Exploring neuronal dynamics through the Izhikevich and Kuramoto models

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Abstract: There are different mathematical models to describe the behavior of neurons, and with different levels of accuracy. In this study, we explored two major models, the biologically realistic Izhikevich model and the less realistic but convenient Kuramoto oscillator. We compared them to investigate whether it is correct to use Kuramoto oscillators to describe collective dynamics, such as synchronization, in large populations of realistic Izhikevich neuronal networks. We show that this is indeed the case when the number of Izhikevich neurons is large, which is demonstrated by coupling 5 groups of Izhikevich 1000 neurons each and showing that the whole system can be simplified as the sum of 5 coupled Kuramoto oscillators.

I. INTRODUCTION

The neuron is the basic building block for the processing of information in the nervous system of animals. Neurons coupled together shape neuronal circuits of high intricacy, where even the nervous system of the worm C. *elegans* (300 neurons) is able to perform complex tasks and fine interaction with the environment. Highly evolved neuronal networks, such as the human brain (10^{11} neurons) are organized as thousands of specialized microcircuits that carry out specific functions that are then put together to understand the world and interact with it. Thus, the human brain could be viewed as a set of thousands of coarse–grained 'worm neuronal circuits' that interact to one another. Interestingly, one of the most fascinating challenges of present neuroscience is to understand, and model, how neuronal circuits operate at different scales, and whether one could approach a 'worm circuit' by a simple model, to later study the interaction of this circuit with others, somehow ignoring the details of the neurons within the circuit itself [1].

Of course, scientist would prefer to model neurons exactly. However, neurons are non–linear systems whose main observable is the membrane potential, which increases or decreases according to inputs from other neurons. When the membrane potential reaches a threshold, it activates an avalanche of processes that lead to the generation of a pulse that becomes the input of other neurons. The accurate modeling of all processes involved is extremely difficult.

Indeed, the neuron's membrane potential gets generated by a difference in the concentration of charged ions, and can be described using elaborate mathematical models. When the potential sudden rises from the resting value to a threshold potential we have a *spike*, *i.e.*, we say that the neuron has fired. The action potential is then transmitted along the axon of the neuron and through the synapse to other neurons of the network [2].

One of the most detailed mathematical models for a neuron is the Hodgkin–Huxley model which provides a highly accurate biophysical description, and that led to the Nobel prize for its authors in 1963. The problem with this model is that it is computationally prohibitive. Instead, the Izhikevich model [3] is a simplification of Hodgkin–Huxley and can be solved with an ordinary personal computer. This model has become a favorite to model networks of few thousand neurons without difficulties.

Both the Hodgkin–Huxley and the Izhikevich are models for single neurons. In the quest for providing models that represent a large group of neurons, scientists observed that some neuronal circuits show collective behavior in the form of synchronous oscillations and that can be treated as harmonic oscillators, in which the average activity (spikes per unit time) oscillate up and down as the circuit receives and processes information. This observation led to the Kuramoto model [4], in which a neuronal circuit is just approach with an oscillator with a characteristic frequency and phase and that interacts with other circuits.

In the present study we want to investigate whether it is possible to bridge the Izhikevich and Kuramoto models. The idea is to explore whether simulations made with Kuramoto oscillators are suitable to describe the dynamics of interconnected spiking Izhikevich neurons. The question we want to address is: are few hundred coupled Izhikevich neurons behaving as a single Kuramoto?

Understanding the dynamics of neural networks is relevant to the comprehension of the brain function, both to understand how it processes information and to understand different diseases of the nervous system such as Parkinson or Alzheimer, which appear to be related to abnormal synchronization of brain cells or with a lack of connection between them [5] [6].

II. METHODS

Here we describe the two used models. Their codes have been obtained from the literature, adapted for the present study and programmed in Matlab [3] [7].

A. Izhikevich

Developed in 2003, this model of spiking neurons allows to describe the behavior of different types of cortical neurons, determined by four parameters, giving rise to the following coupled differential equations that have to be solved:

$$\dot{v} = 0.04v^2 + 5v + 140 - u + I, \tag{1}$$

$$\dot{u} = a(bv - u), \tag{2}$$

$$\text{if } v \geq 30\text{mV, then} \begin{cases} v \leftarrow c, \\ u \leftarrow u + d. \end{cases} \tag{3}$$

Here, v and u are the membrane and recovery potentials, respectively, and I is the synaptic current, which accounts for the number of inputs that a neuron receives, so the potential v depends on the number of firings of the neurons in the network. When the potential v exceeds the threshold potential of 30 mV, the neuron fires and the membrane potential resets to its state at rest [3]. The parameters a, b, c, and d govern neuronal dynamics and allow to model different kinds of neurons: a is the time scale of the recovery variable, u; b is the sensitivity of u; c is the reset value of the membrane potential,v; and d is the reset value of u.

Since neurons are interconnected, the characteristics of the input current I depend on the connectivity of the network, *i.e.*, the matrix S of synaptic connections. S gives the connection weights between neurons according to their type. Each simulation generates random values between $(0, 0.5)$ for excitatory neurons and between $(-1, 0)$ for inhibitory neurons. We note that excitatory neurons increase the membrane potential, while the inhibitory neurons reduce it. Izhikevich's original code is shown below:

```
% Created by Eugene M. Izhikevich,
% February 25, 2003
% Excitatory neurons    Inhibitory neurons
Ne=800;                 Ni=200;
re=rand(Ne,1);          ri=rand(Ni,1);
a=[0.02*ones(Ne,1);     0.02+0.08*ri];
b=[0.2*ones(Ne,1);      0.25-0.05*ri];
c=[-65+15*re.^2;        -65*ones(Ni,1)];
d=[8-6*re.^2;           2*ones(Ni,1)];
S=[0.5*rand(Ne+Ni,Ne),  -rand(Ne+Ni,Ni)];

v=-65*ones(Ne+Ni,1);    % Initial values of v
u=b.*v;                 % Initial values of u
firings=[];             % spike timings

for t=1:1000            % simulation of 1000 ms
 I=[5*randn(Ne,1);2*randn(Ni,1)];% thalamic
                                 % input
 fired=find(v>=30);    % indices of spikes
 firings=[firings; t+0*fired,fired];
 v(fired)=c(fired);
 u(fired)=u(fired)+d(fired);
 I=I+sum(S(:,fired),2);
 v=v+0.5*(0.04*v.^2+5*v+140-u+I);% step 0.5 ms
 v=v+0.5*(0.04*v.^2+5*v+140-u+I);% for numerical
 u=u+a.*(b.*v-u);                % stability
end;
plot(firings(:,1),firings(:,2),'.');
```

B. Kuramoto

Yoshiki Kurmaoto describes the systems of many limit cycle oscillators as systems that can be represented with simple elements called rings, their phase is defined by taking a point that circulates bound to it, when rings interact they adjust their phases and frequencies and can eventually synchronize [4].Kuramoto's equation is given by:

$$\dot{\theta}_i = \omega_i + \frac{K}{N} \sum_{m \neq i}^{N} sin(\theta_m - \theta_i). \tag{4}$$

Each of the oscillators is considered to have a phase θ_i and its own intrinsic natural frequency ω_i. The total N oscillators in the system modify the phase of the ith–oscillator with a coupling strength equal to K, when $K = 0$ there are independent oscillators. K is the same for all oscillators, , *i.e.* they are coupled together in the same way.

We used the Matlab function `ode45.m` to solve the system of N differential equations of Eq. (4). The result is a column vector with the steps of time and a matrix whose columns represent the temporal evolution of each oscillator's phase.

In neuroscience, networks of coupled oscillators provide models for systems governed by interacting periodic processes. Individual oscillators could be viewed as a small group of neurons shaping a microcircuit or neural masses ($\sim 10^6$ neurons) on a more macroscopic level [8].

The importance of noise in Kuramoto's model: Neural activity measured in electrophysiological studies typically shows stochastic fluctuations, even when the brain is at rest. Moreover, there is evidence that under certain conditions noise in neural networks promotes the exchange of signals between neurons through spike trains [9]. If the noise has such an important role in neural networks, it is appropriate to add some type of perturbation to the Kuramoto's model to simulate neural noise. The part of code shown below is the implementation of the noise in our simulation of the Kuramoto's oscillators. It consists of the following:

- For each iteration in solving the Kuramoto's differential equations, we add 1 to the counter i until it is equal to the variable `time_noise`.

- 50 neurons are randomly selected and their intrinsic frequency `omega(n_i)` is increased by a random value in the range $[0, 1]$.
- Then the counter i returns to 0 and the process is repeated.

Therefore, the higher the value of `time_noise`, the lower the intensity of the noise (to perform the simulation without noise, we give to the variable `time_noise` a negative value).

```
% omega are real column vectors
% of length n.
% n are real scalar, total number
% of neurons.
% i is a counter
% more timenoise -> less noise,
if the value is negative, there is no noise
i = i+1;
if i ==time_noise
    % it choice randomly 50 neurons
    [n_i] = randi(n,50,1);
    % the chosen neuron suffers a
    % increase / decrease of omega
    %(intrinsic frequency)
    omega(n_i) = omega(n_i)+randn(1,1);
    i=0;
end
```

III. RESULTS AND DISCUSSION

A. Neurons as Izhikevich units

The Izhikevich model allows us to model different types of neurons by changing the parameters $a, ...d$. As shown in the table below, We can shape two types of inhibitory neurons, FT and LTS, and two types of excitatory neurons, RS and CH.

Neouron Type	a	b	c	d
Regluar spiking (RS)	0.02	0.2	-65	8
Chattering (CH)	0.02	0.2	-50	2
Fast spiking (FS)	0.1	0.2	-65	2
Low-threshold spiking (LTS)	0.02	0.25	-65	2

Excitatory neurons favor neurons to activate, but too much excitation exhausts the neurons and blocks them to fire again for some time. For this reason inhibitory neurons are necessary. Indeed, when a neuron receives an inhibitory stimulus its membrane potential decreases, preventing the membrane potential to go to high too quickly. The combination of excitatory and inhibitory neurons is what makes the Izhikevich model so powerful, approaching the complex behavior and biological reality of the mammalian cortex [11].

To test the simulations, we have first simulated a network of 1000 excitatory, RS type (regular spiking) neurons by using the appropriate parameters (Fig. 1A). Neurons are coupled through the matrix S. Neurons activate synchronously but, since need a lot of time to recover, the interval between consecutive activations is large. In a second simulation, we have added inhibitory neurons (Fig. 1B), with a ratio 1/4 of inhibitory to excitatory, as in actual brains [10]. In this simulation, activity is much richer since inhibition facilitates neurons to fire less strongly and recover faster.

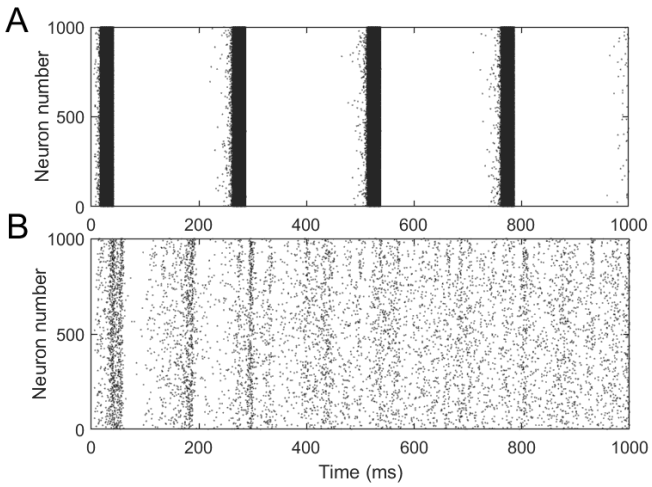

FIG. 1: Simulation of 1000 neurons using the Izhikevich model. (A) Just regular spiking neurons. (B) Combination of excitatory and inhibitory neurons.

B. Neurons as Kuramoto oscillators

We considered now a simulation of 1000 coupled Kuramoto oscillators. When we had the solutions of the differential equations, we accumulated in a vector the number of times each oscillator i has completed a cycle, to then graph the activation of oscillator i in each step of time. As shown in Fig. 2A, a point in the plot is a firing neuron. Next, we considered the above Kuramoto code with noise addition, as explained in the methods section, and repeated the simulations (Fig. 2B).

C. Comparison of the two models

In both simulations, and for the simplest approaches (Figs. 1A and 2A) we can appreciate vertical bands corresponding to the moments when most neurons fire together, followed by periods of relative silence. For the most complex scenarios, with inhibition for Izhikevich and noise for Kuramoto (Figs. 1B and 2B), we observe some similitudes.

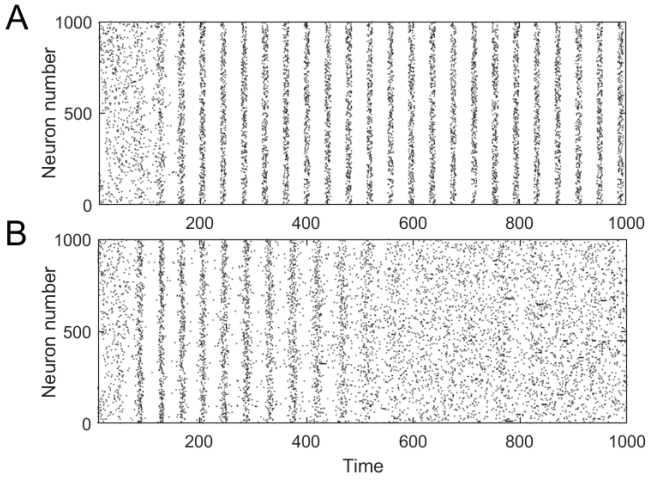

FIG. 2: Simulation of 1000 Kuramoto oscillators with K=0.9 and initial ω_i between $(2\pi 6, 2\pi 8)$. (A) Kuramoto without perturbation. (B) Kuramoto with neural noise, $time\_noise$=450.

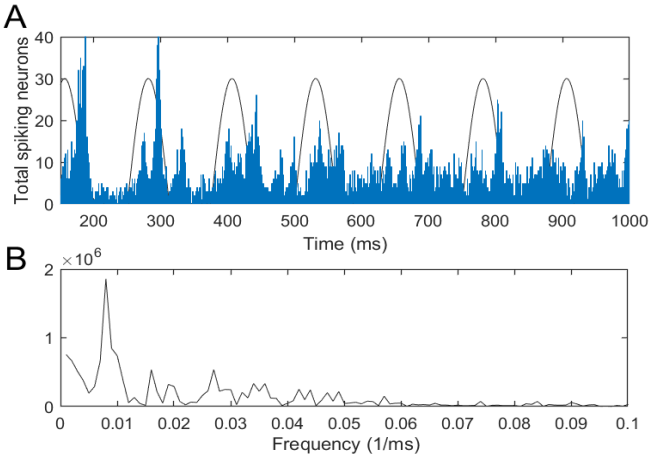

FIG. 3: (A) Histogram corresponding to Figure 1B. of the number of neurons that are activated as a function of time. (B) Fourier analysis of the predominant frequencies in the histogram.

For Izhikevich, on the one hand, the inclusion of inhibition reveals a richer dynamics, with more activity and not so sharp vertical bands. Kuramoto, on the other hand, shows how the added noise causes the system to have stages where there is synchronization and stages where noise predominates, similar to what we see in Izhikevich. Thus, it could be concluded that the implementation of noise in Kuramoto has a similar effect to incorporating inhibitory neurons to the network.

D. Neural masses and Kuramoto oscillators

For the above Izhikevich simulations (Figura 1B), it is interesting to represent in a histogram the number of neurons activating in a given time window. As shown in (Fig. 3A) a periodic behavior can be observed. With a Fourier analysis of this histogram (Fig. 3B) we can analyze the frequency of the peaks, providing about 0.008 ms^{-1}.

Thus, for coupled Izhikevich, the peaks where most of the neurons fire are repeating with a specific frequency, a result that can be approximated by just a single oscillatory movement.

After seeing this result, an interesting question that arises is whether connecting five Izhikevich neuronal networks is analogous to coupling five Kuramoto oscillators with intrinsic frequencies within $(0.007, 0.009)$ ms^{-1}. Thus, using a connectivity matrix, see (Fig. 4A), we created 5 groups of 1000 interconnected neurons, and where each group was connected with another with just 1.5% of connections. To do that, we randomly filled a 5000×5000 matrix with decimal numbers from 0 to 1. Above 0.015, we replaced them with 0 and the rest with 1. On the diagonal of the matrix we built the 1000×1000 sub–matrices that represent the 5 groups. Neurons within each group where strongly connected. The resulting matrix is multiplied element by element by the S matrix, related to synaptic interactions, so we have some neurons sending and receiving synaptic impulses between networks.

The simulations with 5000 neurons are shown in Fig. 4B. From these simulation we also made the histogram to know the total number of neurons that fired in any of the networks and compare it with the sum of 5 Kuramoto oscillators. The results are shown in Fig. 4B. In Figure 4B we can distinguish the different networks by their behavior, each one represented by a different color. What the histogram in Figure 4C shows is that despite having a differentiated behavior in each network, they show certain synchrony between them. When trying to fit the histogram of the 5 networks with the sinusoidal that results from the sum of 5 Kuramoto oscillators, we see that they adjust quite well.

IV. CONCLUSIONS

Firstly, it has been found that it is plausible to use Kuramoto oscillators to model the dynamics of interconnected neurons, whether we consider individual neurons as coupled oscillators or large masses of neurons as a single oscillator that alternate periods of high activity with periods of partial relaxation.

This leads to the conclusion that, for large systems such as neuronal networks in the human brain, it is possible to use Kuramoto oscillators to study large–scale phenomena, related to collective behaviors, for instance synchronization. This 'Kuramoto approach' saves resources associated to doing simulations with all the components of the system, effectively dividing $\sim 10^6 - 10^9$ neurons into $10^2 - 10^3$ groups, where each group would be a Kuramoto oscillator interacting with others through a mean–field description.

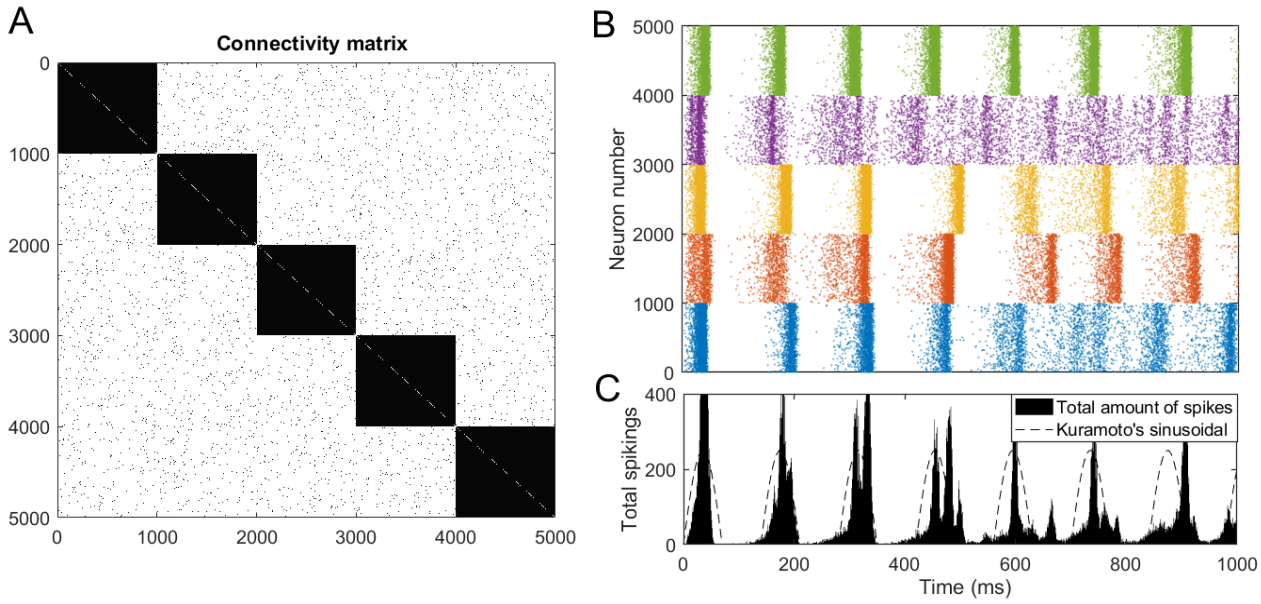

FIG. 4: (A) Matrix of 5 network groups strongly connected within the group itself and weakly connecting with neurons in other groups. The black dots are synaptic connections. (B) Corresponding simulations, showing 5 network groups of 1000 neurons each with 1.5% connections between groups, and where each color identifies the neurons that belong to a specific group. (C) Histogram of the total number of neurons that activated in the whole system. The dashed line shows the equivalent of using 5 coupled oscillators $\sin(\theta_i)$.

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[1] Soriano, J., & Casademunt, J. (2015). Cultiuus neuronals: un sistema model per entendre la complexitat del cervell. *Revista de Física*, **5(2)**, 29–37.

[2] Neuroanatomy, Neuron Action Potential`https://www.ncbi.nlm.nih.gov/books/NBK546639/`

[3] E. M. Izhikevich (2003). Simple model of spiking neurons, *IEEE Trans. Neural Netw. Learn. Syst*, **14**, 1569-1572.

[4] Kuramoto, Y. (1984). Cooperative Dynamics of Oscillator Community: A Study Based on Lattice of Rings. *Progress of Theoretical Physics Supplement*, **79**, 223–240.

[5] Guevara Erra, R., Perez Velazquez, J. L., & Rosenblum, M. (2017). Neural Synchronization from the Perspective of Non-linear Dynamics. *Frontiers in Computational Neuroscience*, **11**.

[6] Tijms, B. M., Wink, A. M., de Haan, W., van der Flier, W. M., Stam, C. J., Scheltens, P., & Barkhof, F. (2013). Alzheimer's disease: connecting findings from graph theoretical studies of brain networks. *Neurobiology of Aging*, **34(8)**, 2023–2036.

[7] Cleve Moler (2022). Kuramoto's model of synchronizing oscillators`https://www.mathworks.com/matlabcentral/file exchange/72534-kuramoto-s-model-of-synchronizing-oscillators`, MATLAB Central File Exchange. Retrieved January 6, 2022.

[8] Bick, C., Goodfellow, M., Laing, C. R., & Martens, E. A. (2020). Understanding the dynamics of biological and neural oscillator networks through exact mean-field reductions: a review. *The Journal of Mathematical Neuroscience*, **10**.

[9] Stein, R. B., Gossen, E. R., & Jones, K. E. (2005). Neuronal variability: noise or part of the signal? *Nature Reviews Neuroscience*, **6(5)**, 389–397.

[10] Xue, M., Atallah, B. V., & Scanziani, M. (2014). Equalizing excitation–inhibition ratios across visual cortical neurons. *Nature*, **511(7511)**, 596–600.

[11] Isaacson, J., & Scanziani, M. (2011). How Inhibition Shapes Cortical Activity. *Neuron*, **72(2)**, 231–243.