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Optimal dynamic range of spiking neurons in critical scale-free networks

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Abstract

According to Stevens' law, the connection between stimulus and response is characterized as a power-law within a stimulus range called the dynamic range. The dynamic range of sensory organs is considered wider than that of a single neuron, indicating that the network configuration is crucial for understanding groups of neurons. To computationally investigate the stimulus-response relationships in firing neurons, we studied scale-free networks of neurons modeled using the Hodgkin-Huxley equations interconnected by chemical synapses. Our results showed that the dynamic range expands with increasing network size, suggesting that the widening of the dynamic range observed in sensory organs, compared to isolated neurons, is an emergent feature of the dynamics of complex networks.

1 Introduction

The basic problem of psychophysics is to understand how physical stimuli (light, sound, pressure, odor concentration, etc.) transduce into psychological sensations (vision, hearing, touch, smell, etc.) [1]. The intensity of stimuli varies by many orders of magnitude; hence, the corresponding sensation must exhibit a wide dynamic range. More

precisely, the dynamic range is the ratio between the maximum and minimum intensities of the response to a given stimulus [2]. The use of a logarithmic scale enables us to measure the dynamic range in decibels. For example, the dynamic range of human hearing is *circa* 120 dB in the 20 Hz – 20 kHz frequency interval, since the minimum threshold is ~ 0 dB (as for whispering) and the threshold of pain is ~ 120 dB (like a jet turbine) [3].

The relation between stimulus and response intensities, denoted as S and F respectively, is clearly nonlinear and has been studied for many years since the pioneering investigations of Weber and Fechner in the 1860's. The latter proposed a relation of the form $F \sim \log S$ [4]. A century later, Stevens proposed a power-law relation $F \sim S^m$, where m is a characteristic exponent [5]. For sound sensations, it turns out that $m = 0.67$ for a frequency of 3 kHz [6]. Note that m can be less than or greater than unity, depending on the type of stimulus and response. A related power-law relation was given by Hill, and other relations have been proposed as well [7].

The stimulus-response relation can be appreciated from two different perspectives: (i) the macroscopic level related to the stimuli caused by physical agents and the responses as the psychological sensations mentioned so far; (ii) the microscopic level related to the neuronal response to an electrical input stimulus [8]. In the latter case, we consider the input as a spike train where the interspike intervals obey a Poisson distribution, and the output is the firing rate of the neuron [9].

One of the major challenges in Theoretical Neuroscience is to relate these two perspectives. A problem of recent interest focuses on the difference between the dynamic ranges observed at both the macroscopic and microscopic levels. For example, in the olfactory system, the dynamic range is ~ 10 dB for individual receptor neurons, whereas the dendro-dendritic neuronal network in the glomeruli has a higher dynamical range of ~ 30 dB, i.e., a threefold increase [10, 11].

Previous results using numerical simulations suggest that, while individual neurons may exhibit a small dynamic range, a substantial increase can be obtained as a collective effect displayed by the neuronal network [12]. Moreover, it has been observed in numerical simulations that the dynamic range is maximized if the network behavior is critical, and the power-law exponent turns out to be $m = 1$ in this critical state [13]. It has been speculated that brain activity exhibits self-organized criticality through the analysis of neuron avalanches, and that this critical state maximizes a number of neuronal functions, one of which being the dynamic range [14].

That simple models of neuronal behavior are capable of validating some of these claims encourages us to further our analysis by searching for critical neuronal networks with complex connection topologies [15]. We have previously verified these findings in a network of Hodgkin-Huxley neurons coupled in small-world networks [16]. This connection architecture has been observed in a variety of anatomical and functional neuronal networks, but there are aspects of the latter that might be better represented by scale-free networks [17].

In scale-free networks, the number of connections k for each unit (such as a neuron) satisfies a statistical power-law dependence [18]. Denoting by $P(k)$ the frequency distribution of the connectivities, in scale-free networks it turns out that $P(k) \sim k^{-\gamma}$, where $\gamma > 1$ [19]. Hence, for scale-free networks, a few neurons are connected to a

large number of others, whereas most of them are connected to a small number of units. In scale-free networks of the Barabási-Albert type, the networks expand in a finite number of steps by the addition of new nodes, such that they attach preferentially to already well-connected nodes, and $\gamma = 3$ for a very large number of nodes [20]. Finite size effects make this exponent slightly lower (in our numerical simulation, using $N = 550$ neurons, it is $\gamma = 2.8$).

In scale-free neuronal networks, the relative sparseness of their coupling topology reduces the memory needed to store a given amount of information and the computational effort needed to perform certain tasks. Indeed, scale-free properties have been observed experimentally in human brain functional networks by Chialvo *et al.* [17]. Another reason to investigate scale-free networks is their ubiquitous presence in systems displaying self-organized criticality, a fact explored in depth by Per Bak and his collaborators [21].

In the present paper, we consider scale-free networks (of the Barabási-Albert type) of Hodgkin-Huxley neurons subjected to external sensory stimuli represented by trains of electrical impulses, or spikes, modeled by stereotyped events [22–24]. The spike trains are such that the interspike time intervals are randomly distributed according to a Poisson process with a given rate [25]. The response, in this case, is modeled by the firing rate of the excitable neurons.

By modeling the neuronal response using a power-law (Stevens), we computed both the dynamic range and the power-law exponent. Our main goal is to verify the existence of an optimal dynamical range that can be assigned to a critical behavior of the network in order to validate the hypothesis that an enhanced dynamic range is essentially a collective effect of the network. We have also investigated the role of the number of neurons in these results.

This paper is organized as follows: in Section 2, we present the model used to describe both the neuronal dynamics based on Hodgkin-Huxley (HH) equations and chemical synapses. Section 3 presents the input spiking rate for a neuron and discusses the scaling region and its characterization. The connection architecture, namely the scale-free network structure, is described in Section 4. A detailed discussion of the numerical results for the coupled HH neurons in scale-free networks is presented in Section 5. The last Section is devoted to our Conclusions.

2 Neuronal dynamics and network models

The Hodgkin-Huxley (HH) model is a mathematical model that describes how action potentials are generated and propagated in neurons [26]. It was proposed in 1952 by its authors to explain the squid giant axon action potentials and is still used to this day as a prototypical conductance-based model for neuronal dynamics [27].

The HH model describes the time evolution of the membrane potential V_i of the i -th neuron (measured in mV), given by the nonlinear ordinary differential equation (with time measured in ms) and $i = 1, 2, \dots, N$,

$$C_m \frac{dV_i}{dt} = -I_{i,Na} - I_{i,K} - I_{i,L} - I_{i,ext} - I_{i,syn}, \quad (1)$$

where $C_m = 1.0 \mu F/cm^2$ is the lipid membrane specific capacitance, $I_{i,K}$ and $I_{i,Na}$ are potassium and sodium currents per unit area, respectively (measured in $\mu A/cm^2$), related to the ion channels embedded in the neuronal membrane, I_{iL} is the leak current, $I_{i,ext}$ is the external current, and $I_{i,syn}$ is the total current from synaptic connections with other neurons.

Voltage-gated ion channels are represented by the specific currents related to ion and leak channels, described by the following conductance-based expressions [28]

$$I_{i,K} = \bar{g}_K n_i^4 (V_i - E_K), \quad (2)$$

$$I_{i,Na} = \bar{g}_{Na} m_i^3 h_i (V_i - E_{Na}), \quad (3)$$

$$I_{i,L} = \bar{g}_L (V_i - E_L), \quad (4)$$

where $\bar{g}_K = 36 mS/cm^2$, $\bar{g}_{Na} = 120 mS/cm^2$, and $\bar{g}_L = 0.3 mS/cm^2$ are the maximum specific conductances of the potassium, sodium, and leak channels, respectively, and the corresponding Nernst (reversal) potentials are $E_K = -72 mV$, $E_{Na} = 55 mV$, and $E_L = -49 mV$.

The potassium activation variable (n_i), the sodium activation variable (m_i), and the sodium inactivation variable (h_i) are non-dimensional quantities that satisfy the following differential equations, respectively,

$$\frac{dn_i}{dt} = -(\alpha_n(V_i) + \beta_n(V_i))n_i + \alpha_n(V_i), \quad (5)$$

$$\frac{dm_i}{dt} = -(\alpha_m(V_i) + \beta_m(V_i))m_i + \alpha_m(V_i), \quad (6)$$

$$\frac{dh_i}{dt} = -(\alpha_h(V_i) + \beta_h(V_i))h_i + \alpha_h(V_i), \quad (7)$$

where $\alpha_{n,m,h}$ and $\beta_{n,m,h}$ have units in $(ms)^{-1}$ and have nonlinear dependencies on the membrane potentials given by

$$\alpha_n(V) = -0.01 \frac{V + 50}{\exp[-0.1(V + 50) - 1]}, \quad (8)$$

$$\beta_n(V) = 0.125 \exp \left[-\frac{(V + 60)}{19.7} \right], \quad (9)$$

$$\alpha_m(V) = -0.1 \frac{V + 35}{\exp[-0.1(V + 35) - 1]}, \quad (10)$$

$$\beta_m(V) = 4 \exp \left[-\frac{(V + 60)}{18} \right], \quad (11)$$

$$\alpha_h(V) = 0.07 \exp \left[-\frac{V + 60}{20} \right], \quad (12)$$

$$\beta_h(V) = \frac{1}{\exp[-0.1(V + 30)] - 1}. \quad (13)$$

The total current reaching the i -th neuron due to its synapses with the other neurons is given by a diffusive electrical coupling

$$I_{i,syn} = \bar{g}_c \sum_{j=1}^N A_{ij} r_j(t) (V_{i,syn} - V_j), \quad (14)$$

with $\bar{g}_c$ being the maximum specific conductance, A_{ij} are the elements of the synaptic adjacency matrix characterizing the network connection architecture; $V_{i,syn} = 20 \text{ mV}$ is the synaptic reversal potential of the i th neuron, and $r_j(t)$ is the fraction of bond receptors (open synaptic channels) for the j th neuron. This fraction obeys the following equation [29, 30]

$$\frac{dr_j}{dt} = \left(\frac{1}{\tau_r} - \frac{1}{\tau_d} \right) \frac{1 - r_j}{1 + \exp(-V_j + V_0)} - \frac{r_j}{\tau_d}, \quad (15)$$

where V_j is the membrane potential of the j th pre-synaptic neuron, $\tau_r = 0.5 \text{ ms}$ and $\tau_d = 8 \text{ ms}$ are the characteristic rise and decay times, respectively, and $V_0 = -20 \text{ mV}$ is the synaptic reversal potential.

The neuronal network is characterized by the adjacency matrix a_{ij} (which is equal to unity if neurons i and j are synaptically coupled, and zero otherwise). Since no neuron can be coupled to itself, the diagonal elements of the adjacency matrix vanish ($a_{ii} = 0$). We take synaptic couplings to be unidirectional, such that the adjacency matrix is not symmetric in general. The degree k_i of the i -th neuron is the number of other pre-synaptic neurons coupled to it; hence $\sum_j a_{ij} = k_i$. In directed graphs like the ones we considered in this paper, we can distinguish between in-degrees (the number of incoming connections) and out-degrees (the number of outgoing connections); however, for statistical purposes, we will not distinguish between them.

We define a degree probability density function $P(k)$ such that $P(k) dk$ is the probability of finding a neuron with a degree between k and $k + dk$. For random networks, for example, it turns out that $P(k)$ is a Poisson distribution. Many neuronal networks of neuroscientific interest, however, display a power-law form $P(k) \sim k^{-\gamma}$, where $\gamma > 1$ [18]. Eguiluz *et al.* have obtained functional networks through magnetic resonance imaging of human brains: the activity of a given voxel from the visual, motor, and posterior-parietal cortices is measured as a function of time [17]. Two voxels are considered functionally correlated if the corresponding Pearson correlation r exceeds a given threshold r_c . If two voxels are functionally correlated (regardless of their anatomical connectivity), the adjacency matrix element is unity, and zero otherwise. The degree distribution function of the resulting functional network showed a power-law form with an exponent $\gamma \approx 2$, the exact value depending on r_c . Usually $2 \leq \gamma \leq 3$.

In scale-free networks like these, most neurons have few connections, while a few hub neurons have many connections. This structure enables robust communication and high-speed information propagation. This is due to the highly connected neurons that facilitate network synchronization, rapid information propagation, and robustness against random failures. Moreover, this structure helps in the spread of neural activity,

which can lead to localized or cascading bursts. In fact, power-law distributions are commonly found in self-organized critical systems in which cascades obey scale-free statistics.

Scale-free networks can be computationally obtained through the Barabási-Albert procedure by starting from a seed network with a few neurons [20]. In each step, we randomly add new neurons to this initial network so that the new neurons preferentially connect to already well-connected neurons until the network reaches a given size. Let us consider that each new connection is linked to other c already connected neurons (or nodes). The probability that a new node is connected to any other node i is $p_i = k_i / \sum_j k_j$. It can be shown that the average degree is $\langle k \rangle = 2c$.

Moreover, the probability degree distribution function obtained through the Barabási-Albert method is, for $k \gg 1$, a power-law $P(k) \sim k^{-3}$, i.e., the power-law exponent is $\gamma = 3.0$ for sufficiently large networks [19]. Smaller networks yield $\gamma \lesssim 3.0$. In fact, for $N = 550$ nodes, we have obtained a Barabási-Albert scale-free network with $\gamma = 2.8$. It is from this generated network that we obtain the corresponding adjacency matrix a_{ij} .

In summary, in the version of the HH model we are using in this work, there are five first-order differential equations for each neuron, resulting in $5N$ equations for a network of N coupled neurons. In the numerical simulations of the model, we take the maximum conductivity $\bar{g}_c$ as the control parameter, while all the others take on fixed values. These equations were integrated numerically using a fourth order fixed step size Runge–Kutta method. We used random initial conditions to start the numerical simulations.

3 Stimulus-response relation for a single neuron

From a microscopic point of view, the stimulus S comes from an input current $I_{i,ext}$ acting on a neuron; see Eq. (1). The input current consists of supra-threshold spike trains. The intensity of the stimulus does not reflect the magnitude of the external input current due to the physiological limitations of the neuron, but rather depends on the frequency of spikes, i.e., on the interspike interval. We model this input spike train as a Poisson process of supra-threshold pulses of fixed magnitude $I = 2 \mu A/cm^2$, [13]

$$I_{i,ext} = I \sum_n \delta(t - t_n^{(i)}), \quad (16)$$

where $t_n^{(i)}$ is the time of the n -th spike acting on the i -th neuron. The interspike intervals $\tau_n = t_{n+1}^{(i)} - t_n^{(i)}$ are distributed according to a Poisson process with a constant average spiking rate r , measured in $(ms)^{-1}$, and following a continuous probability distribution $1 - \exp(-r\tau)$.

A single HH neuron responds to an input spike by emitting an electric pulse, provided the input arrives when the neuron is not in a refractory state. Hence, we expect the distribution of spikes produced by the neuron to be close to the input distribution, perhaps with some delay. We verified this to be valid; an example of a single, isolated neuron being illustrated by Fig. 1(b), where a spike train is shown

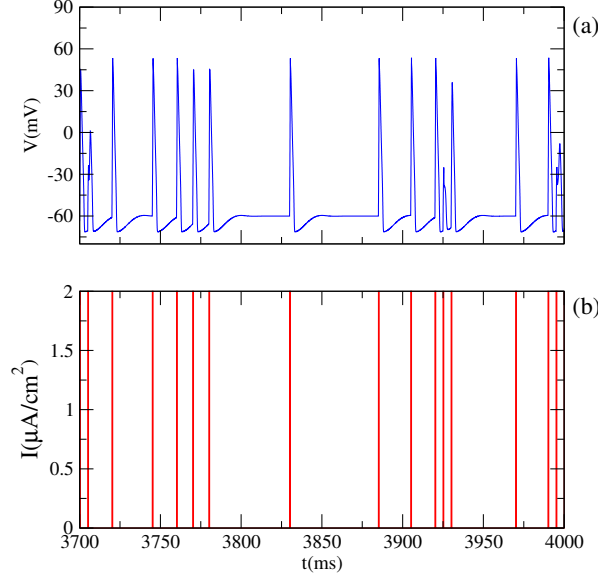


Fig. 1 (a) Time evolution of the membrane potential and (b) input spike train for a single neuron described by the Hodgkin–Huxley equations.

between 3700 and 4000 *ms*. The interspike intervals satisfy a Poisson distribution. The neuron response [Fig. 1(a)] shows a sequence of spikes that is very similar to the input train, with the corresponding interspike interval obeying a displaced Poisson curve. The response peak is slightly shifted to the right relative to the stimulus peak because, for small τ , stimuli reach the neuron more frequently when it remains in its refractory period and does not respond.

The neuron response to the stimuli can be quantified by its firing rate F , which is the average number of pulses it produces per time unit, also measured in $(ms)^{-1}$. The microscopic stimulus-response relation can be obtained by plotting the firing rate F as a function of the input spike rate r , exhibiting a sigmoid relation between F and r . For very small input rates ($r \lesssim 0.01$), the response is close to zero; i.e., the minimum firing rate is $F_{min} \approx 0$.

On the other hand, for high input rates ($r \gtrsim 1.0$), the neuron response is affected by the presence of a refractory period immediately after it fires an action potential. Accordingly, there is a maximum firing rate $F_{max} \approx 0.2$, such that we can estimate the refractory period to be $1/F_{max} \approx 5$ *ms*, which compares well to the action potential duration of ~ 1 *ms*. The response interval of a single firing neuron is therefore $0.1 \lesssim r \lesssim 1.0$, and considering that Stevens' law is valid within this interval, we fit a power-law $F \sim r^m$ with an exponent of $m = 0.8$.

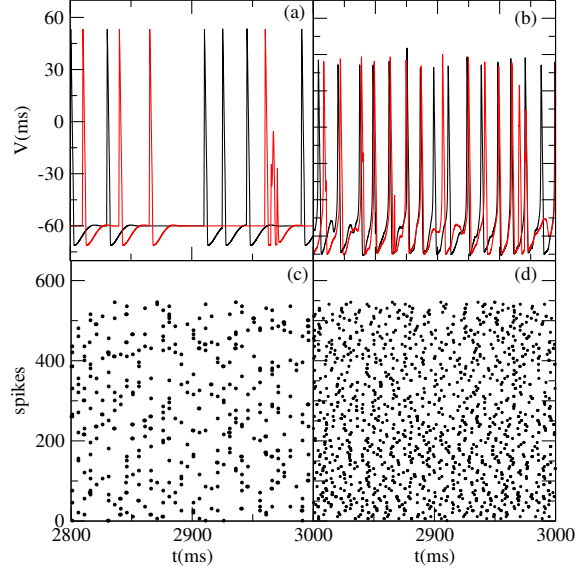


Fig. 2 Time evolution of the membrane potential for a selected number of neurons when (a) $\bar{g}_c = 0$ and (b) $\bar{g}_c = 0.05 \text{ mS/cm}^2$. (c) and (d) are rasterplots indicating the spiking neurons, corresponding to (a) and (b), respectively.

In order to find an adequate window for which Stevens' power-law holds, we consider upper and lower bounds of response given by $F_{upper} = 0.9(F_{max} - F_{min})$ and $F_{lower} = 0.1(F_{max} - F_{min})$, respectively. Let us denote by r_{upper} and r_{lower} the input spiking rates corresponding to F_{upper} and F_{lower} , respectively. The dynamic range, measured in decibels, is defined as:

$$\Delta = (10 \text{ dB}) \log_{10} \left(\frac{r_{upper}}{r_{lower}} \right). \quad (17)$$

4 Stimulus-response relation for coupled neurons

We now consider scale-free networks of coupled HH neurons, taking the maximum specific conductance $\bar{g}_c$ as the parameter describing coupling strength. Indeed, switching off the coupling ($\bar{g}_c = 0$), the neurons exhibit uncorrelated firing [the time series of the membrane potential of two selected neurons is shown in Fig. 2(a)], which can also be observed by considering the corresponding raster plot [Fig. 2(c)], in which we plot the time instants when the neurons fire a peak. Each time, a few dozen neurons spike in an incoherent fashion. As the coupling strength among the neurons increases,

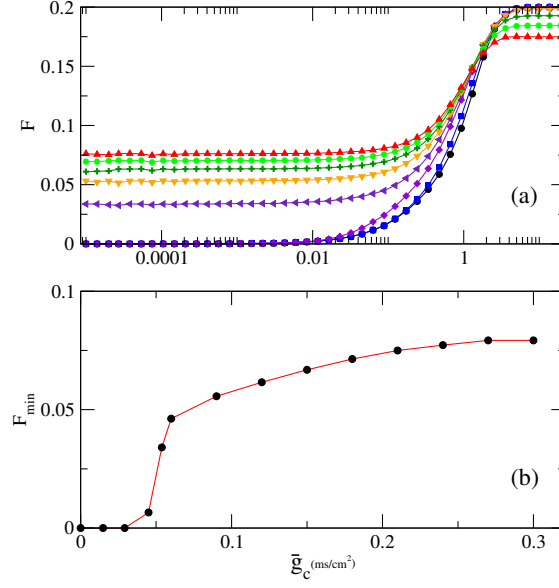


Fig. 3 (a) Firing rate vs. spike input rate for various values of the coupling strength $\bar{g}_c$, from 0.0 (black line with circles) to 0.13 mS/cm^2 (black lines with triangles up). (b) Minimum firing rate as a function of the coupling strength for $N = 550$ neurons.

there is a tendency for them to fire at approximately equal time instants [Fig. 2(b)], producing correlated firing activity in the corresponding raster graph, forming a kind of “firing wave” that propagates along the network [Fig. 2(d)].

The input spikes are applied to all neurons at equal time intervals, such that the input spiking rate is the same for all neurons. The response is again measured by the neuron firing rates. Just like the isolated neurons, the coupled ones exhibit a stimulus-response sigmoidal curve, as illustrated in [Fig. 3(a)], where each curve is obtained using a different value of the coupling intensity $\bar{g}_c$.

The minimum response firing rate increases monotonically from zero (for vanishing coupling) to ~ 0.075 for $\bar{g}_c = 0.3$, showing an evolution similar to a phase transition of the second kind: its value remains close to zero until $\bar{g}_c$ reaches a critical value ~ 0.05 [Fig. 3(b)]. On the other hand, The maximum value of the firing rate (F_{max}), which is 0.2 for uncoupled neurons, decreases with the coupling strength until $F_{max} \sim 0.17$ for $\bar{g}_c = 0.3$.

The dependence of the dynamical range on the coupling strength is depicted in Fig. 4(a). While for uncoupled neurons, the dynamic range is slightly greater than 10 dB . As the coupling strength $\bar{g}_c$ increases, the dynamic range widens until, for a given value of $\bar{g}_c \approx 0.03 mS/cm^2$, the dynamical range achieves a maximum value of

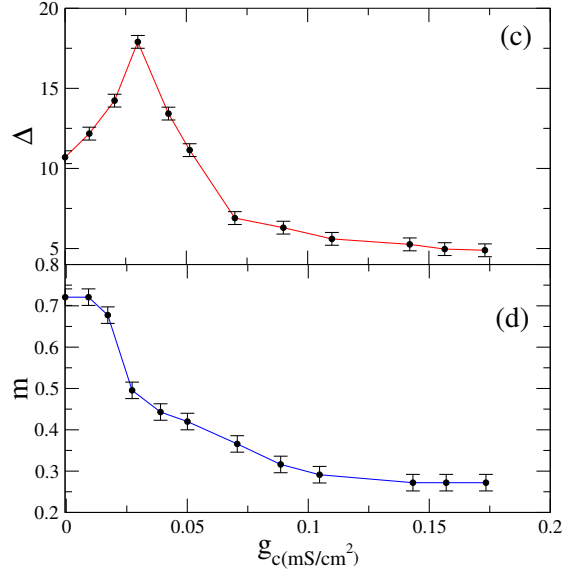


Fig. 4 (a) Dynamic range and (b) exponent of the scaling law $F \sim r^m$ for a scale-free network as a function of the coupling strength.

~ 17 dB and decreases monotonically afterward to values even lower than those for the uncoupled neurons. Moreover, following the values of the power-law exponent m , we see a monotonic decrease from ~ 0.72 for uncoupled neurons to 0.3 for strongly coupled ones [Fig. 4(b)]. It is remarkable that exactly at the point $\bar{g}_c$ where the dynamic range is maximum, the power-law exponent is 0.5. These two results provide additional support for the hypothesis proposed by Kinouchi and Copelli that there is optimization of dynamic range at criticality [13].

The results presented so far refer to relatively small networks with $N = 550$ neurons. It is thus important to investigate the effect of increasing the network size. The variation of the dynamic range with the network size is shown in [Fig. 5(a)]. The dynamic range grows monotonically with N from 12 dB, which is the value for a single neuron, to a saturation value close to 22 dB for large networks. Therefore, the dynamic range increases with the network size but rapidly saturates after *circa* $N = 300$; hence, it could be reasonable to speculate that it becomes size-independent and applicable to networks of neuroscientific interest. Figure 5(b) shows the variation of the scaling exponent m with the number of neurons N . It can be seen that m , which is ~ 0.83 for a single neuron, decreases with N , so that for larger networks, the exponent saturates at ~ 0.28 and appears to remain so, similarly to what occurs for the dynamic range itself.

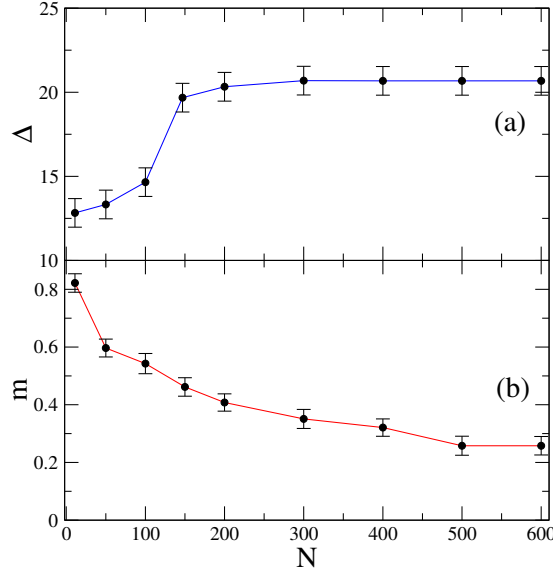


Fig. 5 (a) Dynamic range and (b) exponent of the scaling law $F \sim r^m$ for a scale-free network as a function of the network size, for $\bar{g}_c = 0.05 \text{ mS/cm}^2$.

5 Conclusions

We investigated the dynamic range of a neural system using a scale-free network architecture. We consider Stevens' law, which establishes a power relationship between stimuli and responses, to be valid at different levels of analysis, from the isolated neuron, which in neural networks expands the dynamic range due to the collaboration between neurons. Our main goal was to investigate the factors influencing the dynamic range and scale exposure using a neural network model composed of neurons described by Hodgkin-Huxley equations and connected by chemical synapses within a scale-free network. We observed a sigmoidal curve-shaped relationship between the intensity of stimuli and the system's response. We observed that the dynamic range increases as the number of neurons increases, going from about 11 *dB* for isolated neurons to approximately 22 *dB* in larger networks. Furthermore, the scaling exponent decreases with network size and stabilizes around 0.28.

For a given value of the variable parameter, we observed that the dynamic range achieves a maximum value, and exactly for that value, the exponent in the corresponding power-law is 0.5, within the numerical accuracy, confirming earlier claims that the optimal enhancement of dynamic range is related to the criticality of the network. In

fact, it has been claimed that the optimal functioning of the brain is a consequence of its critical behavior, according to Chialvo and his coworkers [14].

In a previous study, we considered an analogous problem but used a small-world network instead of a scale-free one [16]. Our results suggest a qualitative similarity between these two network connection architectures. However, there are significant differences. Firstly, for strong coupling, the spike rate converges to a single value for small-world networks, whereas the convergence is towards different values in scale-free networks. Secondly, for strong coupling, the dynamical range decreases to values around $\delta \sim 16.5$ for small-world networks and to $\delta \sim 5.0$ for scale-free ones. This last result probably arises from the hub structure of scale-free networks, where a small number of neurons are highly connected, while most of the remaining neurons are poorly connected.

Declarations

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- Author contribution: R.V. initiated and supervised this article. C.B. and R.V. wrote the entire manuscript. C.B. and R.V. conducted the literature search. All authors were also involved in the editing of the manuscript. C.B. ran the numerical simulations and prepared the illustrations.

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