⋆[***Estou fazendo algumas mudanças:***

- ***Nas análises e exemplos pretendo considerar só um tipo de*** Φ***, a integral de uma gaussiana com média*** V_T ***e desvio*** σ***. Isso automaticamente exclui os platôs, tanto em 1 quanto em 0, mas permite regimes quase-estacionários ou quase-periodicos que duram séculos. Ou seja, matematicamente parece que faz uma grande diferença, mas na prática não faz. Vamos ver se simplifica ou complica a discussão.***

- ***Considero inicialmente que cada neurônio tem o mesmo número*** K ***de entradas (com*** K ***grande mas independente de*** N***), vindas de neurônios aleatórios com pesos*** w_{ij} ***aleatórios. Isso significa que o peso médio de entrada de cada neurônio não é a média geral*** W***, mas um valor*** w_i ***que tem uma distribuição gaussiana estreita em torno de*** W***.***

- ***Por conta disso, a distribuição*** $P[t]$ ***não é um pente de Dirac, mas sim um pico de Dirac em*** $U_0 = V_\mathsf{R}$ ***e uma série de picos gaussianos estreitos em*** U_1***,*** U_2***, etc. Isso (pelo que entendo) torna a análise mais semelhante ao tratamento da literatura com a equação de Fokker-Planck.***

- ***Acrescentei uma seção sobre a equação de Fokker-Planck, como a entendo. (Mas não sei se entendi direito.)***

- ***Só depois disso que, na análise mean-field, considero o caso*** $K = N - 1$ ***onde essas gaussianas viram diracs. Creio que podemos argumentar que elas podem ser tratadas como diracs mesmo quando*** $K = 10^4$***. Um obstáculo a fazer essa aproximação é que as gaussianas engordam (acho) à medida que a idade aumenta. Mas creio que tendem a um limite finito por conta do*** μ***. Outro problema é que o espaço entre elas diminui com a idade, e eventalmente podem se juntar numa bolota só. Mas, em regimes interessantes, a amplitude também diminui com a idade, de modo que o problema pode não ser grave.***

- ***Acrescentei uma seção que compara o modelo GLS com o de noisy inputs. São semelhantes mas não totalmente equivalentes.***

- ***Na seção SOC introduzi dois parâmetros para cada neurônio,*** $\gamma_i[t]$ ***e*** $\delta_i[t]$***, que modulam as sinapses de entrada e de saída, respectivamente. Desconfio que qualquer um deles basta para obter SOC (se é que dá para obter). Mas a modulação das sinapses de saída parece mais natural do que as de entrada, devido ao fenômeno de depleção de vesículas. Talvez os dois tenham justificativa biológica, não sei. Aliás note que, na dinâmica de sinapses, todas as sinapses DE SAÍDA são enfraquacidas ao mesmo tempo quando um neurônio dispara. Portanto,*** δ_i ***parece ser quase uma otimização do modelo de sinapses dinâmicas, enquanto que*** γ_i ***é um conceito bem diferente.***

- ***Mudei a notação um tanto:*** W_{ij}/N ***(ou*** W_{ij}/K***) virou*** w_{ij}***. Com isso, some of fator*** $1/N$ ***na fórmula do neurônio individual, e aparece (brevemente) um fator*** K ***no meio da análise do mean-field. Mas,como o valor médio dos*** w_{ij} ***não-nulos é*** W/K***, o parâmetro*** W ***continua com mesmo significado e notação, e a maioria das fórmulas onde ele aparece não mudam.***

]

Phase transitions and self-organized criticality in networks of stochastic spiking neurons

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We report analytic and computational investigations of the behavior of neuronal networks with a general leaky stochastic neuron model, where the neuron probability of firing is given by a function $\Phi(V)$ of the neuron membrane potential V, rather than a sharp firing threshold. We find that the network can operate in various dynamic regimes (phases) depending on the parameters of the model, including the shape of the function $\Phi(V)$. $\star$[***Check:***] In particular, for certain critical parameters we find a continuous phase transition to an absorbing stationary regime, in the directed percolation universality class. In this regime we observe neuronal avalanches whose distributions of size and duration are given by power laws, as reported in real neuronal networks. We also propose the use of dynamical neuronal gains (a form of neuronal short-term plasticity), instead of dynamical synapse strengths, as a more tractable mechanism to produce self-organized criticality (SOC) and neuronal avalanches.

Contents

I. INTRODUCTION

The *integrate-and-fire* (IF) neuron model was introduced the early 20th century [1] and has been extensively used in the simulation of neuronal networks [2–7]. Basically, in IF models, the membrane potential $V(t)$ integrates synaptic or external currents up to a *firing threshold* value V_{T}. Then, a spike is generated and the membrane voltage drops to a *reset potential* V_{R}. The *leaky integrate-and-fire* (LIF) model extends the IF models with a leakage current, that causes the potential $V(t)$ to decay exponentially towards a *baseline potential* V_{B} in the absence of input signals [2, 4]. While not very accurate at the single cell level, these models can provide valuable insights into the behavior of real neuronal networks. Indeed, simulations have reproduced qualitatively some phenomena observed in real neuronal systems, such as firing avalanches [8–11] and multiple dynamical regimes [13, 14].

In the basic LIF models, the response of each neuron is deterministic. It has been claimed that a stochastic model may be more adequate for simulation purposes, since the response of a neuron in a real network is affected by random changes in the cell's electrochemical state and influence from the firing activity of nearby neurons [15]. Several authors proposed to model those random influences by adding noise current inputs to LIF models, both continuous-time [2, 3, 13, 14] and discrete-time [16–19], resulting in the *leaky stochastic integrate-and-fire* (LSIF) model. Galves and Locherbach [20–22] and Larremore *et al.* [23], in contrast, proposed to incorporate stochasticity in LIF models by assuming that the firing of a neuron is a random event, whose probability of occurrence in any time step is a *firing function* $\Phi(V)$ of membrane potential V. We will refer to this approach as the *generic leaky stochastic* (GLS) models. These GLS neurons are interesting because they are simpler to implement and analyze than the LSIF neurons.

Another known feature of biological neural networks is *plasticity*: changes in the electrochemical parameters and other properties of the brain over time scales longer than the firing of a neuron. Long term synaptic changes are widely utilized to model memory formation and learning [5, 24]. Short term changes include temporary reduction of synaptic strength after firing, due to neurotransmitter vesicles depletion or other phenomena [25]. These changes have been observed to drive the parameters of the network towards critical values, which are believed to maximize its computational efficiency [8, 26–29]; a phenomenon called *self-organized criticality* (SOC). Short-term plasticity has typically been incorporated

in models by assuming that the strength of each synapse is lowered after each firing, and then gradually recovers towards a quiescent value [9, 10, 30, 31].

In this article, we first study the dynamics of networks of GLS neurons, by a simple mean-field approximation and by computational simulations of networks with thousands of elements. In the mean-field analysis, we replace the stochastic evolution of the network by a deterministic evolution of a probability distribution according to a variant of the Fokker-Planck equation. ⋆[***Check:***] We find both continuous and discontinuous absorbing state phase transitions in our networks depending on characteristics of the firing function $\Phi(V)$. We find that, for certain firing functions and critical parameter settings, the forced firing of a single neuron causes finite avalanches of firing events, that are statistically similar to those observed in the vertebrate brains [8, 28].

Second, we present a new mechanism for short-term plasticity that is based on dynamical changes of a *gain* parameter associated to each neuron, instead of changing the individual synaptic strengths. This model has the computational advantage of needing only N state variables and equations instead of N^2 required by the synapse dynamics approach. Yet, our simulations show that this model too displays self-organization to criticality.

II. NEURON MODEL

The discrete-time neuron that we use in the rest of this paper can be viewed as a simple Euler approximation of the continuous-time GLS model described in the introduction.

The network generically consists of N neurons with membrane potential V_i ($i = 1, \ldots, N$). Each synapse transmits the signals from some *presynaptic* neuron j to some *postsynaptic* neuron i, and has a numerical attribute w_{ij}, the *synaptic strength*, which is the increment on the potential of neuron i when neuron j fires. When $w_{ij} = 0$, effectively there are no synapses from neuron j to neuron i.

We also assume that the states of all neurons are observed only at equally spaced *sampling times*, and change synchronously in the intervals between them. The time step Δ between sampling times is assumed to be small enough to exclude the possibility of a neuron firing more than once during each step. Thus, the continuously-varying membrane potential of a neuron i is modeled by a sequence of real values $V_i[t]$, indexed by the *discrete time* t, an integer that represents the sampling time $t\Delta$.

In this model, if some neuron j fires between discrete times t and $t+1$, its potential drops to V_R by time $t+1$. This event increments by w_{ij} the potential of every neuron i that does not fire in that interval. A non-firing neuron i may also accumulate an *external input stimulus* $I_i[t]$ that is added to its potential at time $t+1$. This term can be used to model sensory inputs from neurons external to the modeled network, or the effect of artificially injected current (as in certain in vitro experiments) between times t and $t+1$. Apart from these increments, the potential of a non-firing neuron decays exponentially towards a baseline potential V_B by a factor μ in $[0, 1]$, that models the effect of the leakage current. (In the continuous model, with no inputs the the potential evolves as $V'(t) = -(V - V_\mathsf{B})/\tau$, where τ is the characteristic decay time. The same rate of decay is obtained in the discrete model with $\mu = e^{-\Delta/\tau}$.) Note that V_B may be higher or lower than V_R [12]. ⋆[***Tem ref melhor?***]

In neurobiology, the potentials V_R, V_B, and V_T are customarily measured relative to the extracellular fluid, and are typically between -40 and -80 millivolts [12]. ⋆[***Verificar. Ref melhor?***] However, the zero of potential is arbitrary. For this paper, we chose to measure all potentials relative to V_B, since formulas are generally simpler if $V_\mathsf{B} = 0$.

A. The neuron evolution equations

We introduce the auxiliary quantity $X_i[t]$ which is the number of times that neuron i fired between the discrete times t and $t+1$, namely 0 or 1. We also define the *age* of a neuron i at some discrete time t as the number of time steps that have elapsed since its last firing; so that the age is r if $X_i[t-r] = 1$, and $X_i[t-k] = 0$ for all k with $0 \le k < r$.

The neuron potentials then evolve by the formulas

$$V_i[t+1] = \begin{cases} V_\mathsf{R} & \text{if } X_i[t] = 1, \\ \mu V_i[t] + (1-\mu)V_\mathsf{B} + I_i[t] + \sum_{j=1}^{N} w_{ij}\, X_j[t] & \text{if } X_i[t] = 0. \end{cases} \tag{1}$$

The parameter V_B is mathematically redundant, since the term $(1-\mu)V_\mathsf{B}$ in Eq. (1) could be included in the external input term $I_i[t]$. It is justified by reference to biology: V_B and μ are supposed to be intrinsic parameters of the neuron, while $I_i[t]$ is due to external sources and depends on the context of the analysis or experiment.

B. The firing function

Each firing indicator $X_i[t]$ is assumed to be an independent Boolean random variable whose distribution depends on the potential $V_i[t]$ at discrete time t. Namely, $X_i[t]$ is 1 with probability $\Phi(V_i[t])$, for some specified *discrete firing function* Φ [20, 21].

Considering the observed behavior of typical biological neurons, we assume that $\Phi(V)$ is a sigmoidal function, monotonically increasing from $\Phi(-\infty) = 0$ to $\Phi(+\infty) = 1$, with only one inflection point. In the numerical examples and computer simulations of this paper, we specifically assume a *Gaussian firing function*, the cumulative form of a normal distribution

$$\Phi_{\mathsf{G}}(V) \;=\; \int_{-\infty}^{V} \frac{1}{\sigma}\phi\left(\frac{V - V_{\mathsf{T}}}{\sigma}\right) dV \;=\; \frac{1}{2} + \frac{1}{2}\,\mathrm{erf}\left(\frac{V - V_{\mathsf{T}}}{\sigma\sqrt{2}}\right) \tag{2}$$

where ϕ is the Gaussian distribution function with zero mean and unit variance, $\phi(z) = (1/\sqrt{2\pi})e^{-z^2/2}$, and erf is the error function $\mathrm{erf}(z) = (2/\sqrt{\pi})\int_0^z e^{-t^2}\,dt$. See Fig. **??**.

Any sigmoidal firing function Φ, such as Φ_{G}, can also be described as a smoothed version of the shifted Heaviside step function $\Phi_{\mathsf{S}}(V) = \Theta(V - V_{\mathsf{T}})$, that is zero for $V < V_{\mathsf{T}}$ and one for $V > V_{\mathsf{T}}$. Specifically, Φ_{G} is the convolution of Φ_{S} with a Gaussian distribution with mean zero and deviation σ. The parameter σ defines the degree of smoothing, and V_{T} is the potential at which the firing probability is exactly 1/2.

C. Relationship to other models

In the limit when the firing function Φ tends to the sharp step function Φ_{S}, (namely, when $\sigma \to 0$ in Φ_{G}), the GLS model becomes identical to the discrete time version of the deterministic LIF neuron, with firing threshold V_{T}.

Applying the smooth function Φ_{G} of the GLS model to a neuron potential $V_i[t]$ is functionally equivalent to applying the sharp firing function Φ_{S} to the sum $V_i[t] = \delta_i[t]$, where $\delta_i[t]$ is an independent random variable whose density distribution is $D(\delta) = \Phi'_{\mathsf{G}}(V_{\mathsf{T}} + \delta)$. Therefore, the GLS model is quite similar to that of ??? [], which adds to the second line of Eq. (1) a random noise signal $\zeta_i[t]$ before applying the sharp firing function Φ_{S}.

There is a subtle difference, however: in ???'s model, the signals $\zeta_i[t]$ get integrated by the neuron, whereas in the GLS model the virtual noise $\delta_i[t]$ is not. Thus, in ???'s model, the effective firing function becomes smoother as the age r of the neuron increases, and

narrows again when it fires; whereas in the GLS model the firing function is independent of the neuron's age.

However, in ???'s model the past noise inputs $\zeta_i[t-k]$ get attenuated by the leak factor μ^k. If the variance of each $\zeta_i[t]$ is τ^2, the variance of the r accumulated noises $\sum_{k=0}^{r} \mu^k \zeta_i[t-k]$ is $\hat{\tau}_r^2 = \tau^2(1-\mu^{r+1})/(1-\mu)$, which is bounded by $\hat{\tau}_\infty^2 = \tau^2/(1-\mu)$. Moreover, if τ is small compared to V_T, the firing probability (in both models) is significant only after the neuron's age r has reached a certain value, so that $\hat{\tau}_r$ is already close to the limit $\hat{\tau}_\infty$. Therefore, ???'s model with noise variance τ is expected to produce results very similar to the GLS model with $\Phi = \Phi_\mathsf{G}$ and $\sigma = \hat{\tau}_\infty = \tau/\sqrt{1-\mu}$.

D. Firing rate and potential distribution

An important macroscopic attribute of the network is the *firing ratio*, the fraction $\rho[t]$ of the neurons that fired between discrete times t and $t+1$, namely:

$$\rho[t] = \frac{1}{N} \sum_{j=1}^{N} X_j[t] \,, \tag{3}$$

Another important macroscopic attribute is the *potential distribution* $P[t]$ at any discrete time t, such that $P[t](V)\,dV$ is the fraction of neurons with potential in the range $[V, V+dV]$ at time t.

Since the distribution of each variable $X_i[t]$ depends only on the potential $V_i[t]$, the fraction $\rho[t]$ can be computed from $P[t]$:

$$\rho[t] = \int_0^\infty \Phi(V) P[t](V)\, dV \,, \tag{4}$$

The neurons that fire between t and $t+1$ have their potential reset to V_R. They contribute to the distribution $P[t+1]$ a Dirac impulse at potential $V = V_\mathsf{R}$, with amplitude (integral) $\rho[t]$ given by Eq. (4). In subsequent time steps, the potentials of all neurons will evolve in response to the firings of other neurons, according to Eq. (1). This process modifies $P[t](V)$ also for $V \neq V_\mathsf{R}$.

III. ISOLATED NEURONS

$\star$[***Refazer para*** Φ ***generica:***]

A popular experiment in neurobiology consists in injecting a constant current J into an isolated neuron in vitro. In this experiment, $N = 1$ and there are no synaptic inputs. Assuming that the current is turned on at $t = 0$ when the neuron's potential is $V[0] = V_{\mathsf{B}} = 0$, the second line of Eg. e.model reduces to

$$V[t+1] \;=\; \mu V[t] + I\,, \tag{5}$$

where $I = J\Delta/C$, and C is the effective capacitance of the neuron. The potential $V[t]$ is therefore $(1-\mu^t)/(1-\mu)I$ until the first firing, and thereafter it is $U_r = \mu^r V_{\mathsf{R}} + (1-\mu^r)/(1-\mu)I$ for a neuron with age r. These potentials converge exponentially to $U_\infty = I/(1-\mu)$. Note that this limit does not depend on V_{R}.

The probability that the neuron will reach age r or greater after each firing is therefore:

$$q_r \;=\; \begin{cases} 1 & \text{if } r = 0, \\ q_{r-1}\,(1 - \Phi(U_{r-1})) & \text{if } r > 0. \end{cases} \tag{6}$$

and therefore the probability of a neuron firing again after exactly r steps is $Q_r = q_r - q_{r+1}$. This is the distribution of intervals between firings. As r increases, the distribution of spacings tends to an exponential distribution with decay ratio $1-\Phi(U_\infty) = 1-\Phi(I/(1-\mu))$ per step. That is, the firing tends to a Poisson process with rate $1/(1-\Phi(I/(1-\mu)))$. Increasing the ratio $I/(1-\mu)$ reduces the mean age when the regime becomes Poisson-like, and reduces the mean time between firings.

However, for smaller r the firing is not Poisson-like. Assuming $\Phi = \Phi_{\mathsf{G}}$, the firing probability Q_r is essentially zero until $U_r > V_{\mathsf{T}} - 6\sigma$, so that value of r is an effective lower bound to the time between firings. In particular, if $I < (1-\mu)(V_{\mathsf{T}} - 6\sigma)$ (meaning that the injected current J cannot overcome the leakage current), the neuron is essentially unreactive, since the mean time between firings would be more than a hundred million steps. On the other hand, if $I > (1-\mu)(V_{\mathsf{T}} + 3\sigma)$, there will be an effective maximum age r.

With $\Phi = \Phi_{\mathsf{G}}$, the key parameters that determine the shape of the distribution Q_r of firing intervals are the relative position of U_∞ in Φ_{G}'s effective domain, namely $A = (I/(1-\mu) - V_{\mathsf{T}})/\sigma$, and the decay rate μ. A secondary parameter is $D = -log_{1-\mu}((V_{\mathsf{R}} - V_{\mathsf{T}})/\sigma)$, that basically shifts the distribution in r without changing its shape. ⋆[***Conferir***] Figure **??** shows the distribution of firing intervals for some values of A and μ.

⋆[***Verificar e adaptar:***] Interestingly, this kind of Michaelis-Menten function is frequently used to fit the (normalized) firing response of biological neurons to constant input

currents [36, 37]. In both cases the usual firing rate can be written as $F(I) = 2\rho F_{max}$, where the maximal firing rate F_{max} is a parameter to be empirically determined. These functions $F(I)$ can be seen in (Fig. **??**).

IV. MEAN-FIELD ANALYSIS

Now we provide a *mean-field* analysis of a large network that is exact in the $N \to \infty$ limit.

A. The Fokker-Planck equation

In the mean-field approach, one abstracts from the potentials of individual neurons and works instead with the potential distribution $P[t]$, which can be considered the *stochastic state* of the network. The neuron evolution equation (1) is replaced by the Fokker-Planck equation, that describes the evolution of $P[t]$:

$$P[t+1](V) \;=\; \int_{-\infty}^{+\infty} P[t](U)\,\Lambda[t](U,V)\,dU \tag{7}$$

where $\Lambda[t](U,V)$ is the probability distribution of the potential at time $t+1$ of the neurons that, at time t, have potential U. From Eq. (1), with $V_\mathsf{B} = 0$, we can conclude that $\Lambda(U,V)$ has two components: a Dirac impulse at $V = V_\mathsf{R}$, with amplitude (integral) $\Phi(U)$, resulting from the probability of the neuron firing in that interval; and some distribution resulting from the neurons that do not fire, with integral $1 - \Phi(U)$. The latter is the distribution of values $\mu U + \sum_{j=1}^{N} w_{ij}\,X_i[t]$ when i ranges over all neurons. To determine this distribution, we must choose a specific stochastic model for the synapse strengths.

B. The K-random network model

For simplicity, in this section we assume that each neuron has a fixed number $K < N$ of input synapses and the same number of output synapses; that is, for each i there are only K input strengths w_{ik} and K output strengths w_{ki} that are non-zero. In particular, we assume that there are no self-couplings; that is, $w_{ii} = 0$ for all i. We also assume that the external inputs $I_i[t]$ are zero for all neurons and all times.

In the mean-field approximation, one disregards correlations between the activity X_j of a presynaptic neuron j and the strength w_{ij} of its synapse to neuron i. Therefore, the final term of Eq. (1) can be factored as follows:

$$\sum_{j=1}^{N} w_{ij}\, X_j[t] \;=\; \left(\sum_{j\in\mathcal{K}_i} w_{ij}\right)\left(\frac{1}{K}\sum_{j\in\mathcal{K}_i} X_j[t]\right) \;=\; W_i\,\rho_i[t]\,. \tag{8}$$

where $\mathcal{K}_i$ is the set of the K neurons that make input synapses with neuron i, W_i is the sum of the strength of those synapses, and $\rho_i[t]$ is the fraction of those K neurons that fired in the previous interval. Therefore, every neuron i that does not fire between t and $t+1$ (that is, with $X_i[t]=0$) has its potential changed by the formula:

$$V_i[t+1] \;=\; \mu V_i[t] + W_i\,\rho_i[t]\,, \tag{9}$$

We now assume that the non-zero synaptic strengths w_{ij} are drawn from a global distribution with mean W/K and standard deviation κ/K. We will call the parameter W is the *mean neuron integration gain*. It is the average increase in the potential of a neuron that would result if all its inputs fired at once.

By the law of large numbers, the total input synaptic strength W_i of each neuron will be a random variable with approximately Gaussian distribution, with mean W and variance κ^2/K. The quantity $\rho_i[t]$ will also be a random variable, with mean $\rho[t]$ and variance $\rho[t](1-\rho[t])/K$. For large enough K, we can assume that the product $W_i\,\rho_i[t]$ will also have an Gaussian-like distribution with mean $W\rho[t]$ and deviation $\lambda[t]$ such that

$$\lambda^2[t] \;=\; \frac{\rho[t]}{K}\Big(\rho[t]\kappa^2 + W^2(1-\rho[t])\Big) \tag{10}$$

Therefore, the second component of the Fokker-Planck kernel $\Lambda[t](U,V)$ can be approximated by a Gaussian with mean $U + W\,\rho[t]$, deviation $\lambda[t]$, and integral $1-\Phi(U)$. ★[***Não garanto minha álgebra!***]

C. The shape of the potential distribution

In this section we describe in more detail the nature of the distribution $P[t]$ implied by Fokker-Planck equation, after most neurons have fire at least once.

Let $S_k[t]$ be the set of neurons with age k at discrete time t. Recall that the distribution $P[t]$ had a Dirac impulse component at potential $U_0 = V_{\mathsf{R}}$ and amplitude (integral) $\eta_0[t] =$

$\rho[t-1]$, representing the set $S_0[t]$ of neurons that fired in the interval from $t-1$ to t. Some of the neurons in $S_0[t]$ neurons may fire again in the next step, with probability $\Phi(U_0)$, and remain at potential U_0. According to Eq. (9), the neurons of $S_0[0]$ that do not fire become the set $S_1[t+1]$, and contribute to the distribution $P[t+1]$ a narrow Gaussian peak with mean $U_1 = U_0 + W\rho[t]$, deviation
$lambda[t]$, and integral $\eta_1[t+1] = \eta_0[t](1 - \Phi(U_0[t]))$.

A fraction $\Phi(U_1)$ of the neurons in $S_1[t+1]$ will fire again between times $t+1$ and $t+2$; the remainder will comprise $S_2[t+2]$. Assuming that $\kappa/\sqrt{K}$ is very small compared to σ, we can assume that the distribution of potentials of $S_1[t+1]$ will continue to be Gaussian, but with its integral reduced by the factor $1 - \Phi(U_1)$, its mean shifted to $U_2 = \mu U_1 + W\rho[t+1]$, and its variance changed to $\tau_2^2 = \mu^2\tau_1^2 + \rho^2[t]\kappa^2/K$. ★[***???***] And so on. It follows that, once all neurons have fired at least once, the distribution will be approximately a superposition of Gaussian peaks with integrals $\eta_0[t], \eta_1[t], \eta_2[t], \ldots$, mean potentials $U_0[t], U_1[t], U_2[t], \ldots$, and deviations $\tau_0, \tau_1, \tau_2, \ldots$, that evolve according to the recurrences

$$U_0[t+1] \;=\; V_\mathsf{R}\,, \tag{11}$$

$$U_k[t+1] \;=\; \mu U_{k-1}[t] + W\rho[t]\,, \tag{12}$$

$$\eta_0[t+1] \;=\; \rho[t]\,, \tag{13}$$

$$\eta_k[t+1] \;=\; (1 - \Phi(U_{k-1}[t]))\,\eta_{k-1}[t]\,, \tag{14}$$

$$\tau_0[t+1] \;=\; 0\,, \tag{15}$$

$$\tau_k^2[t+1] \;=\; \mu^2\tau_{k-1}^2[t] + \lambda^2[t]\,, \tag{16}$$

for all $k \geq 1$. See Fig. **??**). ★[***Conferir a recorrência de*** τ_k***.***]

★[***Variância de*** U_0 ***deveria ser um*** τ_R ***em vez de 0. Ou seja, mesmo o pico em*** V_R ***deveria ser uma gaussiana.***]

The amplitude $\eta_k[t]$ is the fraction of neurons with "age" k at discrete time t (that is, neurons that fired between times $t-k-1$ and $t-k$, and did not fire between $t-k$ and t). The mean potential of those neurons, at time t, is $U_k[t]$. In particular, $\eta_0[t]$ is the fraction $\rho[t-1]$ of neurons that fired in the previous time step, between discrete times $t-1$ and t and $U_0[t]$ is always V_R. For this type of distribution, the integral of Eq. (4) becomes a discrete sum:

$$\rho[t] \;=\; \sum_{k=0}^{\infty} \Phi(U_k[t])\,\eta_k[t]\,. \tag{17}$$

⋆[***Completar***]

⋆[***Citar o artigo com esta análise***]

D. The Dirac train approximation

The widths $\tau_k[t]$ of the Gaussian peaks are inversely proportional to $\sqrt{K}$, and are therefore fairly narrow for the typical values of K in vertebrate brains (10^4 or more). In what follows, we will assume that the peak widths $\tau_k[t]$ are zero; that is, we approximate each peak of $P[t]$ by a Dirac impulse with the same mean and integral. While this approximation may disturb some subtle effects, it seems to still yield useful information about the behavior of the network, as confirmed by simulations with finite N and smaller K. ⋆[***Confirmar.***]

⋆[***Colocar em algum lugar:***] As observed before, we can limit our consideration to systems where $\Phi(V_{\mathsf{R}}) \ll 1$ and $\Phi(V_{\mathsf{B}}) \ll 1$.

⋆[***Dizer mais sobre*** W ***muito grande,*** $W \to 0$***, etc. Notar que se*** $\Phi(U_k[t]) = 1$***, então*** $\eta_{k+1}[t+1]$ ***será zero, etc. Dar exemplos de soluções periódicas.***]

With this approximation, the stochastic state of the network at time t can be defined as the list of the parameters of those Dirac impulses, namely $x[t] = ((\eta_0[t], U_0[t]), (\eta_1[t], U_1[t]), (\eta_2[t], U_2[t]), \ldots)$. Formulas (11–14) can be summarized by a *deterministic* (but non-linear) function F that maps states to states: $x[t+1] = F(x[t])$. This general type of recurrence is known as a *dynamic system* in mathematics, or as a *map* (specifically, a coupled maps lattice) in physics [32, 33]. Such systems have been extensively studied in chaos theory and other disciplines, and are known to exhibit many types of complex behaviors (such as fixed points, periodic and quasi-periodic sequences, and strange attractors), depending on F and on the initial conditions.

E. General considerations

⋆[***Repensar esta seção.***]

In the mean-field context, assuming $V_{\mathsf{B}} = 0$, the evolution of the network's state depends on the function Φ, the other model parameters (V_{R}, μ, K, W, and κ), and the starting state (the initial distribution of potentials $P[0]$). The following general considerations apply for any Φ of the type described above.

1. Dead regime

If $\Phi(V_\mathsf{B})$ is exactly zero, the network admits a *dead* regime, where the neuron potentials are all equal to V_B, and there is no firing. This regime is stable if $\Phi'(V_\mathsf{B})$ <???, meaning that any spontaneous firing is unlikely to cause ⋆[***???***]

In the tend to V_B, in spite of occasional spontaneous firings. In this regime, P has only one significant peak $U_0 = V_\mathsf{B}$ and $\eta_0 = 1$.

In this case, however, interesting dynamics is observed if $\Phi'(V_\mathsf{B}) > 0$ and W has a precise value W_C In that situation, a single firing creates an avalanche of firings with a characteristic distribution of size and duration. Se the companion paper [**?**].

If instead $\Phi'(V_\mathsf{B}) = 0$, and $\mu < 1$, the dead regime is attractive: starting from any state where the neuron potentials are sufficiently close to V_B, they will tend to due to the leakage current.

F. Stationary phases

In the context of mean-field analysis, a *stationary phase* is a potential distribution $P(V)$ of membrane potentials that does not change with time. In such a regime, quantities U_k and η_k do not depend on the time t. Therefore, the evolution equations (**??**–**??**) become a pair of recurrence equations:

$$\eta_0 \;=\; \rho = \sum_{k=0}^{\infty} \Phi(U_k)\eta_k \,, \tag{18}$$

$$U_0 \;=\; 0 \,, \tag{19}$$

$$\eta_k \;=\; (1 - \Phi(U_{k-1}))\, \eta_{k-1} \,, \tag{20}$$

$$U_k \;=\; \mu U_{k-1} + W\rho \,, \tag{21}$$

for all $k \geq 1$. Since these equations are homogeneous in the η_k, one needs to add the normalization condition $\sum_{k=0}^{\infty} \eta_k \;=\; 1$. The recurrences (18–21) can be solved numerically, given the values of μ and W and the firing function $\Phi(V)$ (Supplementary Fig. **??**). In some special cases, the solution has a closed analytic formula.

⋆[***Note that a stationary phase in the mean-field context is not stationaryat the individual neuron level. The neurons are constantly firing and shifting between the potentials*** U_k***. It is only the number of neurons of each age that***

is constant.]

G. Degenerate stationary periodic regimes

⋆[***Repensar esta seção. Deveria virar uma justificativa para evitar*** Φ***s com plateaus 0 ou 1, pois isso cria regimes estacionários e periódicos "não interessantes". Estes regimes não existem com*** $\Phi = \Phi_{\mathsf{G}}$***, mas em seu lugar existem transientes muito longos semelhantes a esses regimes estacionários.***]

⋆[***Mencionar as conjeturas de que regimes periódicos são guarda de memória. Será que podemos mostrar que regeneram memória?***]

Some combinations of parameters allow (or even require) uninteresting regimes. If $\Phi(V_{\mathsf{B}}) > 0$, a fixed fraction of the neurons will fire spontaneously even without any input. ⋆[***Does this converge to a steady regime, or periodic, or chaotic, or...?***]

If $\Phi(V_{\mathsf{B}}) = 0$ but $\Phi(V_{\mathsf{R}}) > 0$, there are still "uninteresting" stationary or periodic regimes where every neuron fires repeatedly in a short and repetitive or quasi-repetitive pattern, even if it does not receive any inputs. ⋆[***Example?***]

For this reason, in what follows we will assume that $Phi(V_{\mathsf{B}}) = 0$ and $\Phi(V_{\mathsf{R}}) = 0$. Even so, the network admits uninteresting regimes if W is too large. In particular, if $W \geq W_{\mathsf{B}}$ where $W_{\mathsf{B}} = 2(V_{\mathsf{S}} - \mu V_{\mathsf{R}})$, there is a stationary regime P with only two peaks, with potentials $U_0[t] = V_{\mathsf{R}}$ and $U_1[t] = U_{\mathsf{M}}$, where $U_{\mathsf{M}} = \mu V_{\mathsf{R}} + W/2$, and intensities $\eta_0 = \eta_1 = 1/2$. In this regime, half the neurons fire on alternate sampling times, and their inputs are sufficient to raise the potential of the other half to U_{M}, above the saturation limit V_{S}, causing them all to fire in turn at the next time.

If W is strictly greater than W_{B}, this stationary regime is surrounded by an infinitude of cyclic regimes with period 2, where the potential $U_1[t]$ alternates between $U_{\mathsf{M}} + \varepsilon W$ and $U_{\mathsf{M}} - \varepsilon W$, and $\eta_0[t]$ alternates between $1/2 + \varepsilon$ and $1/2 - \varepsilon$, for a sufficiently small ε. The stationary regime and these periodic regimes are marginally stable,and have no attraction basin: they are stable in the absence of external disturbances, but any sufficiently small disturbance will shift the network to a slightly different regime in that class.

More generally, for any $p \geq 2$ and suitable values of W, μ, V_{R}, V_{T}, and σ, there is a stationary state whose potential distribution has exactly p peaks, with intensities $U_k = \mu^k V_{\mathsf{R}} + (W/p)(1 - \mu^k)/(1 - \mu)$ and equal amplitudes $\eta_k = 1/p$. This regime is possible if

$\Phi(U_{p-1}) = 0$ but $\Phi(U_p) = 1$. Then, at each sampling time, the fraction $1/p$ of the neurons with potential U_p will fire and drop to U_0, while the rest moves up the ladder without firing. As in the case $p = 2$, this stationary regime is surrounded by $p - 1$ dimensions worth of cyclic regimes with period p (or sub-multiples thereof), where the potentials and fractions deviate slightly from those of the stationary state.

Note that these regimes are "uninteresting" also because they do not depend on the shape of the function Φ, since they only sample it where it is 0 or 1. Therefore, they appear also in the mean-field analysis of the deterministic (non-stochastic) LIF model, where Φ is a step function with any threshold between U_{p-1} and U_p [**?**].

⋆[***Observar que o caso normal tem*** $\Phi(V_\mathsf{R}) = 0$ ***mas*** $\Phi'(V_\mathsf{R}) = 0$***, logo não tem o comportamento crítico de monomial*** $r = 1$***.***]

⋆[***Repensar o que é o*** W ***crítico quando*** $\mu > 0$ ***e*** Φ ***é simoidal mesmo e*** $\Phi(V_\mathsf{R}) = 0$ $\Phi'(V_\mathsf{R}) = 0$ ***(o caso realista) e*** $W < W_\mathsf{B}$ ***(o caso interessante).***]

⋆[***Temos avalanches ed alguma forma?***]

V. THE MODEL WITH DYNAMIC PARAMETERS

It is known that we always can turn a model, which is critical only with a fine tuned parameters, into a model with dynamic parameters that spontaneously adjusts them toward the critical values, a phenomenon known as *self-organized criticality* (SOC) [8–10, 26, 28–31]. In this section, we modify the basic GLS model (Eq. 1) to achieve this behavior.

Several authors have successfully obtained SOC in neural networks by means of dynamically varying synaptic strengths; that is, replacing the constants w_{ij} in Eq. (1) by time-varying parameters $w_{ij}[t]$. The general idea is to reduce the strength of a synapse after the presynaptic neuron has fired, and let it slowly recover towards a higher *resting strength* in the absence of pulses [9, 10, 30, 31]. This dynamics is intended to mimic biological phenomena like the the local depletion of neurotransmitter vesicles after a synaptic discharge.

The main drawback of this approach is that it involves KN independent state variables and equations for N neurons with K input synapses each. Therefore, we propose instead to introduce two time-varying parameters for each neuron i, the *input gain* $\gamma_i[t]$ and the *output gain* $\delta_i[t]$, that modulate the strength of its input and output synapses, respectively. That

is, we replace the second line of Eq, (1) by

$$V_i[t+1] = \mu V_i[t] + (1-\mu)V_{\mathsf{B}} + I_i[t] + \gamma_i[t] \sum_{j=1}^{N} w_{ij}\, \delta_j[t]\, X_j[t] \tag{22}$$

The input gain parameters γ_i evolve according to the equation

$$\gamma_i[t+1] = \begin{cases} \gamma_{\mathsf{R}} & \text{if } X_i[t] = 1, \\ \mu_\gamma \gamma_i[t] + (1-\mu_\gamma)\gamma_{\mathsf{B}} & \text{if } X_i[t] = 0. \end{cases} \tag{23}$$

where μ_γ is related to the characteristic recovery time for the neuron's sensitivity to input signals; γ_{R} is the value that γ_i assumes immediately after the firing of neuron i; and γ_{B} is the target value for its recovery while neuron i does not fire. The output gain δ_i evolves by a similar equation

$$\delta_i[t+1] = \begin{cases} \delta_{\mathsf{R}} & \text{if } X_i[t] = 1, \\ \mu_\delta \delta_i[t] + (1-\mu_\delta)\delta_{\mathsf{B}} & \text{if } X_i[t] = 0. \end{cases} \tag{24}$$

where μ_δ, δ_{R}, and δ_{B} are analogous to μ_γ, γ_{R}, and γ_{B}, respectively. We call the list $(\gamma_{\mathsf{B}}, \gamma_{\mathsf{R}}, \mu_\gamma, \delta_{\mathsf{B}}, \delta_{\mathsf{R}}, \mu_\delta)$ the *hyperparameters* of this extended GLS model, in contrast to the fixed parameters $(V_{\mathsf{B}}, V_{\mathsf{R}}, \mu, V_{\mathsf{T}}, \sigma, N, K, W, \kappa)$.

The great advantage of this new mechanism is that we have only $2N$ new variables and evolution equations for $\gamma_i[t]$ and $\delta_i[t]$, instead of KN equations for the $w_{ij}[t]$.

We give an example of self-organization of the average gain $\gamma[t] =< \gamma_i[t] >$ towards the point $\gamma_c = 1$, (for $W_{\mathsf{C}} = 1$) starting from an average $\gamma[t=0] \neq 1$ (Fig. 1a). Of course, this can be done for any value of W since $\gamma_c(W) = 1/W$. See (Fig 1b) for the avalanche size distributions and (Fig. 1c) for data collapse.

The good thing is that this dynamics has as attractor the value W_{C} (for large N, [30, 31]) since if the network is in the active phase, $W[t]$ decreases due to the sites with $X_j[t] = 1$ and if the network is in the silent state then $W[t]$ increases due to the synaptic recovery.

VI. DISCUSSION

A. Static phase transitions

The discrete time integrate-and-fire models of Soula *et al.* [16] and Cessac [17–19] have a crucial difference to the Galves-Locherbach model [20, 21]: their source of stochasticity is

FIG. 1: a) Self-organization to criticality of the average gain $\gamma[t]$ by using dynamical neuronal gains $\gamma_i[t]$ starting from different initial conditions, with $\gamma_i(t=0) \in [0, \gamma_{max}]$ so that the average gain is $\gamma(t=0) = \gamma_{max}/2$. The horizontal dashed line is the value $\gamma_c = 1$. b) Avalanche size distribution (with exponential bins) $P(S)$ for several N. The solid line $S^{3/2}$ is a guide to the eyes. c) Data collapse of the complementary accumulated function $g = S^{1/2}C(S/N^c)$. The cutoff exponent is $c = 1/2$.

noise in the currents, not a stochastic firing function $\Phi(V)$. So, the models are different and it is not clear, from the literature, if these model equations present phase transitions as a function of W. Larremore *et al.* model [23] corresponds to the monomial case with $r = 1$, but these authors do not report any phase transition (which may be a feature produced by the inhibitory neurons in their model). Although they used $r = 1$, the ceaseless activity found by these authors is very similar to the $W_{\mathsf{C}} = 0$ case that we have obtained with $r < 1$.

The phase transitions found in this paper are of the class of absorbing state phase transitions (continuous and discontinuous) [26–28]. It seems to us that such kind of transitions are not possible with deterministic IF neurons, that present in general transitions to synchronized states or transitions between two different kinds of activity [5, 13, 14]. Perhaps, a stochastic firing function $Phi(V)$ is a necessary ingredient to this kind of transitions. If so, this is the first time that such transitions are reported in discrete-time integrate-and-fire neuronal networks.

B. Self-organized criticality

To obtain self-organized criticality by using dynamical synapses is by now a well diffused idea [9, 10, 30, 31]. This idea is plausible biologically, but is costly for simulations since we must work with a number of equations equal to the number of synapses.

In this work we propose a new SOC mechanism, based in the presence of the critical surface $\gamma_c(W, \mu) = (1 - \mu)/W$ instead of a single critical point W_{C}. The idea is to have N dynamical gains $\gamma_i[t]$ instead of the $N(N-1)$ synapses $w_{ij}[t]$. We have demonstrated by simulations that the idea seems to work very well. A mean-field calculation of this result is reserved to another paper.

This mechanism has never been examined in the literature but is plausible biologically,

being related to the well know phenomenon of *spike adaptation* [34, 35]. The neuronal gain hardly would be a fixed quantity, but is related on the somatic membrane features that could be depend on the neuron activity. Indeed, our mechanism Eq. gt says that if the neuron fires, its gain (probability of a next firing) slightly diminishes, recovering to the original value after that. This is a plausible effect.

VII. CONCLUSION AND PERSPECTIVES

In this paper we studied phase transitions in the class of stochastic neuronal networks introduced by Galves and Locherbach [20, 21]. We developed a mean-field analysis framework (the $N \to \infty$ limit) and explored some of the phase diagram by simulations. We found basically tree kinds of behavior: second order phase transition for $\Phi(V_{\mathsf{R}}) = 0$ and $\Phi'(V_{\mathsf{R}}) = 1$, first order phase transitions for $\Phi(V_{\mathsf{R}}) = \Phi'(V_{\mathsf{R}}) = 0$ and no phase transition (ceaseless dynamics for small W) for $\Phi(V_{\mathsf{R}}) = 0$, $\Phi'(V_{\mathsf{R}}) = +\infty$.

From the second order critical surface $\gamma_c(W, \mu) = (1-\mu)/W$ found in the phase diagrams, we proposed a new mechanism for self-organized criticality in neuronal networks based in dynamical gains $\gamma_i[t]$ instead of synaptic dynamics as previously done in [9, 10, 30, 31]. This new SOC mechanismo is one of the most important results of the paper.

We conclude that the stochastic neuronal networks introduced by Galves and Locherbach˜[20, 21] present very rich behavior in terms of phase transitions. The next research steps could be the study of different network topologies, different firing functions, different kinds of synapses (probabilistic, chemical, electric), the effect of inhibitory neurons [23], balanced networks [13], external inputs, learning in such networks, a more rigorous analysis of Self-organized Criticality, as done in [10, 30, 31]. We suggest that this research program with GLS neurons promise new and very interesting results.

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IX. METHODS

The phase diagrams for the mean-field model with $\mu > 0$ (Figs. ???) were obtained by simulating the evolution of the potential distribution $P[t](V)$ according to Eqs. (**??**–**??**), starting from an arbitrary initial distribution, until reaching a stable distribution. Only the first 100 peaks (U_k, η_k) were considered, since, for the given μ and Φ, there was no significant probability beyond that point. In any case, those 100 probabilities η_k were renormalized for unit sum after each time step.

The avalanche statistics were obtained by simulating the evolution of a finite network of

N neurons, with uniform synaptic strengths $w_{ij} = W$ (except $w_{ii} = 0$) and critical parameter values. Each avalanche was started with all neuron potentials $V_i[0] = V_\mathsf{R} = 0$ and forcing the firing of a single neuron by setting $X_i[0] = 1$. The network was then simulated according to Eq. (1) until all activity ceased and all potentials had decayed to such low values that further firings would not be expected for thousands of steps.

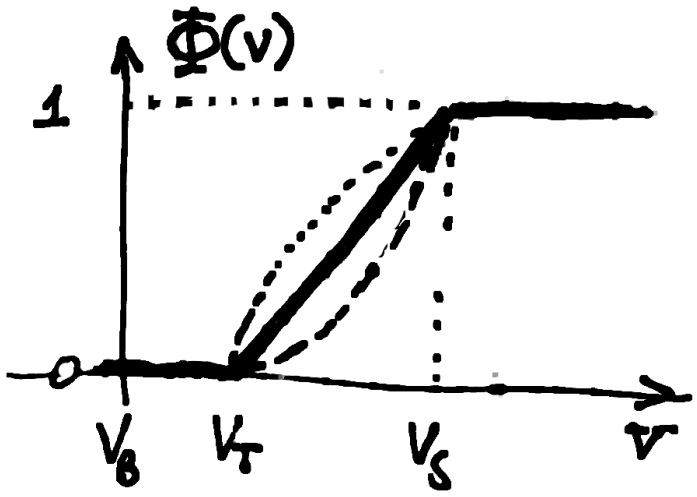

FIG. 2: SKETCH - monomial function with $V_{\mathsf{T}} > V_{\mathsf{B}}$

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