal moments depend themselves on the emission rate $\nu(t)$ and therefore on the system state, so that $L = L(p)$.

The assumption that the probability of firing $\nu(t)\,dt$ in the interval $(t, t+dt)$ is the same for all the neurons in the population is known as the *mean-field approximation*. It allows to completely describe the population dynamics following the only observable $\nu(t)$. Indeed, as we will see in what follows, the evolution equation for ν, at least in the linear approximation, can be expressed in such a way that the $p(v,t)$ no longer appears. It is then tempting to speculate that, even in the general non linear case, since the time evolution of $p(v,t)$ is ultimately determined by ν through μ and σ^2, the dynamics of the probability flow ν is in fact a complete description of the dynamics of $p(v,t)$ (once the initial conditions are given). The seemingly non recoverable loss of information which takes place when reducing the motion of $p(v,t)$ to that of $\nu(t)$ could be avoided because of the peculiar dependence of the Fokker-Planck equation on the ν itself; a related concept will be touched upon in Section 3.3, where we emphasize that different 'histories' $V(t)$ in the ensemble described by $p(v,t)$ only communicate to each other via spikes.

3.2 The "emission rate" equation

We now compute the dynamical equation for the emission rate $\nu(t)$ following [20] through an eigenfunction expansion of the p.d.f. of V.

The Fokker-Planck operator (11) has a set of eigenfunctions and associated eigenvalues:

$$L\,|\phi_n\rangle = \lambda_n(t)\,|\phi_n\rangle\,. \tag{31}$$

Defining the inner product $\langle\psi|\phi\rangle = \int_{v_{\min}}^{\theta} \psi(v,t)\phi(v,t)\,dv$, the adjoint operator L^+ ($\langle\psi|L\,\phi\rangle = \langle L^+\,\psi|\phi\rangle$) has eigenfunctions $|\psi_m\rangle$ and eigenvalues $\tilde{\lambda}_m$, that are in general different from those of L, because L is not Hermitian. In the above expressions the time dependence is implicitly due to the time dependence of μ and σ^2. Assuming ϕ_n as a complete set of eigenfunctions, the boundary conditions (16), (17) and (18) must be satisfied by each $\phi_n(v,t)$, and they determine also the boundary conditions for ψ, and the expression for L^+ [21, 22].

The following conditions on the eigenfunctions ψ_n of L^+ result:

$$\begin{aligned}
\psi_n(\theta,t)\,S_{\phi_n}(\theta,t) &= \psi_n(H,t)\,S_{\phi_n}(\theta,t-\tau_0)\\
\partial_v\psi_n(v_{\min},t) &= 0\\
\partial_v\psi_n(H^+,t) &= \partial_v\psi_n(H^-,t),
\end{aligned}$$

assuming ψ_n and ϕ_n to be continuous functions in the interval $(v_{\min},\theta)$.

The adjoint operator is then given by

$$L^+(v,t) = [f(v)+\mu(v,t)]\,\partial_v\, + \frac{1}{2}\sigma^2(v,t)\partial_v^2\,, \tag{32}$$

which is the evolution operator for the backward Kolmogorov equation, completely equivalent to Eq. (10).

The L^+ definition implies that eigenfunctions with different eigenvalues are orthogonal; for the completeness assumption ($\mathbf{I} = \sum_n |\phi_n\rangle\langle\psi_n|$), L and L^+ have the same eigenvalues ($\lambda_m = \tilde{\lambda}_m$), and with an appropriate normalization the two set of eigenfunctions are biorthonormal: $\langle\psi_n|\phi_m\rangle = \delta_{nm}$.

$p(v,t)$ can then be expressed as

$$|p\rangle = \sum_n a_n|\phi_n\rangle, \tag{33}$$

where $a_n = \langle\psi_n|p\rangle$ are the time dependent coefficients of the modal expansion. Since p is real $a_n^* = a_{-n}$.

The dynamics of the a_n can be determined directly from the Fokker-Planck equation (10) (see for instance Ref. [22])

$$\begin{aligned}
\dot{a}_n &= \langle\psi_n|\partial_t\,p\rangle + \langle\partial_t\,\psi_n|p\rangle\\
&= \langle\psi_n|L\,p\rangle + \sum_m a_m\langle\dot{\nu}\,\partial_\nu\,\psi_n|\phi_m\rangle\\
&= \langle L^+\,\psi_n|p\rangle + \dot{\nu}\,\sum_m a_m\langle\partial_\nu\,\psi_n|\phi_m\rangle
\end{aligned}$$

and then

$$\dot{a}_n = \lambda_n \, a_n + \dot{\nu} \sum_m a_m \langle \partial_\nu \, \psi_n | \phi_m \rangle \tag{34}$$

Here we have used the fact that the only time dependence of ψ is implicitly due to the moments of the current, μ and σ^2, which are in turn functions of the rate $\nu(t)$ (in other words, external input is assumed to be stationary). If several populations are present, their emission rates will contribute to $\partial_t \, \psi$ (see [20]), including external neurons, and $\langle \partial_\nu \, \psi_n | \phi_m \rangle$ should be regarded as a *population coupling term*; it vanishes if ν does not enter the afferent current.

The formalism can be extended to time-dependent external input, which would give rise to additional time derivatives and coupling terms. When a first order perturbative approach is viable, the transfer function of the system can be computed, and its the frequency response to time-varying inputs can be characterized (in [19] the single neuron case with oscillatory input is treated).

We also mention, but do not discuss, that the formalism can be extended to several interacting populations [20]. See also [23, 24, 4, 25, 8, 6] for other discussions of the multi-population case in the framework of the diffusion approach.

Closing the above mentioned loop which defines the mean field approximation, implies writing $\nu(t)$ for given $p(v,t)$. From Eqs. (15) and (33) one gets:

$$\nu = \left. -\frac{1}{2} \sum_n a_n \, \sigma^2(v,t) \, \partial_v \, \phi_n(v,t) \right|_{v=\theta} \tag{35}$$

Equations (34) and (35) describe completely the dynamics of the neural population and provide an effective way to reduce the dimensionality of the problem because, as we will see later, a finite (and small) number of as is often enough for an adequate description of the time evolution of ν.

The following remarks provide a simplification. It can be proved that only the stationary mode contributes to the normalization condition for $p(v,t)$, from which it follows that $a_0 = 1$ at all times. Since $\psi_0 = 1$, the coupling term $\langle \partial_\nu \, \psi_0 | \phi_m \rangle = 0$. Furthermore the flux due to the stationary mode ϕ_0 is the current-to-rate transfer function $\Phi(\mu, \sigma^2)$:

$$\Phi(\nu) = \Phi(\mu(v,\nu), \sigma^2(v,\nu)) = \left. -\frac{1}{2} \, \partial_v \, \sigma^2(v,t) \, \phi_0(v,t) \right|_{v=\theta} .$$

The (non linear) *emission rate equation* system can be written in matrix form as:

$$\left\{ \begin{array}{rcl} \dot{\vec{a}} & = & (\mathbf{\Lambda} + \mathbf{C} \, \dot{\nu}) \, \vec{a} + \vec{c} \, \dot{\nu} \\ \nu & = & \Phi + \vec{f} \cdot \vec{a} \end{array} \right. , \tag{36}$$

where $\vec{a}$ is the vector of the modal expansion coefficients with $n \neq 0$; the elements of $\vec{f}$ are the fluxes across the absorbing barrier for the non-stationary modes[2]

$$f_n = -\frac{1}{2}\partial_v \sigma^2(v,t)\,\phi_n(v,t)\Big|_{v=\theta} \quad \forall n \neq 0, \tag{37}$$

the elements of $\vec{c}$ are the coupling terms between the n-th mode and the stationary one

$$c_n = \langle \partial_\nu \psi_n | \phi_0 \rangle \quad \forall n \neq 0,$$

while $\mathbf{C}$ is the matrix of the coupling terms between the non-stationary modes

$$C_{nm} = \langle \partial_\nu \psi_n | \phi_m \rangle \quad \forall n, m \neq 0.$$

$\mathbf{\Lambda}$ is a diagonal matrix whose elements are the eigenvalues of L

$$\Lambda_{nm} = \lambda_n\,\delta_{nm} \quad \forall n, m \neq 0.$$

Such matrices and vectors depend on time through $\nu(t)$. If spikes are transmitted with delay δ, the afferent currents depend on the delayed activity $\nu(t-\delta)$ [20].

We remark in passing that δ is meant here and in the following as an *effective* delay, integrating the time needed for the spike to propagate, and characteristic times of the synaptic currents induced by the spike (see also [26]).

A non-stationary $\nu(t)$ embodies the changes in time of the average statistical properties of the neurons' afferent current, and can correlate the activities of two given neurons; this should be regarded as a "trivial" correlation due to the input part of the current that neurons have in common (self-consistently taken into account in the above mean field treatment). This does not imply a breakdown of the *independence* hypothesis which is at the heart of the formalism. The collective activity is still adequately described by a renewal, non-stationary Poisson process completely determined by ν: Neurons are independent, conditionally to the average emission rate.

3.3 The "finite-size" effects

The incoherent fluctuations accounted for by σ^2 are ultimately the expression of the independence of neurons' firing; either because of a sparse connectivity, such that neurons share a negligible portion of common input, and/or because of the quenched randomness which even for high connectivity arises from a variability of synaptic efficacies, the fluctuations sensed by any given neuron add incoherently.

[2]Not to be individually considered as probability currents, since the ϕ_ns are not individually probability density functions.

Besides those fluctuations, the finite number of neurons N determines, as we discuss below, both a stochastic modulation of the moments μ and σ^2 (thus providing a kind of second level of stochastic description of the system), and an additional correction due to finite sampling.

The first finite-N effect arises as a coherent modulation of the stochastic afferent current: the number of spikes emitted in a time interval dt by the network is a Poisson variable with mean and variance $N\nu(t)dt$, as observed in Refs. [26, 27]. The estimate of $\nu(t)$ is then a stochastic process $\nu_N(t)$, well described in the limit of large $N\nu$ by

$$\nu_N(t) = \nu(t) + \eta(t) = \nu(t) + \sqrt{\frac{\nu(t)}{N}}\,\Gamma(t)\,, \tag{38}$$

where $\Gamma(t)$ is a white noise as in the Langevin equation (12), and $\nu(t)$ is the firing probability per unite time in the infinite network.

Since the recurrent part of the afferent current to a neuron is proportional to ν_N, i.e. the global population activity, such finite-N fluctuations are coherently felt by all neurons in the network: The, now stochastic, moments $\mu(v, \nu_N(t))$ and $\sigma^2(v, \nu_N(t))$ of the afferent current, all experience the same fluctuation, since they are driven by the collective activity ν_N. This approach leads then to a " stochastic version" of the Fokker-Planck operator L, L_N, and consequently of the equation (10). Stochasticity disappears in the limit $N \to \infty$ because

$$\lim_{N\to\infty} \nu_N(t) = \nu(t).$$

Besides the above modulation of μ and σ^2, finite-N effects show up as a stochastic component of the boundary conditions. This is in fact merely a reflection of a trivial fact, i.e. that sampling the p.d.f. $p(v,t)$ through a finite sample involves fluctuations in the counting process; this has non-trivial consequences on the formulation of the finite-N emission rate equation.

For the sake of the argument, let us think about a fully 'finite-N' treatment of the problem. This would trivially require the solution of the N Langevin equations for the N neurons; the resulting ν_N is, as we remarked, a fluctuating quantity. A random instantaneous excess/defect of ν_N with respect to the infinite-N counterpart ν would, via the boundary condition requiring each history to be rewound in H after the spike, produce an excess/defect in the flow entering H, with respect to the infinite-N S_p.

While the implementation of such a program would be cumbersome, a hint at a simplification is provided by the trivial observation that neurons only talk to each other through spikes: the membrane potentials of different neurons never interact

directly in the considered models. This suggests that, for the purpose of the dynamical description of the emission rate $\nu_N(t)$, the above finite-N effects could be incorporated by a suitable effective fluctuation in the boundary condition enforcing the conservation of the probability flux in the Fokker-Planck equation for the infinite- N p.d.f. $p(v,t)$. N-dependent fluctuations in S_p emulate the effects of the above random excess/defects through a singular source/sink term in the probability flow in H:

$$\begin{aligned} \partial_t\, p(v,t) &= L_N\, p(v,t) + \delta(v-H)\, [\nu_N(t) - \nu(t)] \\ &= L_N\, p(v,t) + \delta(v-H) \sqrt{\frac{\nu(t)}{N}}\, \Gamma(t) \end{aligned} \tag{39}$$

This equation, together with Eq. (38), describes the dynamics of a population of neurons for finite N, from which one can derive the finite- N emission rate equation analogous to Eq. (36).

In summary, the interplay between the two levels of description, ν_N and ν, can be viewed as follows: For finite N each V still evolves, as already remarked, according to the Langevin equation (12), since to a very good approximation its afferent current is a δ-correlated Gaussian process; so, the purely diffusive part of the collective dynamics is still captured by the Fokker-Planck equation for p, the evolution equation for an infinite ensemble of neurons. Then, the finite N is taken into account on the boundaries (i.e., upon spikes emission), which in a sense make a finite subset of the infinite number of neurons "real".

As a complete set over which to expand the above stochastic Fokker-Planck equation we still take the eigenfunctions of L_N, with their eigenvalues, which are now stochastic, explicit functions of ν_N. The use of this stochastic moving basis leads to the following expression for the emission rate equation:

$$\left\{ \begin{array}{rcl} \dot{\vec{a}} &=& (\mathbf{\Lambda} + \mathbf{C}\, \dot{\nu}_N)\, \vec{a} + \vec{c}\, \dot{\nu}_N + \vec{\psi}\, \sqrt{\nu/N}\, \Gamma \\ \nu &=& \Phi + \vec{f} \cdot \vec{a} \\ \nu_N &=& \nu + \sqrt{\nu/N}\, \Gamma \end{array} \right. , \tag{40}$$

where the elements of $\vec{\psi}$ are the non-stationary eigenfunctions of the adjoint operator L_N^+, evaluated at the reset potential, $\psi_n(H,t)$. For simplicity we omitted the dependence on time, which is the same as in the Eq. (15). It should be noted that the above stochastic emission rate equation exhibits a complicated dependence on the finite-size noise, being $\mathbf{\Lambda}$, $\mathbf{C}$ and $\vec{c}$ all functions of ν_N: This is the expression of the noisy nature of the operator L_N in this context.

The above fluctuations act as an ongoing series of instantaneous endogenous perturbations, and as such they probe the characteristic times of the system. This

will show up very clearly in the study of the finite-N power spectral density of the collective activity, as we will see later.

3.4 "Noise-dominated" and "Drift-dominated" regimes

We now discuss the phenomenological implications of two quite distinct dynamical regimes for a system of IF neurons: a quasi deterministic one, in which fluctuations are a small correction, and one in which the dynamics is dominated by fluctuations.

When the mean driving force alone is not enough to make V cross the threshold θ, so that a positive diffusion term is necessary to have the emission of a spike, the neurons are evolving in a *noise-dominated, sub-threshold* regime, whereas when the emission of an action potential can occur also in the absence of noisy afferent currents, and the neurons are in a *drift-dominated, supra-threshold* regime of activity [4, 3, 9, 26, 23]. We remark in particular that for the IF neuron with constant leakage, the linear neuron introduced in [10], the noise-dominated regimes requires a reflecting barrier, preventing V from drifting towards $-\infty$ whatever the noise; the linear model endowed with a reflecting barrier was first studied in [9].

Fig. 4 illustrates how the activity regime determines the statistical properties of the single neuron spike train. Left panels: irregular firing (high coefficient of variation, the ratio $CV = \sigma_{\rm ISI}/\mu_{\rm ISI}$ between the standard deviation and the mean of the inter-spike intervals) corresponds to a noise-dominated regime, while regular spike trains (right panels) are related to a drift-dominated regime. Such a spread in the coefficient of variation of inter-spike intervals of the single neuron does not spoil the hypothesis of the theory, as long as the independence of the firing of different neurons holds [9, 23], which is reasonable in biologically plausible conditions [28].

From Fig. 4 it is also apparent how much the dynamics of $p(v,t)$, and then of $\nu(t)$, differs in this two regimes: For the noise-dominated case (left) the $p(v,t)$ spreads rapidly, as in a diffusive medium, and V stays most of the time well below the emission threshold, due to the strong negative deterministic force; consequently $\nu(t)$ smoothly approach the asymptotic stationary emission rate ν_0. In the drift-dominated regime (right), because of the small fluctuations in the afferent current, more time is required to spread the $p(v,t)$, and at the beginning the neurons approach coherently the emission threshold, driven by the dominating positive deterministic force. The transient of $\nu(t)$ is then characterized by neurons emitting spikes almost synchronously, until the noise of $I(t)$ completely erases the memory of the initial conditions.

The very simple case shown in Fig. 4 is that of an infinite population of non-interacting neurons. Since μ and σ^2 do not depend on ν, $\partial_\nu \psi_n = 0$ and the coupling terms vanish ($\mathbf{C} = 0$ and $\vec{c} = 0$). The emission rate equation (36) has

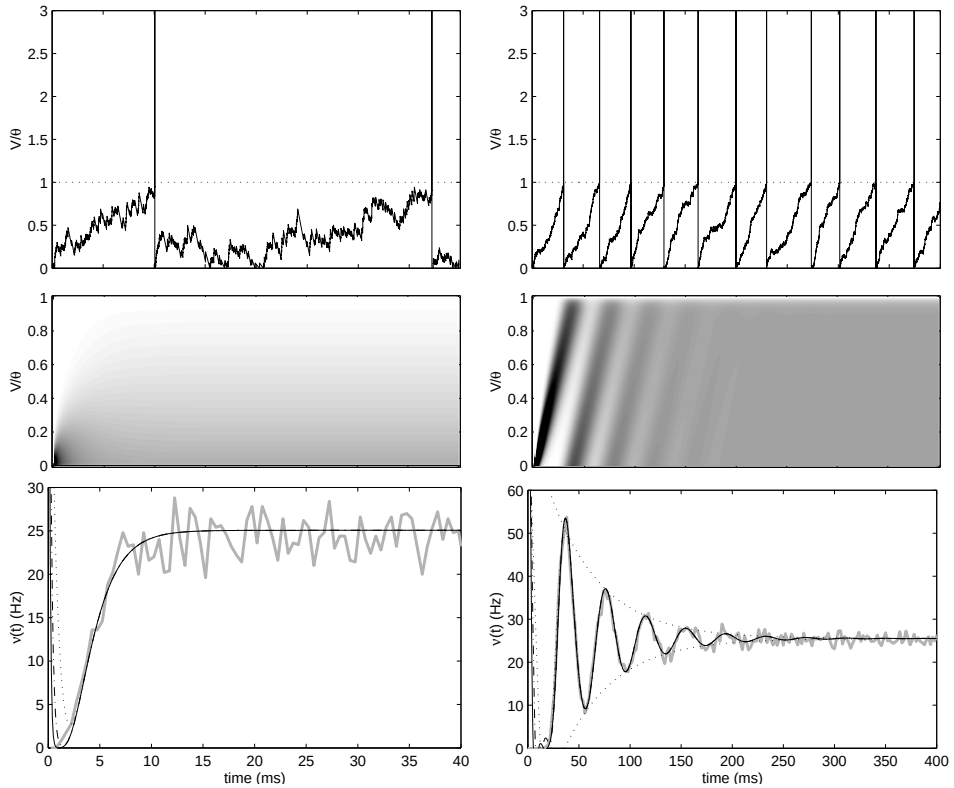

Figure 4: Transient behavior of a population of uncoupled neurons at noise-dominated (left) and drift-dominated (right) regime: At $t = 0$ all the neurons starts with $V(0) = v_{\min} = 0$. Upper plots: The membrane potential *versus* time of a neurons in the population. Middle plots: p.d.f. $p(v,t)$ of V, darker regions correspond to high density of realizations. Lower plots: emission rate $\nu(t)$ *versus* time, thick gray lines data from simulations, thin black curves the emission rate dynamics predicted by Eq. (41) when only the first one (dotted), two (dash – dotted) or three (solid) couples of modes are taken into account.

now an explicit solution

$$\nu(t) = \Phi(\mu(t), \sigma^2(t)) + \vec{f}(t) \cdot e^{\int_0^t \mathbf{\Lambda}(t')\, dt'} \vec{a}(0).$$

If the afferent current is stationary, the eigenvalues, the flux vector and the current-to-rate transfer function are constants and the emission rate is

$$\nu(t) = \Phi(\mu, \sigma^2) + \sum_n f_n\, a_n(0)\, e^{\lambda_n\, t}. \tag{41}$$

so that the spectrum of L determines directly the characteristic times of the population dynamics (the same result for uncoupled neurons was derived in [29]). As $t \to \infty$, $\nu \to \Phi(\mu, \sigma^2)$, consistently with a negative real part of the eigenvalues.

For Re $\lambda_n < 0$, after a time greater then $1/\min_n |\text{Re } \lambda_n|$, initial conditions and transients are forgotten, and the stationary population activity is the same as the static emission rate $\Phi(\mu, \sigma^2)$ of the single neuron. A hierarchy of characteristic times ($1/|\text{Re } \lambda_n|$) emerges (see [20] for linear IF neurons and [30, 31, 29] for leaky IF neurons in drift-dominated regimes) such that for non stationary conditions, only a small number of modes are required in order to have a good approximation to the time evolution of the emission rate $\nu(t)$ [22, 29, 20].

Fig. 4 also shows the theoretical prediction from Eq. (41) of $\nu(t)$ superimposed to simulation results: It is apparent that even in the severe condition of a transient due to a sudden stepwise external stimulation, the $\nu(t)$ is well described by the theory, and only a small number of mode (3 pairs of eigenfunctions in the case shown) are needed in order to accurately reproduce the emission rate from few millisecond after the stimulation onset.

Starting from this general features of the IF neurons we conjecture in [20] that the eigenvalues of L are real for noise-dominated regimes and complex conjugates for drift-dominated regimes. The conjecture was confirmed by explicit calculation in the case of the linear IF neurons.

When the interaction is turned on the "population characteristic times" are obviously a complex mixture of single neuron properties and the properties of the collective activity.

3.5 Interacting neurons

Starting from the formulation of the population dynamics via an emission rate equation, in what follows we characterize the behavior of an ensemble of homogeneous interacting IF neurons. We remark that, while the mean-field description of networks of interacting spiking neurons, in the absence of fluctuations, is a long studied subject, the study of the mean-field including fluctuations (sometimes termed *extended mean-field*) is relatively recent (see [6] for a review of results).

3.5.1 Asynchronous states and their properties

The population activity is in an "asynchronous" state when, in the limit $N \to \infty$, the neurons fire at a constant rates: $\nu(t) = \nu_0$. An example of such a state is the one asymptotically approached in Fig. 4 after a time long enough to forget the initial conditions.

From the point of view of the nonlinear dynamics, asynchronous states are fixed points of the autonomous system (15):

$$\left\{ \begin{array}{rcl} \vec{a} & = & 0 \\ \nu & = & \Phi(\nu) \end{array} \right. . \tag{42}$$

This is the self-consistency equation introduced in Ref. [2], and used in the context of a mean field treatment in Ref. [4]. As expected, the condition $\vec{a} = 0$ implies that the p.d.f. of V at the fixed point is the stationary mode ($p(v) = \phi_0(v)$).

With a time dependent perturbation approach we can study the local stability of the fixed points, their nature and the relaxation times to approach them. We start setting $\vec{a} = \varepsilon\,\vec{a}_1 + \varepsilon^2\,\vec{a}_2 + \ldots$ and $\nu = \nu_0 + \varepsilon\,\nu_1 + \varepsilon^2\,\nu_2 + \ldots$, where ν_0 is the solution of the self-consistency equation (42), and ε is the size of the perturbation from the fixed point.

Expanding the emission rate equation around $(\vec{a} = 0, \nu_0)$, to first order in ε we obtain

$$\left\{ \begin{array}{rcl} \dot{\vec{a}}_1(t) & = & \mathbf{\Lambda}(\nu_0)\,\vec{a}_1(t) + \vec{c}(\nu_0)\,\dot{\nu}_1(t-\delta) \\ \nu_1(t) & = & \Phi'(\nu_0)\,\nu_1(t-\delta) + \vec{f}(\nu_0)\cdot\vec{a}_1(t) \end{array} \right. , \tag{43}$$

where we have taken into account a delay δ in the spike transmission. From this system of ordinary differential equations with constant coefficients the Laplace transform of the perturbation $\nu_1(s) = \int_0^\infty \nu(t)\,\mathrm{e}^{-s\,t}\,dt$ can be computed [20]. The stability conditions and the characteristic times of the transient dynamics are in principle derived by standard methods, by calculating the poles of $\nu_1(s)$, the zeros of its denominator, satisfying the equation

$$(e^{s\,\delta} - \Phi') - \vec{f}\cdot(s\,\mathbf{I} - \mathbf{\Lambda})^{-1}\,\vec{c}\,s = 0\,. \tag{44}$$

where $(s\,\mathbf{I} - \mathbf{\Lambda})^{-1}$ is a diagonal matrix with elements $1/(s - \lambda_n)$.

To characterize the poles we resort to approximations; we will see in the following two subsections how two kinds of small-coupling approximation allow us to characterize two sets of poles of $\nu_1(s)$, which expose very different dynamical features.

Local stability The asynchronous state $\nu(t) = \nu_0$ is stable if all the poles s_n of $\nu_1(s)$ have a negative real part. To evaluate the stability conditions, we first look, in the limit of small coupling $\vec{c}$, for poles on the imaginary axes which, if they exist, separate the region of stability from that of instability. A crude, small coupling approximation to Eq. (44) (zeroth order in $\vec{c}$) is

$$e^{s\,\delta} = \Phi'\,, \tag{45}$$

Its solutions are quite different depending on the excitatory or inhibitory nature of the neurons. For a population of excitatory neurons Φ is a monotonically increasing function of ν, $\Phi'(\nu) > 0$. In the case $\Phi'(\nu_0) = 1$ it is easy to see that $s = 0$ is a real pole of $\nu_1(s)$. This is an exact solution also of the Eq. (44) and determines a transition from stable to unstable steady states for the system because when

$$\Phi'(\nu_0) > 1 \tag{46}$$

the real pole becomes positive and ν_0 is an unstable state. We can get an insight into the dependence of the real part of the pole on Φ' from the solutions of (45):

$$s_n^{(0)} = (\ln \Phi' + i\, 2\, n\, \pi)/\delta \tag{47}$$

for excitatory neurons and

$$s_n^{(0)} = (\ln |\Phi'| + i\, (2\, n - 1)\pi)/\delta \tag{48}$$

for inhibitory neurons, whose transfer function Φ decreases when inhibitory recurrent activity increases ($\Phi' < 0$). n runs over the integers. Better approximations for s_n can be obtained considering the first-order perturbation in $\vec{c}$ [20]. At first order one finds that for an inhibitory network an asynchronous state is stable when

$$\Phi' \gtrsim -1\,. \tag{49}$$

Note that in the zeroth-order perturbation the couplings still appear through the slope of the transfer function Φ': the stronger the coupling the higher the sensitivity of the system to a fluctuation of the collective activity. It turns out that the poles s_n move towards the imaginary axis for increasing $|\Phi'|$. So, not surprisingly, increasing the intensity of the recurrent coupling brings the network towards instability.

A noteworthy implication of the above stability analysis is that a *single neuron* feature, the slope of Φ, determines in general the stability of the fixed point ν_0 for a *population* of neurons.

The infinite set of poles responsible for the stability of the system is due to the presence of the effective delay δ: we therefore call them *transmission poles* $s_n^{(t)}$. For a system close enough to the stability boundary, activity at very high frequencies of order $1/\delta$ can arise. Transmission poles disappear for uncoupled neurons.

It is important to realize that the above oscillations are a genuine network property, not needing the single neurons to fire regularly at the frequency of the oscillation (see also [26] and the discussion in Section 3.5.2).

This set of of poles was first observed in a mean-field approach not taking into account fluctuations in the afferent current in Ref. [24], where they are termed the *gross structure* of the spectrum.

Relaxation times For uncoupled neurons the poles of $\nu_1(s)$ are the eigenvalues of the Fokker-Planck operator. This suggests a guess for finding other sets of poles as small-coupling perturbations of the λ_n. For a population of neurons in a drift-dominated regime (complex conjugates eigenvalues) a first order approximation in $\vec{c}$ for the first pair of eigenvalues gives [20]:

$$s_1 = \lambda_1 \left(1 + \frac{f_1 \, c_1}{1 - \Phi' + \lambda_1 \, \delta} \right), \tag{50}$$

and $s_{-1} = s_1^*$.

For the excitatory population ($\Phi' > 0$), from Eq. (50) it can be seen that when, starting from an uncoupled network, the recurrent excitatory coupling increases, the numerator of the fraction in the parentheses increases, and the denominator decreases as $\Phi' \to 1$. Therefore $\tau_1 \equiv -1/\mathrm{Re}(s_1)$, the longest characteristic time of the system, becomes small, so that the system reaches more quickly the steady state. A new family of poles can then be obtained by repeating the procedure for all the eigenvalues. The new poles have an opposite dependence on the intensity of the coupling with respect to the transmission poles $s_n^{(t)}$: A stronger interaction moves them away from the imaginary axis; this, combined with the fact that the eigenvalues (zeroth order approximation to the new poles) always have a negative real part, tells that they are never responsible for the instability of the asynchronous states.

Since the latter poles s_n are intimately related to the pure "free" diffusion process, directly related to the eigenvalues of the Fokker-Planck operator, we term them *diffusion* poles $s_n^{(d)}$.

Far from the stability boundary, $s_n^{(d)}$ have real parts smaller in module than those of the transmission poles, so that they are, in the situations of interest, responsible for the characteristic time of the approach to an asynchronous state ν_0.

In Fig. 5 we illustrate the time course of the population mean activity $\nu(t)$ for a network of inhibitory neurons which, starting from an asynchronous state of very low activity, undergoes a sudden increase of the excitatory external afferent input and relaxes to a new stable asynchronous state of higher activity. The black line shows $\nu(t)$ from a simulation (see the Caption for details); the gray line is the theoretical prediction resulting from keeping only four pairs of terms in the spectral expansion (which in particular implies four pairs of diffusion poles, corresponding to the four pairs of eigenvalues).

In view of the potential of the theory for a computationally inexpensive description of the system, it is important to notice that the apparent good agreement between theory and simulation is obtained with a very small number of terms.

In the low noise limit the diffusion poles can be associated with the characteristic times and resonant frequencies observed in previous works using a mean-field

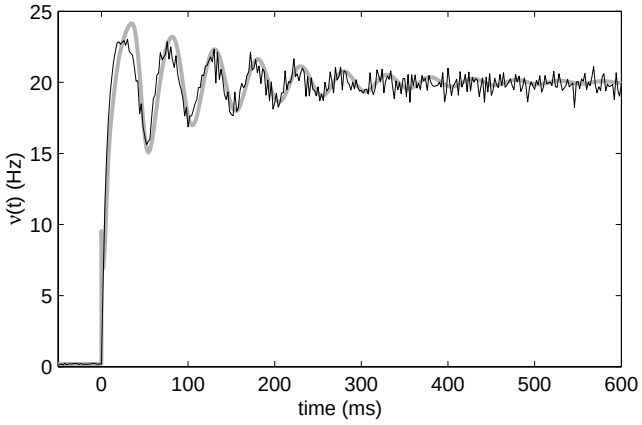

Figure 5: Transient response to a step change in the external emission rate of a population of inhibitory neurons in a drift-dominated regime: Simulations *vs* theory. For $t < 0$ the network is in an asynchronous stationary state with mean emission rate $\nu = 0.2Hz$. At $t = 0$ an instantaneous increase of the rate of external neurons, 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 linear IF neurons). The thick gray line is the theoretical prediction, obtained from the first 4 pairs of diffusion poles (adapted from [20]).

approach with deterministic afferent current [30, 24, 31, 27], and called in [24] the *fine structure* of the spectrum. The variance of the recurrent afferent current, taken into account in the present theory, dramatically affects the behavior of the system, as it was recognized in Ref. [31] and [29] for the case of external noise.

Even if the resonant response due to the diffusion poles is never enough to challenge the network local stability, for suitable (large) delays a 'coupling' between the transmission and the diffusion poles emerges which facilitates the ignition of an unstable regime (driven anyway by the transmission poles) at frequencies around multiples of ν_0 [32].

Power spectrum of the collective activity It turns out that, as we will see shortly, the above discussion of the characteristic times of the neural population is not affected by finite size effects. In general, though, the dynamical properties of the system are sensitive to finite-N corrections, and it is appropriate at this point to briefly sketch how the emission rate equations are affected by the finiteness of N.

For finite N asynchronous states are represented by a distribution of emission rates around the mean field fixed point ν_0. The local analysis leading to the emission rate equation Eq. (43) can be applied in the same way to this case.

We assume N to be large enough, for given ν_0, that the fluctuations $\eta(t)$ of $\vec{a}_1$ induced by the finite-size, of amplitude $\sqrt{\nu(t)/N} = \sqrt{(\nu_0 + \nu_1(t) + \ldots)/N}$, are small enough in order not to spoil the linear approximation.

Retaining only the leading order in $\eta(t)$,

$$\eta_0(t) = \sqrt{\frac{\nu_0}{N}}\,\Gamma(t)$$

we will then write

$$\left\{ \begin{array}{rcl} \dot{\vec{a}}_1(t) & = & \mathbf{\Lambda}\,\vec{a}_1(t) + \vec{c}\,\dot{\nu}_1(t-\delta) + \vec{\psi}\,\eta_0(t) \\ \nu_1(t) & = & \Phi'\,\nu_1(t-\delta) + \vec{f}\cdot\vec{a}_1(t) + \eta_0(t) \end{array} \right. , \tag{51}$$

where all the time-independent terms are evaluated at $\nu(t) = \nu_0$.

As anticipated, the previously discussed questions about the stability and the transients are not altered by the finite-size effects, because the pole composition of the Laplace transform $\nu_1(s)$ of $\nu_1(t)$ is unaffected by the presence of η_0, which enters the numerator of $\nu_1(s)$.

From the above reduction of the finite size network activity to a linear stochastic system, it is of interest to compute the frequency content of the $\nu_N(t)$, i.e. its power spectral density $P(\omega)$

$P(\omega)$ is computed from the $\nu_1(s)$ (calculated for imaginary argument and neglecting the terms due to the initial conditions) and, up to an additive $\delta(\omega)$ term, is given by

$$P(\omega) = \frac{\left|1 + \vec{f}\cdot(i\,\omega\,\mathbf{I} - \mathbf{\Lambda})^{-1}\,\vec{\psi}\right|^2}{\left|(e^{i\,\omega\,\delta} - \Phi') - i\,\vec{f}\cdot(i\,\omega\,\mathbf{I} - \mathbf{\Lambda})^{-1}\,\vec{c}\,\omega\right|^2}\,\frac{\nu_0}{N}\,. \tag{52}$$

From the above equation it is natural to view $P(\omega)$ as the power spectrum of the output of a linear system which receives a white noise with power spectrum $|\eta_0(\omega)|^2 = \nu_0/N$ as input, with a transfer function $T(\omega)$:

$$P(\omega) = |T(\omega)\,\eta_0(\omega)|^2$$

Therefore, the finite-size fluctuations giving rise to the η_0-terms in Eq. (51) effectively act as an endogenous source of white noise, and as such they continuously probe the frequency response of the system even for stationary input.

$P(\omega)$ has two series of peaks: the first is centered around the imaginary part of the poles s_n, whose width is proportional to $\mathrm{Re}\,s_n$. Combined with the above

discussion of the repertoire of poles of $\nu_1(s)$, and the wide range they span in the complex plane, this tells us that in a system of coupled spiking neurons the resonant frequencies cover a range from very low ($\sim 10Hz$) to very high values of order $1/\delta$; these latter peaks have been recognized in [24, 26].

It is interesting to see from the above discussion how even the simple scenario of the linearized analysis of asynchronous states exhibits a phenomenology in which many different characteristic frequencies coexist, comparable in richness with the wide range of characteristic oscillations emerging from the experimental studies of cross-correlations.

The numerator of the first term in Eq. (52) induces the second set of peaks corresponding to the λ. So we can recognize two qualitatively different finite-N contributions to $P(\omega)$: one is related to going from L to L_N in Eq. (39) and produces the first set of peaks; it has in principle a global effect on $P(\omega)$, but it turns out to significantly affect only the high-ω part related to transmission poles.

The other finite-N contribution to $P(\omega)$ is the one determined by the fluctuations of the re-entering flux at H, and has a major effect for low-ω. This provides phenomenological evidence for the role of the latter source of finite-N noise. A dramatic manifestation of its effect is illustrated by the case of uncoupled neurons, where the coherent finite-N correction to μ and σ^2 is absent.

In fact in this case the numerator of Eq. (52) is the only element that does not vanish ($\Phi' = 0$ and $\vec{c} = 0$):

$$P(\omega) = \left| 1 + \vec{f} \cdot (i\,\omega\,\mathbf{I} - \mathbf{\Lambda})^{-1}\,\vec{\psi} \right|^2 \frac{\nu_0}{N}\,. \tag{53}$$

It provides a non-trivial contribution only at low frequency, since

$$\vec{f} \cdot (i\,\omega\,\mathbf{I} - \mathbf{\Lambda})^{-1}\,\vec{\psi} = \sum_{n \neq 0} \frac{f_n \psi_n(H)}{i\,\omega - \lambda_n}$$

tends to zero when $\omega \to \infty$, where $P(\omega)$ approaches the power spectrum of a white noise. In drift-dominated regimes resonant peaks are predicted around $\mathrm{Im}\,\lambda_n \sim n\,\nu_0$, typically at low frequency (remember we conjectured, and verified in the case of Linear IF neuron, that $\mathrm{Im}\lambda_n = 0$ for noise-dominated regimes).

Figure 6 illustrates the main feature emerging from the analysis of the power spectrum $P(\omega)$. The top panel shows the theoretical prediction and simulation data for a population of inhibitory neurons in a noise-dominated regime: the only spectral peaks are those related to the transmission poles (the delay is 2 ms in the case shown). In the drift-dominated regime (bottom panel) the spectrum gets a low-ω structure because of the λ_n contribution, which gives rise to resonances at multiples of the population emission rate. The asymptotic $P(\omega)$ is ν_0/N in both

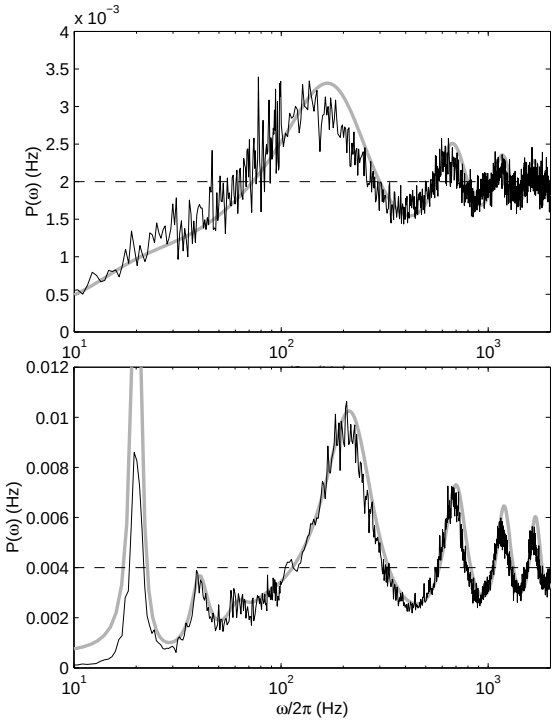

Figure 6: Power spectrum of the activity of a population of inhibitory neurons in a stationary, drift-dominated (bottom) and noise-dominate (top) regime: Simulations *vs* theory. The solid black line is the power spectrum from a 60 seconds simulation; the thick gray line is the theoretical prediction; the dashed line is the power spectrum of the white noise with variance ν_0/N, being $\nu_0 = 20Hz$ and $N = 5000$ for drift-dominated case, and $\nu_0 = 4Hz$ and $N = 2000$ for noise-dominated case (adapted from [20]).

cases, as predicted. The agreement between theory and simulation is very good, apart from a small discrepancy at very low frequencies for the drift-dominated case.

If a distribution of delays is introduced, it can be argued and verified in simulations, that the high-ω part of the spectrum gets flattened, thus affecting mostly the transmission part of the spectrum [33].

As expected, this has implications for the stability of the network, since the damping of the high frequency tail of the spectrum can be viewed as an effective increase of the real part of the high-ω, transmission poles, thereby helping keeping the system away from the stability boundary (see also Ref. [26] for a similar remark).

3.5.2 Oscillatory states

The linearized analysis illustrated so far exposed a very rich structure of the frequency response properties of the neural population, and showed how the stability properties are related to the single-neuron current-to-rate transfer function and to the distribution of spike transmission delays.

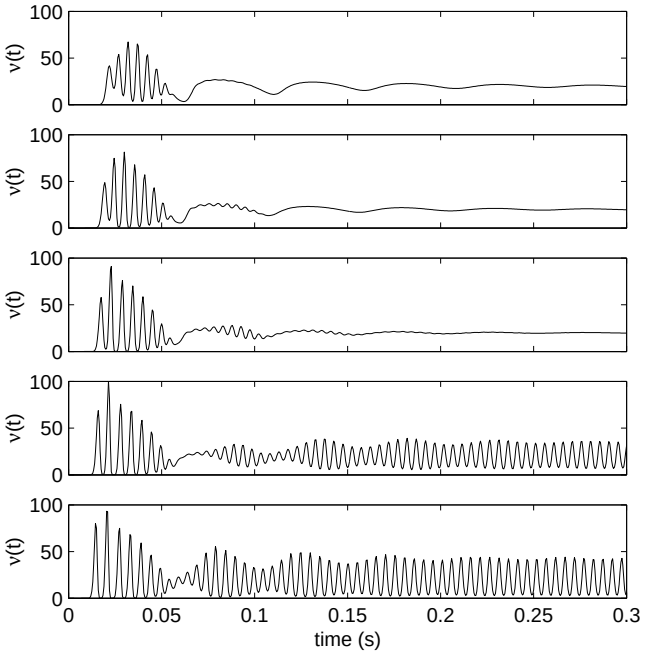

Figure 7: Time course of the population activity $\nu(t)$ of an inhibitory network, from the numerical solution of the Fokker-Planck equation. From top to bottom, the panels show, for increasing values of the recurrent inhibitory coupling, $\nu(t)$ following the onset of an external current at $t = 10$ ms, which is kept stationary for the whole interval, and such that the asymptotic emission rate is 20 Hz for all the panels.

In particular we showed that, when inhibition is present, the instability of the population can show up as an oscillatory departure from the asynchronous state. This is suggested by a nonzero imaginary part of the transmission poles guiding the instability, and indeed Fig. 7 illustrates how, for the same inhibitory network in the bottom panel of Fig. 6, a global oscillatory state sets in upon crossing the stability boundary; the oscillation has the frequency predicted from the imaginary part of the transmission pole which first develops a positive real part. From top to

bottom in the figure, we show the predicted $\nu(t)$ from the numerical integration of the Fokker-Planck equation for the same inhibitory population of neurons firing at the same mean rate, for increasing values of the synaptic coupling (and therefore of $|\Phi'|$). For the value of Φ' corresponding to the fourth panel the asynchronous state of the network is predicted by the theory to become unstable. Consistently with the theoretical prediction, as the system approaches the stability boundary the transient high frequency oscillation (related to the relevant transmission pole) require more time to be forgotten (the real part of the pole gets smaller, while the imaginary part stays essentially constant); the low frequency oscillation enveloping the high frequency one, related to the diffusion pole, is more and more quickly extinguished.

On the other hand, in general the linear analysis is unable to describe the system beyond the stability boundary, and further steps have to be taken to accomplish this. In [26, 34] this program was undertaken, for an inhibitory network, and the results were then extended, under some approximations, to an excitatory-inhibitory network in [23].

The methods involved a third order perturbation analysis, and allowed the characterization of different regimes of synchronous and asynchronous activity (see Fig. 8): *synchronous regular*, in which the system exhibits global oscillations, with underlying very regular single neuron spike trains of comparable frequency; *synchronous irregular*, with a fast oscillating collective activity, underlain by single neurons firing irregularly at a rate much lower than the frequency of the global oscillation; *asynchronous irregular*, the global asynchronous state we have mostly been dealing with in the previous Sections, built up by irregular single spike trains. As expected, transitions between the above (and others) regions in the system's 'phase diagram' are mainly guided by the balance between the excitatory and inhibitory coupling, for given neuron's parameters and transmission delays.

3.6 Scope of the diffusion-based population approach

The above discussion, and a much larger body of phenomenology, show that the repertoire of collective dynamical states accessible to a recurrent network of IF neurons is huge, and includes regimes as disparate as asynchronous states, fast or slow globally oscillatory states (with a range of possibilities as to the properties of the single neuron firing underlying them), bursting states, and a variety of point-like or chaotic attractors.

Besides, depending on the external input, or the internal dynamics, the neural population can make transitions between these states, which can be very quick or gradual.

In view of the potential of the diffusion-based population approach for describing an interesting subset of the above dynamical regimes, it is relevant to under-

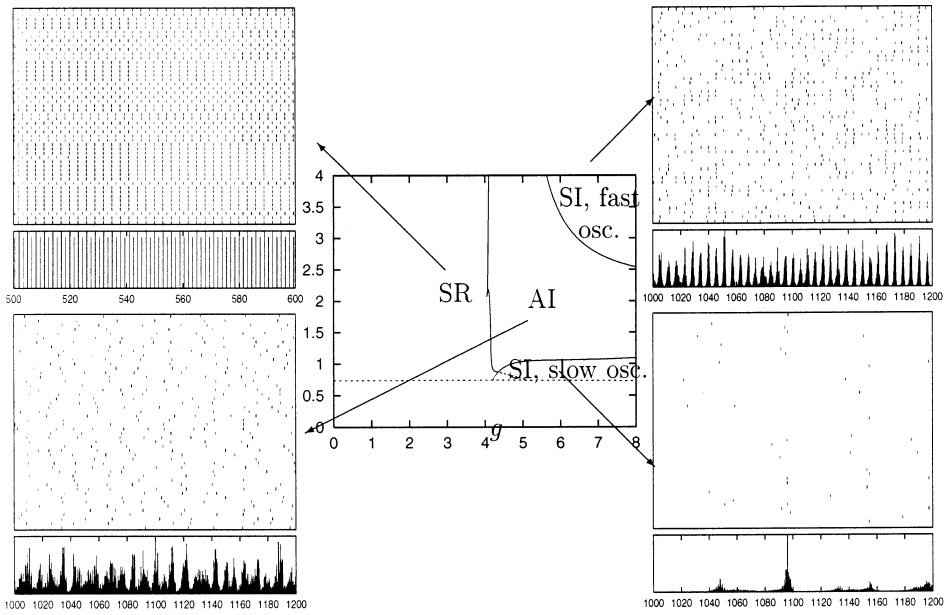

Figure 8: Illustration of the 'phase diagram' of a simulated excitatory-inhibitory network. For each of the four examples the temporal evolution of the global activity $\nu(t)$ of the system is reported, together with the firing times (raster plots) of fifty randomly chosen neurons. In the *synchronous regular* (SR) state, the network is almost fully synchronized and neurons fire regularly at high rates. In the fast oscillatory *synchronous irregular* (SI) state, there is a fast oscillation of the global activity, and neurons fire irregularly at a rate which is lower than the global frequency. In the *asynchronous irregular* (AI) state, the global activity is stationary (up to the finite-size fluctuations) and neurons fire irregularly. In the slow oscillatory *synchronous irregular* (SI) state, there is a slow oscillation of the global activity, and neurones firing irregularly at very low rates. From [35], by permission.

stand to what extent the hypotheses at the roots of the approach could be broken in different scenarios.

Essentially, the breakdown of at least three hypotheses could spoil the approach: the independence of the afferent spikes composing the input current to the neurons (conditional to the moments μ and σ^2); the smallness of each post-synaptic contribution and the large number of afferent spikes per unit time; the homogeneity of the neural population.

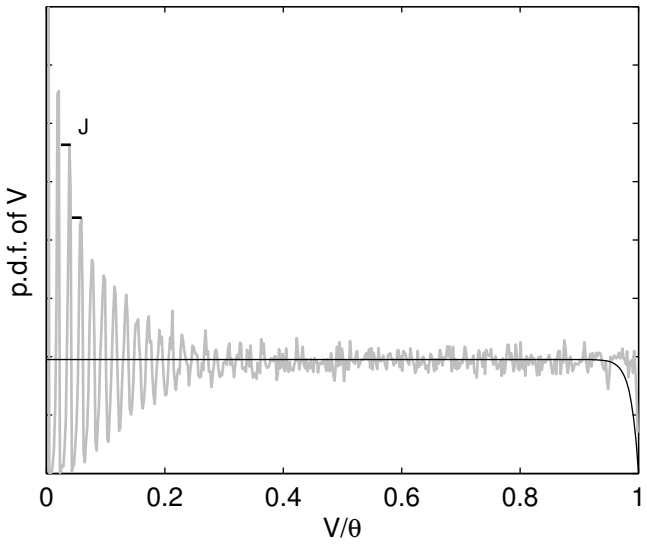

Figure 9: Comparison between the depolarization distribution predicted by the Fokker-Planck equation (solid gray line) and the one from a simulation of 100000 uncoupled Linear IF neurons subject to Poissonian excitatory input (in black). The initial condition for the depolarization distribution is concentrated around $V = \theta/2$. The picture shows the p.d.f. after $1s$; this is enough to reach equilibrium. The short black horizontal lines show the size of the EPSP of each afferent spike. The population emission rates from the Fokker-Planck equation and the simulation are in excellent agreement. Neurons are in a signal dominated regime. It is seen that near the reset potential ($V = 0$), which is also a reflecting barrier for the process, the diffusion is poorly reproduced by the simulation: the granularity determined by the finite size of the jumps J is not yet compensated for by spreading due to random noise and deterministic leakage. Yet, the good agreement in the region near threshold is enough to guarantee an excellent estimate of rates ($\sim \partial_v p(v,t)|_\theta$)

Without aiming at a thorough discussion, we just provide in the present Section some hints at a wide range of applicability of the dynamical description based on the Fokker-Planck equation.

First, it turns out that, even when the diffusion approximation is not strictly valid, and the dynamics of the membrane potential $V(t)$ is not accurately described in its full range by the Fokker-Planck equation, the dynamics of the collective spiking activity $\nu(t)$ is still captured by the approach.

We provide an example in Fig. 9, where simulation and theory are compared for a purely excitatory network (see also [36]). We mentioned above that the diffusion approximation requires both excitation and inhibition, in order to properly scale the infinitesimal moments; indeed, the p.d.f. of V is poorly reproduced by the Fokker-Planck equation far from the spike emission threshold; it also turns out that, though, the agreement is recovered near the threshold, and this is enough to guarantee a good description of the dynamics of $\nu(t)$.

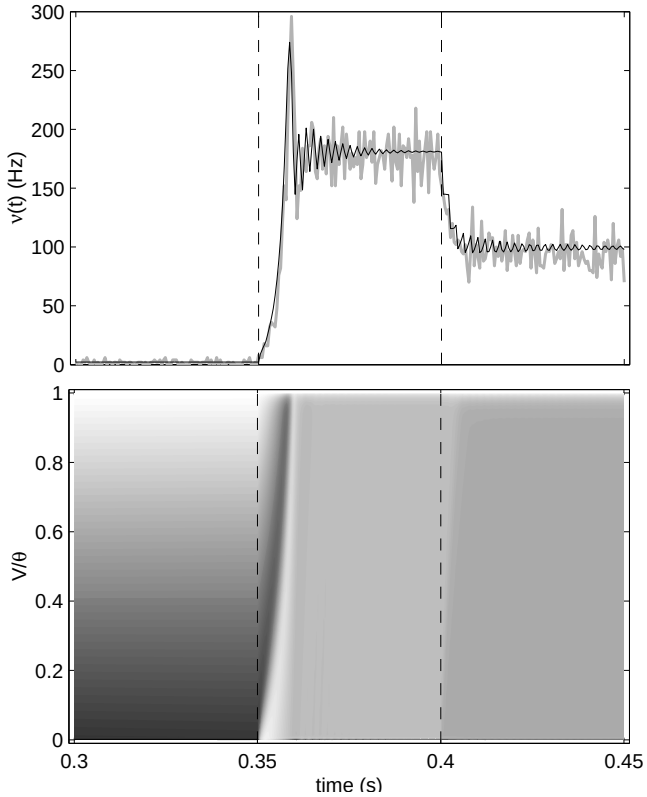

Figure 10: Simulation *vs* Fokker-Planck predictions for a recurrent network of 1000 excitatory LIF neurons. Synaptic couplings are such as to support a stable sustained activity after releasing a $50ms$ stimulation. Before stimulation the network is in a state of stable low frequency spontaneous activity (see [9]). Top: emission rate *vs* time from Fokker-Planck equation (black line) and network simulation (gray line); Bottom: Intensity plot of the depolarization p.d.f. $p(v,t)$ *vs* time.

Fig. 10 illustrates a sample case in which the Fokker-Planck equation is used to describe the time course of the collective activity for an excitatory network which starts in a in a very low-rate asynchronous state, undergoes a sudden and big jump in activity upon receiving an external stimulation, and when the stimulation is released relaxes in a *selective* asynchronous stable state with emission rate much higher than the spontaneous one. The synaptic couplings are chosen in such a way to allow such a double fixed point attractor in the system: starting from a situation in which the basin of attraction of the spontaneous activity covers the whole of the system's state space, structuring the pattern of synaptic couplings generates a new point attractor, and partitions the state space into two corresponding domains ('breaks the ergodicity' in the limit of an infinite network); the perturbation induced by the stimulation lets the system cross the barrier separating the attractors and jump into the newly formed one.

In principle, in the relatively small network of Fig. 10, the strong and sudden stimulation could, for example, challenge the hypotheses of the diffusion population approach, since in such conditions one might expect 'coherence' effects arising in the transient, and breaking the independence hypothesis; besides, for a not too large network, also the central limit theorem (the diffusion approximation) assuring that at each instant of time the afferent current is well described by a white Gaussian noise could be violated. Besides, for an accurate description of the transient also the homogeneity property should stay valid all along; this could be non trivial, since for a finite connectivity the distribution of emission rates inside the network can in principle get wider during the transient, which could break the homogeneity property.

The case in the figure shows how the Fokker-Planck can accurately capture both the stationary and the transient behaviors of the interacting network in the situation described (even though the example shown is for illustrative purposes, and of course the dangers mentioned above can in fact materialize in other cases).

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