Quantifying wave propagation in a chain of FitzHugh-Nagumo neurons

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Understanding how external stimuli propagate in neural systems is an important challenge in the fields of neuroscience and nonlinear dynamics. Despite extensive studies over several decades, this problem remains poorly understood. In this work, we examine a simple "toy model" of an excitable medium, a linear chain of diffusely coupled FitzHugh-Nagumo neurons, and analyze the transmission of a sinusoidal signal injected into one of the neurons at the ends of the chain. We measure to what extent the propagation of the wave reaching the opposite end is affected by the frequency and amplitude of the signal. To quantify these effects, we measure the cross-correlation between the time-series of the membrane potentials of the end neurons. This measure allows, for instance, to detect threshold values of the parameters, delimiting regimes where wave propagation occurs or not.

Keywords: neural dynamics, FitzHugh-Nagumo model, excitability, noise

I. INTRODUCTION

Neuronal models are crucial in the exploration and understanding of oscillation modes of neural systems[1, 2], serving as foundational tools to investigate both, normal and pathological neuronal states. The FitzHugh-Nagumo (FHN) model [3, 4] has been extensively employed to study neuronal excitability and the dynamic behavior of single neurons and neuronal ensembles. Its ability to capture essential features of neuronal firing, while remaining computationally efficient, makes it an ideal model for examining large-scale neural dynamics, including synchronization, wave propagation, and pattern formation [5, 6].

The coupling of FHN neurons through diffusive interactions has been a subject of intense study, providing insights into how local interactions can lead to complex global behaviors such as synchronization and desynchronization [7, 8]. Diffusive coupling, which mimics the electrical interactions between adjacent neurons, is fundamental in coordinating neuronal activity. A coordination which is critical in various brain functions, such as sensory processing, motor control, and cognitive tasks [9, 10].

External inputs stimulate neuronal dynamics, influencing both physiological and pathological states. Local stimulation can induce long-range order [11] and transient coherence [12]. Therefore, it is relevant to study how an external stimulus propagates and affects the neural dynamics. In this respect, due to their periodic and elementary shape, sinusoidal signals have been used to study the response properties of single neurons and neuronal ensembles [13–19]. These studies reveal that neuronal systems can exhibit resonance phenomena, where certain frequencies of external inputs maximize or optimize the system's response. Studies have also explored how resonant response to external stimuli might modulate or disrupt pathological synchronization [20, 21]. In addition to neuronal models, networks of biochemical oscillators have also been used as excitable media where local perturbations can be enhanced [11, 12]. In such systems, it was found that an external periodic perturbation of an oscillator produced, for certain ranges of the stimulation frequency, the appearance of globally coherent states.

However, how external signals propagate in neural systems is still far from being understood, and the purpose of the present work is to analyze signal propagation in a linear chain of diffusely coupled FHN neurons, considering sinusoidal and noisy signals. Our study contributes to ongoing efforts to better understand how external inputs influence neural behavior and timing.

II. MODEL

We analyze the response of a one-dimensional excitable medium to an applied sinusoidal signal, using a model that was previously studied in Refs. [17, 22]. The excitable medium consists of a chain of N diffusively-coupled identical FHN neurons and the sinusoidal signal is injected into one of the neurons at the ends of the chain. The governing equations are

$$\frac{dx_i}{\epsilon dt} = x_i(a - x_i)(x_i - 1) - y_i + I_{ext} + D_i + \theta_i(t)/\epsilon,$$

$$\frac{dy_i}{\epsilon dt} = bx_i - cy_i,$$
(1)

where x_i is the membrane potential and y_i is the recovery current of the *i*th neuron. I_{ext} is the external stimulus current, and a, b and c are positive parameters, which we keep fixed as $(a, b, c, I_{ext}) = (0.1, 0.015, 0.015, 0.62)$. We choose these parameter values because they place the single neuron dynamics just above the critical threshold, enhancing the sensitivity of the neurons to excitation inputs. ϵ denotes a time scaling coefficient, which allows to adjust the resonant frequency of the neurons. Finally D_i are diffusive coupling functions defined with zero-flux boundary conditions:

$$D_1 = D_x(x_2 - x_1),$$

$$D_i = D_x(x_{i+1} - 2x_i + x_{i-1}) \text{ for } i = 2, 3, \dots, N-1,$$

$$D_N = D_x(x_{N-1} - x_N),$$

with coupling strength D_x . Diffusive coupling means that the activity of one neuron is influenced by the difference between its activity and that of its nearest neighbors. We select D_x to be large enough to produce local excitation but not long-range order [17]. Throughout our study, we use $D_x = 0.04$ for networks of size N = 20, and set $\epsilon = 10$, following Refs. [17, 22] to allow comparisons. Additionally, the neurons are subjected to external time-dependent inputs $\theta_i(t)$ with zero mean. To study the propagation of a single external input, after a transient time t_0 , a sinusoidal signal of amplitude A and angular frequency w is added to the first neuron. Therefore,

$$\theta_1(t) = A\sin(\omega t), \quad \forall t > t_0, \tag{2}$$

$$\theta_i(t) = 0, \quad \forall t, \forall i \neq 1.$$
 (3)

The model equations were simulated using a standard 4th order Runge-Kutta algorithm with $dt = 10^{-2}$. A typical space-time plot obtained for N = 20 neurons, when no external input is applied, is presented in Fig. 1. We can observe that for the coupling strength considered, the neurons do not oscillate regularly and are only partially synchronized.

III. QUANTIFYING SIGNAL PROPAGATION

To quantify the wave propagation induced when a signal is injected, we measure the cross-correlation between the time series of the first and last neurons in the chain. This is an



Figure 1. (a) Space-time plot of the membrane potentials, $x_i(t)$, i = 1, ..., N in gray scale, for a chain on N = 20 FHN neurons, without external input (A = 0.0). (b) Time-series of the membrane potential of three neurons, x_1 , x_{10} and x_{20} . The maximum of the cross-correlation between x_1 and x_{20} , estimated over 100 simulations with random initial conditions is $C_{max} = 0.61 \pm 0.14$.

effective procedure to assess whether the signal injected into the first neuron reaches the last and to quantify their similarity as a function of the time lag τ , defined as

$$C \equiv C_{x_1 x_N}(\tau) = \frac{\langle (x_1(t) - \langle x_1 \rangle) (x_N(t+\tau) - \langle x_N \rangle) \rangle}{\sigma_{x_1} \sigma_{x_N}},$$
(4)

where $\langle \cdots \rangle$ denotes average, which was computed at intervals $\Delta t = 5dt$ and σ_x is the standard deviation of x.

IV. RESULTS

In this section we perform a systematic quantitative study of the effects of the frequency and amplitude of the sinusoidal signal added to the first neuron; we also discuss the effects of additive white noise. In all cases we use the cross-correlation to detect threshold values of the parameters that define the regimes where propagation occurs.

A. Effect of the signal's frequency

In Fig. (2), we plot the space-time diagrams for a typical network where we injected, from time $t_0 = 150$ up to the end of the simulation, the signal $\theta_1(t)$ defined in Eq. (2), considering different values of the angular frequency w.

For some frequencies the signal resonates with the neurons' oscillations, reaching the end of the chain, while for other frequencies the signal does not propagate far enough to reach the



Figure 2. Space-time plots of the neurons' oscillations (top panels: $x_i(t)$, i = 1, ..., N in gray scale), when the linear chain has N = 20 neurons, for different values of the signal's angular frequency, $\omega = 0.4$ (a), 0.7 (b), 1.5 (c) and 1.8 (d). The signal's amplitude is A = 0.3 and the coupling strength is $D_x = 0.04$. We also present the time evolution of the two end neurons (central panels – x_1 in black and x_N in magenta) and the cross-correlation between them vs. the time lag τ (lower panels), measured in the time interval $t \in [800, 1000]$. We observe that in the time interval considered, when $\omega = 0.4$ and 1.8 the signal does not reach to the end of the chain, while when $\omega = 0.7$ and 1.5 the signal reaches the end neuron.

opposite end, as already known from Ref. [17]. This can be noticed more clearly in the central plots where we display the time series $x_1(t)$ (dark black) and $x_2(t)$ (light magenta). Similar outcomes have been observed when, instead of neurons, the units in the chain model calcium oscillations [11, 12].

When propagation occurs, the neurons' dynamics becomes periodic and the spiking activity of the two neurons at the two ends of the linear chain is very similar, as it can be seen when comparing x_1 and x_N in Figs. 2(b) and 2(c). Furthermore, larger frequencies allow faster propagation, denoted by the larger slope of the fronts in the space-time plots (this can be seen when comparing the space-time plot for $\omega = 1.5$ shown in Fig. 2(c) with that for $\omega = 0.7$, shown in Fig. 2(b)).

Now, to quantify the degree of propagation and detect possible thresholds, we use the cross-correlation C between the membrane potentials of the end neurons, defined in Eq. (4),



Figure 3. Maximum value of the cross-correlation between x_1 and x_N , vs. the signal's angular frequency w, for a chain that has (a) N = 20 neurons (here $D_x = 0.04$), and (b) N = 100 neurons (here $D_x = 0.1$). In both panels the signal's amplitude is A = 0.3. The cross-correlation is calculated over the time window $t \in [800, 1000]$. Averages (symbols) and standard deviation (bars) over 100 realizations, starting from random initial conditions, are shown.

that we plot as a function of the time lag τ for each value of ω in Fig. 2.

In the cases where the signal does not propagate (e.g., for $\omega = 0.4$), the cross-correlation between the first and last neurons is not periodic and its maximum value (C_{max}) is less than 0.5. On the other hand, for $\omega = 0.7$, which is close to a resonant value, the signal propagates reaching the end of the chain. In such case, the correlation between the first and last neurons is periodic (with period $\simeq 2\pi/\omega$) and its maximum value is ≈ 1 , which characterizes a perfect correlation and means that the (normalized) perturbed time series x_1 propagated without deformation. Note that, since the membrane potential is not symmetric, when x_1 and x_N are in counter-phase the correlation in absolute value is smaller that when they are in phase.

To summarize the outcomes obtained when varying the angular frequency, we plot the maximum value of the crosscorrelation vs. ω in Fig. 3(a) for a network of size N = 20. As expected, resonance occurs for frequency bands around to integer multiples of the natural frequency of the limit cycle which corresponds to $\omega \simeq 0.75$. In Fig. 3(b), we show the response profile for N = 100. Increasing system size, while keeping constant the coupling D_x , prevent propagation, then we adjusted its value to $D_x = 0.1$, when N = 100. In this case, the frequency bands that facilitate or prevent propagation are similar to the case N = 20, although some differences are observed mainly in the band borders, which may require a finer adjustment of D_x to achieve higher similarity between the plots in Fig. 3(a) and Fig. 3(b).

B. Effect of the signal's amplitude

Space-time plots illustrative of the neurons' behavior for two different amplitudes, with w = 0.7 (nearly resonant case) are presented in Fig. 4. Even if the frequency of the signal is close to the resonance frequency of the neurons and thus facilitates the propagation of the signal, we observe that, for small amplitude, the signal does not reach the other end. In panel (b), for larger amplitude, we see that the signal propagates and reaches the other end.

The maximum cross-correlation C_{max} as a function of A, for $\omega = 0.7$, is shown in Fig. 5(a), where we see that there is a threshold value of the amplitude below which the signal does not reach the opposite end (for the parameters considered here, $A \simeq 0.08$). For both regimes (i.e., for signal's amplitudes above or below the threshold for signal propagation), the cross-correlation deteriorates with increasing amplitude. That is, the network is selectively responsive to excitation inputs, acting as a filter that ensures that only significant (but not too large) inputs spread fast, which may be crucial for maintaining coherent transmission in neuronal networks.

We also checked the effect of the amplitude when the signal's frequency is non-resonant, e.g., w = 0.4, The plot of C_{max} vs. A displayed in Fig. 5(b) reveals that the signal fails to propagate, because C_{max} is small and tends to decrease with increasing A.

Let us remark that the results obtained in this section are consistent with the results reported in Refs. [11, 12] that studied chains of calcium oscillators, also modelled by FHN diffusely coupled units.



Figure 4. Space-time plots of the neurons' oscillations, when the chain has N = 20 neurons. In (a) the signal's amplitude, A = 0.05, does not allow the signal to propagate; in (b) the amplitude is larger, A = 0.1, and allows for signal propagation. Other parameters are $D_x = 0.04$ and $\omega = 0.7$.



Figure 5. Maximum value of the cross-correlation between x_1 and x_N , vs. the signal's amplitude A in log scale. The signal's angular frequency is (a) $\omega = 0.7$, and (b) $\omega = 0.4$. Other parameters are N = 20 and $D_x = 0.04$. As in previous figures, the cross-correlation is calculated over the time window $\in [800, 1000]$. Averages (symbols) and standard deviation (bars) over 100 realizations, starting from random initial conditions, are shown.

C. Injecting white noise

Here we study the effect of white-noise added to the sinusoidal signal, and also, the effect of injecting solely whitenoise, where all frequencies are present.

Here we generate the white noise via the Box-Muller method for generating pairs of independent, standard normally distributed (Gaussian) random numbers, given two independent uniformly distributed random numbers r_1 and r_2 in the interval (0, 1), yielding Gaussian distributed numbers G with zero mean and standard deviation g, through the expression $G = g\sqrt{-2\ln(r_1)} \cos(2\pi r_2)$.

Space-time plots of signals with A = 0.3 in presence of



Figure 6. Maximum cross-correlation between x_1 and x_N , vs. the signal's frequency ω , as in Fig. 3(a) with the addition of white noise with g = 0.3. Averages (symbols) and standard deviation (bars) over 100 realizations, starting from random initial conditions, are shown.

white-noise with g = 0.3 added to the input, as a function of ω , shown in Fig. 6, do not exhibit significant differences, as a function of ω , when compared to Fig. 3(a), where noise is absent. Moreover, for $\omega = 0.7$, we plotted the maximal correlation as a function of $g \in [0, 2]$ yielding a very flat profile at a level $C_{max} \simeq 0.96$ (not shown). These comparisons indicate that the results are robust enough under the addition of noise.



Figure 7. Spatio-temporal dynamics of the neurons when the input signal is white noise with different amplitudes of the Gaussian noise, $g \in [0, 5]$. We see that strong enough noise induces spikes that can reach the other end of the chain, but the spiking dynamics does not persist.



Figure 8. Time series of the membrane potentials of the end neurons, x_1 (black) and x_N (magenta), when the input signal injected in the first neuron is Gaussian white noise with amplitude g = 2.0. Other parameters are N = 20, $D_x = 0.04$.

We inspected the effects of white-noise signals injected alone (without regular oscillations) into the system (with A = 0). We note, in Fig. 7, that even for high amplitudes of the noise, persistent propagation does not occur. Although all fre-

quencies are contained in the white-noise, in particular resonant ones, their contribution does not have enough amplitude for propagation. However, note that, despite not reaching the end of the chain, the fronts tend to propagate temporarily. In Fig. 8 we show segments of the time-series of x_1 and x_N for which transient propagation has occurred. Also note that the induced spikes that reach the last neuron have a shape similar to that of the sinusoidal input with fundamental resonant frequency, as in Fig. 2(a).

D. Subcritical neurons

Let us finally comment about the propagation in a linear chain of neurons that have a slightly subcritical external current, e.g., $I_{ext} = 0.05$. In this case, when adding the sinusoidal signal to a single (isolated) FHN neuron, after a transient the neuron evolves in a limit cycle, while without the sinusoidal signal, the neuron remains in a stable steady state (a focus) in the absence of external perturbations.

For a chain of 20 subcritcal neurons, with the same coupling coefficient as before, $D_x = 0.04$, signals do not propagate, even those with frequencies equal or close to the resonant frequencies. This motivated us to study the effect of the coupling coefficient, D_x . In fact, we observe a threshold value of D_x , above which propagation can indeed occur, as shown in Fig. 9 and Fig. 10 where the space-time plots for D_x just above and below the threshold are shown. Therefore, with the sinusoidal input stimulus, neurons that are in an inactive state can be excited to propagate information, if the coupling strength is high enough.



Figure 9. Maximum cross-correlation between x_1 and x_N , vs. coupling strength, D_x , for a linear chain of N = 20 subcritical neurons with $I_{ext} = 0.05$. The signal's parameters are $\omega = 0.7$ and A = 0.3. As in previous figures, the cross-correlation is calculated over the time window $\in [800, 1000]$. Averages (symbols) and standard deviation (bars) over 100 realizations, starting from random initial conditions, are shown.

V. CONCLUSION AND OUTLOOK

We have studied the propagation of simple sinusoidal signals (which can also be noisy) in excitable media, using a toy model that consists of a linear chain of diffusively coupled



Figure 10. Space-time plots of the neurons' oscillations, when the chain has N = 20 subcritical neurons and the signal's parameters are A = 0.3 and $\omega = 0.7$. The coupling coefficient is (a) $D_x = 0.08$ and (b) $D_x = 0.09$.

FHN neurons. We measured and quantified the transmission efficiency in terms of the cross-correlation coefficient between the time series of the membrane potential of the neuron that perceives the signal and the time series of the membrane potential of the neuron at the other end of the chain. We have shown that the method allows to detect threshold values of the parameters, delimiting regimes with or without propagation.

Our study offers a basic reference and method of analysis for the use of linear chains of neurons as excitable media to study the emergent dynamics and coherence that can be generated by complex artificial or physiological signals which have different components and scales, along the line of previous studies [11, 12, 22]. This is important as these systems could have a practical application as a tool for pattern recognition, to detect for instance neurological disorders, which would be a natural extension to applythe quantifier used in this work.

It would also be interesting to study other neuron models[5, 23–27] that exhibit complex dynamics, such as bursts of spikes. In addition, it would be worth to study other coupling schemes, such as pulse-based coupling, effects of heterogeneities, in the couplings or in the parameters of the neurons, . and to go beyond the simplest near-neighbour coupling considered here, and consider long-range couplings with distant-dependent delays. Another interesting perspective for feature work would be to quantify the propagation of the signal using nonlinear quantifiers, in particular, instead of correlating the whole time series of the membrane potential of the two neurons at the end of the linear chain, one could correlate the timing of the spikes fired by those neurons, by using spike-based synchronization measures[28].

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