Coexistence of oscillations and neuronal avalanches in spiking neurons networks

NeuroMat
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FAPESP

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Abstract: Self-organized criticality (SOC) with conservative systems is by now a very understood subject, fully integrated to the Statistical Mechanics of non-equilibrium systems. However, for dissipative systems, the situation is not clear and several open problems remain. Models of dissipative systems (earthquakes, forest fires, neuronal avalanches) have been proposed since the 90´s with the claim that they are examples of true SOC systems. The present consensus is that they are not: even if one can propose slow drive mechanisms to counterbalance dissipation, so that the system is at least conservative in average, these systems present stochastic oscillations hovering over the critical region (this has been called self-organized quasi-criticality or SOqC). Here we explain the origin of these stochastic oscillations in networks of spiking neurons as an outcome of a different criticality scenario: instead of the presence of the standard transcritical transition point, usually found in SOC models, we have a Neimark-Sacker bifurcation from a stable spiral to an indifferent one. In the critical region, finite size noise excites and maintain the stochastic oscillations. Also, fluctuations in the presence of the silent (absorbing) state close to the critical region interrupts the oscillations generating power law avalanches and Dragon King events. By using simple Gerstner stochastic neurons, we obtain very direct and transparent mean-field results. In our model, the fundamental ingredient for the emergence of stochastic oscillations is firing rate adaptation (dynamic neuronal gains).

Our model: Stochastic discrete time spiking neurons

$i = 1, 2, \ldots, N$ neurons $\quad X_i[t] = 1$ (spike)

$V_i[t+1] = 0 \quad$ reset if $X_i[t] = 1$

$V_i[t+1] = \mu V_i[t] + I + N^{-1} \sum_j W_{ij} X_j[t] \quad$ if $X_i[t] = 0$

$\mathrm{Prob}(X_i[t+1] = 1) = \Phi(V_i[t+1])$

Φ = firing probability function

$\Phi(V) = \Gamma V / (1 + \Gamma V)$

Γ = neuronal gain

All-to-all network

Mean field approach

- Order parameter: $\rho[t] = 1/N \sum_i X_i[t]$
- MF approximation:

$1/N \sum_i W_{ij} X_i[t] = W \rho[t]$, where $W = \langle W_{ij} \rangle$

- Voltages evolve as:

$V_i[t+1] = 0 \quad$ if $X_i[t] = 1$
$V_i[t+1] = \mu V_i[t] + I + W\rho \quad$ if $X_i[t] = 0$

- $\rho[t] = \int \Phi(V)\, p_t(V)\, dV$

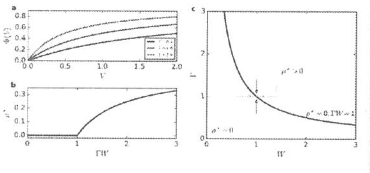
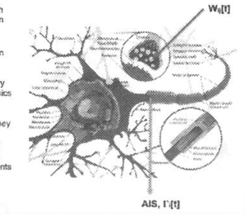


Figure 1: **Firing function $\Phi(V)$, firing density and phase diagram for the static model.** **a**, Rational firing function $\Phi(V)$ for $\Gamma = 0.5$ (bottom), 1.0 (middle) and 2.0 (top). **b**, Firing density $\rho(\Gamma W)$. The absorbing state $\rho^0 = 0$ looses stability after $\Gamma W > \Gamma_c W_c = 1$. **c**, Phase diagram in the $\Gamma \times W$ plane. ASOC can be done by adapting synapses (horizontal arrows) or adapting neuronal gains (vertical arrows) toward the critical line.

Why to separate the gain Γ from the synaptic weight W?

- In a biological network, each neuron i has a neuronal gain $\Gamma_i[t]$ located at the *Axonal Initial Segment* (AIS). Its dynamics is linked to sodium channels.
- The synapses $W_{ij}[t]$ are located at the dendrites, very far from the axon. Its dynamics is due to neurotransmitter vesicle depletion.
- So, although in our model they appear always together as ΓW, this is due to the use of point like neurons. A neuron with at least two compartments (dendrite + soma) would segregate these variables.



Dynamic synapses vs dynamic gains

- Dynamic synapses (Levina *et al.*, 2007; Levina *et al.*, 2009; Bonachela *et al.*, 2010; Costa *et al.*, 2015, Campos *et al.*, 2016):

$W_{ij}[t+1] = W_{ij}[t] + 1/\tau\, (A - W_{ij}[t]) - u\, W_{ij}[t]\, X_j[t]$ $\qquad$ $O(N^2)$ equations

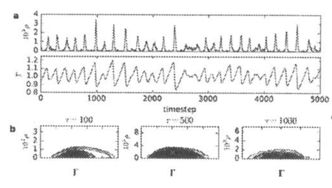
- Dynamic gains (Brochini *et al.*, 2016, Costa *et al.*, 2017, Kinouchi *et al.*, 2018):

$\Gamma_i[t+1] = \Gamma_i[t] + 1/\tau\, \Gamma_i[t] - u\, \Gamma_i[t]\, X_i[t]$ $\qquad$ $O(N)$ equations!

It is possible to simulate very large networks by using dynamic gains (firing rate adaptation) instead of dynamic synapses

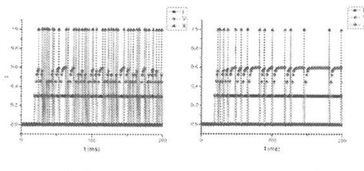
A, τ ⇒ W_c, Γ_c ⇐ u

Population activity ρ[t], average gain Γ[t] and ρ vs Γ orbits



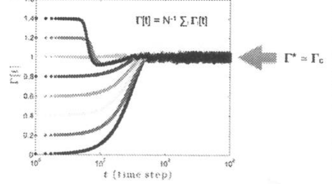
Dynamic gain Γᵢ[t] = a mechanism to produce firing rate adaptation

I = current, V = membrane voltage, X = spike



Constant gain $\qquad$ Dynamic gain

Self-organization of the average gain toward the critical region



Distribution of avalanche sizes P(s)
Large avalanches = Dragon Kings

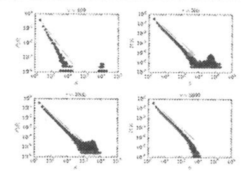
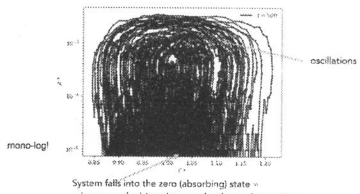


Figure 2: Avalanche size distribution $P(S)$ for the one parameter neuronal gain model. From left to right, τ = 100, 500, 1,000 and 5,000; all plots have N = 100,000. The straight line corresponds to the exponent −3/2.

Avalanches when orbit is close to ρ = 0
Dragon Kings when orbit is in the large loop



System falls into the zero (absorbing) state =
= interrupted orbits = large avalanches = dragon kings

Origin of the stochastic oscillations/dragon kings: Demographic noise close to the critical point

- Bidimensional mean-field map

$\rho[t+1] = F(\rho[t], \Gamma[t])$
$\Gamma[t+1] = G(\rho[t], \Gamma[t])$

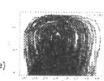
- Linear stability analysis:

Complex eigenvalues with modulus $|\lambda^{\pm}| = \sqrt{1 - \frac{\tau + 2}{\tau(\tau - 1)}}$

- Large (biological) recovery time $\tau > 100$ ms:

$|\lambda^{\pm}| = 1 - O(1/\tau) \approx 0.999$

- = Almost indifferent spirals (Neimark-Sacker bifurcation = critical point: $\lambda = 1$)
- + finite size network fluctuations (demographic noise) = HSO

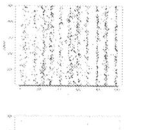


Conclusion: Power law avalanches + quasiperiodic Dragon Kings produced by a single ingredient: adaptive firing rates (dynamic gains)

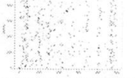
For small amplitude oscillations:

$$\omega = \arctan \frac{\sqrt{\tau + 2/\tau - 4}}{\tau - 2}$$

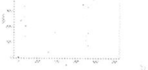
τ = 40ms, f = 25 Hz



τ = 100ms, f = 16 Hz



τ = 500ms, f = 7 Hz



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