Noise-induced artificial intelligence

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We show that unavoidable stochastic fluctuations are not only affecting information processing in a destructive or constructive way, but may even induce conditions necessary for the artificial intelligence itself. In this proof-of-principle paper we consider a model of a neuron-astrocyte network under the influence of multiplicative noise and show that information encoding (loading, storage, and retrieval of information patterns), one of the paradigmatic signatures of intelligent systems, can be induced by stochastic influence and astrocytes. Hence, astrocytes, recently proved to play an important role in memory and cognitive processing in mammalian brains, may play also an important role in the generation of a system's features providing artificial intelligence functions. Hence, one could conclude that intrinsic stochasticity is probably positively utilized by brains, not only to optimize the signal response but also to induce intelligence itself, and one of the key roles, played by astrocytes in information processing, could be dealing with noises.

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I. INTRODUCTION

Our brain has to operate under very noisy conditions. There are random intrinsic fluctuations [1], and variability because of a single-neuron-specific response [2]. There are even no identic neurons from a genetic point of view [3]. The numbers of vesicles transmitted from terminal to terminal and through special channels variate a lot, and, finally, there is intrinsic stochasticity in any gene expression [4] linked to the neuron's function. Some noise can be theoretically avoided or reduced, such as noise from the surroundings, e.g., noise from the street, but some fluctuations are intrinsic and cannot be avoided in principle. Hence, mammalian brains, showing intelligence and consciousness, have to be evolved under stochastic conditions, and, naturally, a question arises as to what the role of stochasticity and variability in the function and evolution of brains was.

Recently, it was shown that noise can play a constructive role in the dynamics of complex systems, to mention well-known stochastic resonance [5], coherence resonance [6], noise-induced transitions [7], and noise-induced transport [8] in ratchets. Moreover, it was shown that not only time-dependent noise, but a heterogeneity can play an optimizing role in complex networks [9]. Hence, a natural question arises, what is the role of noise in the functioning of neuron-astrocyte networks, and whether noise can not only optimize the signal processing but induce information encoding itself. As an example of this process, we consider a generic example of information pattern storage and recognition. In this proof-of-principle paper, we study a paradigmatic model of a neuron-astrocyte network and show that multiplicative noise of a specific form can induce an ability of a system to form short-term memory. Moreover, we show that astrocytes, these local and tentative integrators of neuronal activity [10–12], play an important role in this ability, hence, probably explaining one of the astrocyte's functional key roles [13]. Recently, using computational models we have shown that astrocyte-induced spatial synchronization in neuronal ensembles [14–19] plays a crucial role in information processing in the brain, organizing short-term working memory [20,21],

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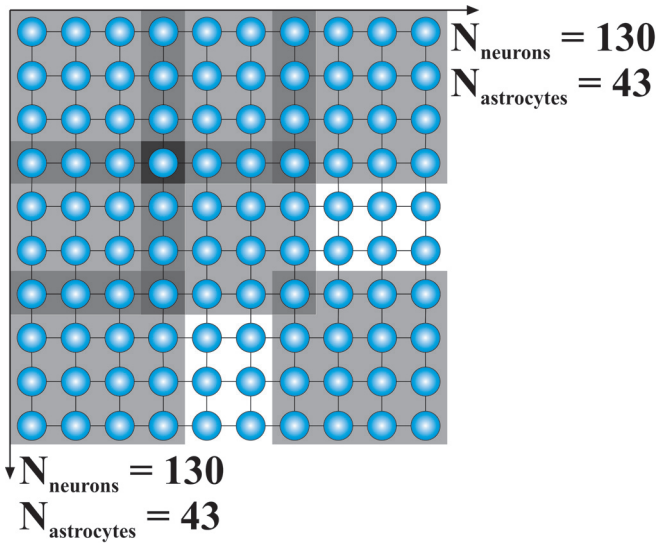

FIG. 1. Network structure. Blue nodes stand for neurons and square gray fields for astrocytes which cover uniformly all neurons. The gray fields, representing astrocytes, overlap by one neuron-wide layer.

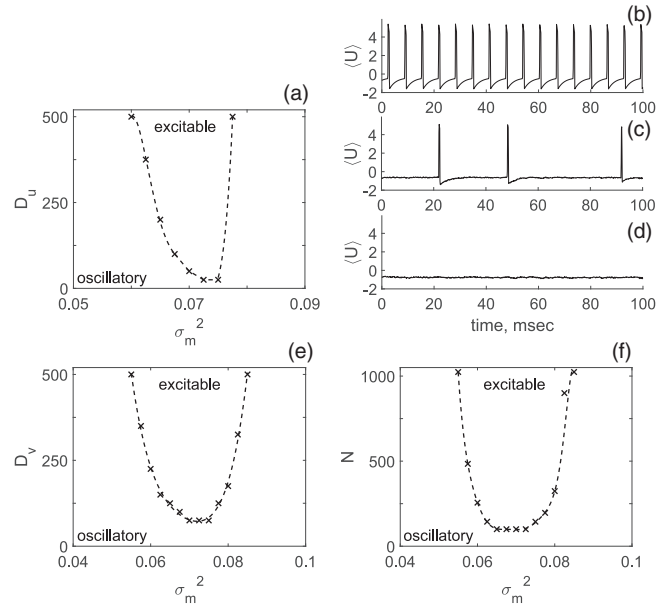

FIG. 2. Phase diagrams for the transition from a self-sustained oscillatory regime to noise-induced excitability. (a) Coupling strength D_u versus multiplicative noise intensity σ_m^2 for 400 coupled elements, $D_v = 100$. (e) Coupling strength D_v versus multiplicative noise intensity σ_m^2 for 400 coupled elements, $D_u = 100$. (f) Number of coupled elements N versus multiplicative noise intensity σ_m^2 for $D_u = 300$, $D_v = 300$. Time series of the mean field of the fast variable U with increasing multiplicative noise intensity: (b) $\sigma_m^2 = 0.0$, (c) $\sigma_m^2 = 0.065$, (d) $\sigma_m^2 = 0.09$.

and, even, mediating analogous memory in multilayer spiking neuron networks [22]. Naturally, a question has arisen, could these manifestations of artificial intelligence appear, if a spiking regime exists because the excitability of neurons is induced by noise.

II. METHODS AND MODEL

The proposed neuron-astrocyte network consists of two components; the first is the network of the FitzHugh-Nagumo neurons (FHNs) with a dimension 130×130, and the second network is a network of astrocytes with a dimension 43×43. Both networks overlap each other organizing one multilayer network [23]. Taking the FHN as a model of a neuron is not so important, but it is crucial that excitability is supported by multiplicative noise. Fully in correspondence with recent studies [13], astrocytes actively participate in information processing in the brain by releasing gliotransmitters (e.g., glutamate) that influence neuronal excitability and synaptic transmissions [24], thus forming a bidirectional communication between the neuron and the astrocyte layer. Each astrocyte interacts with a 4×4 neuronal ensemble with one overlapping row, to allow communications between astrocytes (Fig. 1). In response to neuronal activity, astrocytes emerge with the elevations of intracellular Ca^{2+}. When the concentration of Ca^{2+} in astrocyte exceeds the threshold, it releases gliotransmitters that may affect the neuronal excitability. A detailed description of the neuron-astrocyte network model is presented in the Appendix.

III. RESULTS

Without noise, each neuron will oscillate, and these oscillations will be soon synchronized via coupling. Noise-induced excitability means that in the presence of optimal intensity of multiplicative noise and coupling, oscillations of the neuronal layer are suppressed as a result of a noise-induced phase transition (Fig. 2). Figures 2(b)–2(d) display the time series of the activator's mean field, $\langle U \rangle = \frac{1}{N}\sum_{i=1}^{N} U_i$, for a system of 400 coupled elements, with the coupling strengths $D_u = 100$ and $D_v = 100$. The increase in multiplicative noise intensity σ_m^2 leads to an increase in the time interval between consecutive spikes. For sufficiently large noise intensity, no spikes appear. This corresponds to the suppression of oscillations due to multiplicative noise and coupling.

Figures 2(a) and 2(e) depict a phase diagram in the plane of parameters D_u-σ_m^2 (a) and D_v-σ_m^2 (e) distinguishing the regions where the original oscillatory behavior and the noise-induced excitability regime exist. Figure 2(f) displays the dependence of the transition to noise-induced excitability on the number of coupled elements of the system. The region of noise intensity values for which noise-induced excitability exists becomes larger as the number of elements increases. The quantitative description of the transition from oscillatory to excitable state depends on the minimum number of coupled elements required for a transition (approximately 100) and the coupling strength between the elements [25].

Noise-induced excitability occurs as a result of a second-order phase transition and, hence, is possible only due to noise and coupling, but, surprisingly, after the transition, the neuron network is capable of exhibiting propagation of excitable structures, as it allows the propagation of the plane wave front and spiral waves [25]. Noise-induced phase transitions of this kind have been first reported in [26]. A mechanism is not linked with timely dynamics of noise like in stochastic

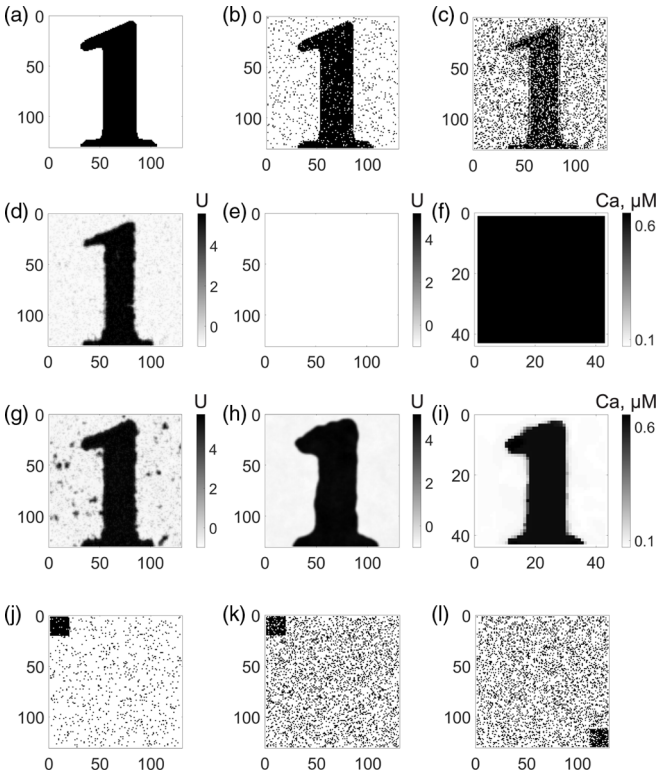

FIG. 3. Input patterns: (a) without noise, (b) training sample with 10% salt and pepper noise, (c) testing sample with 40% salt and pepper noise. Noise-induced neuron-astrocyte network response and short-time associative memory [(d)–(i)]. First without multiplicative noise: $\sigma_m^2 = 0.0$ [(d)–(f)] and then with multiplicative noise $\sigma_m^2 = 0.09$ [(g)–(i)]. Here (d), (e) neuronal network response during and after testing, respectively, (f) the intracellular Ca^{2+} concentration in astrocytes without noise, and (g), (h) neuronal network response during and after testing, (i) the intracellular Ca^{2+} concentration in astrocytes with noise. It is clearly seen that the presence of multiplicative noise makes a response to the input pattern possible: compare (e) and (h). Examples of not overlapping input patterns: (j) training sample with 10% salt and pepper noise, (k), (l) testing samples with 40% salt and pepper noise.

resonance. A combined effect of multiplicative and additive noise on such systems has been investigated in [27,28]. Note that also many other noise-induced stabilization effects are possible in biological systems, see, e.g., [29,30], including noise-induced synchronization of uncoupled excitable systems [31]. We found that the noise-induced excitability is preserved under the influence of astrocytes; hence, the naturally appearing question was whether astrocytes may help process information if overlapped over a network with noise-induced excitability as they do if overlapped over a network of excitable elements.

Next, we show how this system responds to an input signal. As an example, the input signal was a binary image of digit 1 with a size of 130×130 pixels [see Fig. 3(a)]. During training, the network is presented with 5 instances of an image with 10% salt and pepper noise [Fig. 3(b)] for 5 msec, and with a period of 100 msec between each instance.

The input image is first processed by the neuronal network that obtains the applied current I_{app} in Eq. (A1) and is further

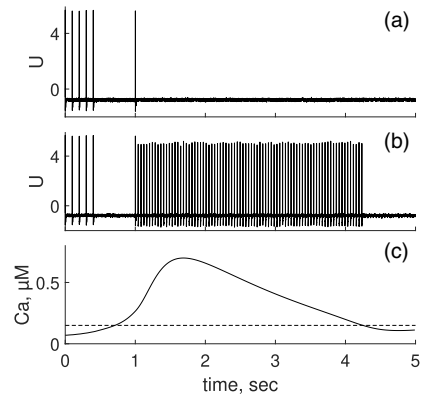

FIG. 4. Membrane potential of neurons in target pattern during and after training without (a) and with (b) astrocytes, $\sigma_m^2 = 0.09$. (c) The intracellular Ca^{2+} concentration in active astrocyte.

converted into spikes. During training, each astrocyte monitors the activity of the associated 16 neurons. If more than half of this neural ensemble were spiking then the astrocyte receives an input signal I_{neuro}, which models the glutamate-induced IP_3 production in response to neural activity, inducing an elevation of the intracellular calcium concentration. The activity of a neuron under the input signal and corresponding astrocyte is shown in Figs. 4(a) and 4(b). Neurons inside the target pattern respond to the input signal and produce a spike. During training, the intracellular Ca^{2+} concentration of astrocyte associated with these active neurons is slowly increasing during the training phase and eventually exceeds the threshold [Fig. 4(c)]. It is important to note that information pattern storage realized by such astrocytic modulation is characterized by one-shot learning and is maintained for the lifetime of the calcium signal in astrocytes (about 3.5 sec). The testing image with 40% salt and pepper noise [Fig. 3(c)] is presented to the network for 10 msec at 1 sec. The increased concentration of Ca^{2+} in the astrocyte is giving feedback to the neurons and causes the neurons in the target area to continue to fire even after the testing image is no longer present as shown in Fig. 4(b). Feedback from the astrocyte to the neuron is switched on if the Ca^{2+} concentration in the astrocyte is above the threshold in 0.15 μM and more than 8 of the associated neurons are still active. This feedback is modeled as an additional current I_{astro} in Eq. (A1), which is determined by its biophysical mechanisms of an astrocyte-mediated increase of postsynaptic neuronal excitability via activation of extrasynaptic NMDAR [32] and other glutamate receptors [33] by the astrocytic gliotransmitter—glutamate.

Without astrocytes, due to the diffusion coupling, the neural network is still able to suppress noise in the input image. But neurons respond to an input signal for a very short period of time, and during training, a neural network without astrocytes cannot store the pattern. Therefore, there is no activity on the neurons after testing. The presence of astrocytes qualitatively changes the situation enabling short-term memory. Indeed, if we analyze the response of the neuron-astrocyte network during and after testing, with significant noise contribution, the neuron-astrocyte network can store the pattern after training for a period of time, which is determined by the

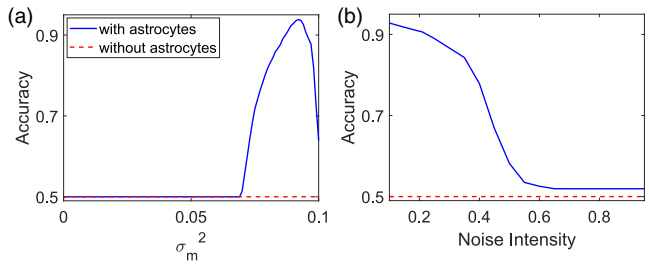

FIG. 5. Noise-induced intelligence. (a) Accuracy against noise intensity in the system. (b) Accuracy against noise intensity in the training sample, $\sigma_m^2 = 0.09$.

duration of the astrocytic calcium pulse [see Figs. 3(g)–3(i)]. Hence, the presence of astrocytes, measured by I_i^{astro}, is absolutely necessary for this kind of noise-induced information encoding. Noise is also a necessary ingredient of the effect because without noise the neuronal layer is in an oscillatory state and the network cannot store the pattern [Figs. 3(d)–3(f)].

Due to the features of the FitzHugh-Nagumo coupled oscillators, a signal is able to propagate throughout the network and decreases the accuracy of retrieval. Hence, the accuracy of the network is different if taken at a different time frame. To measure the memory performance of the system, we calculate the correlation of a recalled pattern with the ideal item in the following way:

$$M_{ij} = I\left[\left(\frac{\sum_{k=t_1}^{t_1+T} I[U_{ij}(k) > \text{thr}_1]}{T}\right) > \text{thr}_2\right], \quad (1)$$

$$A = \frac{1}{2}\left[\frac{1}{|P|}\sum_{(i,j)\in P} M_{ij} + \frac{1}{WH - |P|}\sum_{(i,j)\notin P}(1 - M_{ij})\right], \quad (2)$$

where t_1 is the moment in time when the testing sample is presented, $T = 500$ msec, P is a set of pixels belonging to the ideal pattern, W and H are network dimensions, thr_1 is the spike threshold, thr_2 is the frequency threshold, and I is the indicator function. First, we calculate the frequency of the neuronal network for 500 msec after the testing sample is presented and convert it into a binary image (1 if the frequency of a neuron is greater than 0.0016 Hz, 0 otherwise). Then we compare the input image and output binary signal and calculate the accuracy of the neural network retrieval. If there is no output signal, the accuracy = 0.5. The accuracy is plotted against the multiplicative noise intensity [Fig. 5(a)] and against the salt and pepper noise intensity in the training sample [Fig. 5(b)].

We find that the system with astrocytes performs much better than its counterpart. These plots show that the astrocyte network is indeed assisting the neuronal layer to store the pattern. With increasing the multiplicative noise intensity, the accuracy of the neuron-astrocyte network increases and reaches high values, but it decreases again at significant noise intensity. With increasing salt and pepper noise intensity in the training sample, the accuracy of the neuron-astrocyte network decreases. The system works best at low noise intensity in the training pattern.

To demonstrate the ability of the neuron-astrocyte network to remember the input images, we tested the system with the sample that did not participate in the training, i.e., does not overlap with the training pattern [Fig. 3(j)]. The testing patterns are shown in Figs. 3(k) and 3(l). We calculate the accuracy by comparing the input pattern with the frequency of the neuronal network during 500 msec after the testing sample is presented. If the network is tested by the sample that is shown in Fig. 3(k), the accuracy is 0.994. But if we test the system with the pattern from Fig. 3(l), the accuracy is 0.5; i.e., the network does not respond to the input signal that did not participate in the training.

IV. DISCUSSION

In summary, in this proof-of-principle paper, we have shown that stochastic fluctuations can induce information encoding via noise-induced phase transition in neural networks and a pattern recognition organized by astrocytes. For the study, we have used well-established models of a neuron-astrocyte network, a variation of the model proposed in [20]. Moreover, we have found that, surprisingly, astrocyte signaling plays a very important role in this effect. This could potentially lead to further investigations of the role of astrocytes as cells that help neurons to deal with noise, a functional role that was not previously reported for astrocytes despite recent intensive investigations of these cells' functionality [10]. Integration of astrocytic signaling in cognitive processing has implications for understanding the mechanisms of cognitive impairment [13,34,35].

The code is available at [36].

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APPENDIX: DESCRIPTION OF THE MODEL

The neurons are modeled as a set of N coupled FitzHugh-Nagumo oscillators, where each oscillator is coupled to its nearest neighbor with fixed zero boundary conditions. The dynamic of each neuron is governed by the following equations [25]:

$$\dot{U}_i = \frac{1}{\epsilon}[F(U_i) - V_i] + D_u(\bar{U}_i - U_i) + I_i^{\text{app}} + I_i^{\text{astro}}, \quad (A1)$$

$$\dot{V}_i = cU_i + d + V_i\xi_i + D_v(\bar{V}_i - V_i), \quad (A2)$$

where $\bar{x}_i \equiv \frac{1}{\mathcal{N}}\sum_{j=1}^{\mathcal{N}} x_j$, $x_i = U_i, V_i$ is the mean value at site i by summing over the $\mathcal{N} = 4$ nearest neighbors. I_i^{app} simulates an input signal and is a supra-threshold for the noise-induced excitable system:

$$I_i^{\text{app}} = \begin{cases} 60, & \text{if an input signal is present,} \\ 0, & \text{otherwise.} \end{cases} \quad (A3)$$

TABLE I. Table of parameter values and units.

Parameter	Value	Unit
a	1.0	–
b	2.0	–
c	0.2	–
d	0.075	–
g	0.2	–
ϵ	0.01	–
D_u	100	–
D_v	100	–
c_0	2.0	μM
c_1	0.185	μM
v_1	6.0	s^{-1}
v_2	0.11	s^{-1}
v_3	2.2	μM s^{-1}
v_4	0.3	μM s^{-1}
v_5	0.025	μM s^{-1}
v_6	0.2	μM s^{-1}
k_1	0.5	s^{-1}
k_2	1.0	μM
k_3	0.1	μM
k_4	1.1	μM
d_1	0.13	μM
d_2	1.049	μM
d_3	0.9434	μM
d_5	0.082	μM
IP_{3s}	0.16	μM
$1/\tau_r$	0.14	s^{-1}
α	0.8	–
a_2	0.14	$\mu M^{-1}\, s^{-1}$

I_i^{astro} simulates astrocytic modulation of the synaptic activity:

$$I_i^{\text{astro}} = \begin{cases} 60, & \text{if } [Ca^{2+}] > 0.15\ \mu M, \\ 0, & \text{otherwise.} \end{cases} \tag{A4}$$

The voltage dependence $F(U)$ is given by

$$F(U) = \begin{cases} -1 - U + b, & U \leqslant -\frac{1}{2}, \\ gU + b + \frac{1}{2}(g-1), & -\frac{1}{2} < U < \frac{1}{g} - \frac{1}{2}, \\ +1 - aU + b - \frac{1}{2} + a\left(\frac{1}{g} - \frac{1}{2}\right), & U \geqslant \frac{1}{g} - \frac{1}{2}. \end{cases} \tag{A5}$$

In a neural context, the membrane potential of a neuron is represented by the fast activator variable U, as indicated by the small timescale-ratio parameter ϵ, and V is associated with the time-dependent conductance of the potassium channels in the membrane [37]. ξ_i is the δ-correlated multiplicative Gaussian noise representing random fluctuations with zero mean and a correlation $\langle \xi_i(t)\xi_j(t')\rangle = \sigma_m^2 \delta(t-t')\delta_{i,j}$, and is interpreted in the Stratonovich sense [38].

The astrocytic activity is determined by the dynamics of intracellular Ca^{2+} concentration, $[Ca^{2+}]$, which is controlled by the intracellular concentration of IP_3, $[IP_3]$, and by the fraction of calcium channels on the membrane of the intracellular calcium store—the endoplasmic reticulum—that are in the open (non-inactivated) state, h. To describe the dynamics of the intracellular Ca^{2+} in each astrocyte (j) of our network, we used the well-established biophysical Li-Rinzel model [39], which qualitatively reflects the main features of the calcium dynamics of astrocytes (for more details about this model and the biophysical meaning of all flows and parameters, see [39]). This model consists of the following differential equations:

$$[\dot{Ca^{2+}}]_j = I_{er} - I_{\text{pump}} + I_{\text{leak}} + I_{\text{in}} - I_{\text{out}}, \tag{A6}$$

$$[\dot{IP_3}]_j = \frac{[IP_3]_S - [IP_3]}{\tau_r} + I_{plc} + I_j^{\text{neuro}}, \tag{A7}$$

$$\dot{h}_j = \frac{H - h}{\tau_n}, \tag{A8}$$

where

$$I_{er} = c_1 v_1 \left(\frac{[IP_3]}{[IP_3] + d_1}\right)^3 \left(\frac{[Ca^{2+}]}{[Ca^{2+}] + d_5}\right)^3 \times h^3 \left(\frac{c_0 - [Ca^{2+}]}{c_1} - [Ca^{2+}]\right), \tag{A9}$$

$$I_{\text{leak}} = c_1 v_2 \left(\frac{c_0 - [Ca^{2+}]}{c_1} - [Ca^{2+}]\right), \tag{A10}$$

$$I_{\text{pump}} = v_3 \frac{[Ca^{2+}]^2}{[Ca^{2+}]^2 + k_3^2}, \tag{A11}$$

$$I_{\text{in}} = v_5 + v_6 \frac{[IP_3]^2}{k_2^2 + [IP_3]^2}, \tag{A12}$$

$$I_{\text{out}} = k_1 [Ca^{2+}], \tag{A13}$$

$$H = \left(d_2 \frac{[IP_3] + d_1}{[IP_3] + d_3}\right) \Big/ \left(d_2 \frac{[IP_3] + d_1}{[IP_3] + d_3} + [Ca^{2+}]\right), \tag{A14}$$

$$\tau_n = \left[a_2 \left(d_2 \frac{[IP_3] + d_1}{[IP_3] + d_3} + [Ca^{2+}]\right)\right]^{-1}, \tag{A15}$$

$$I_{plc} = v_4 \frac{[Ca^{2+}] + (1-\alpha)k_4}{[Ca^{2+}] + k_4}, \tag{A16}$$

and $I_j^{\text{neuro}} = 1$, if more than 50% of the neurons, interacting with this astrocyte, are spiking and the average spiking frequency in the neural ensemble is more than 25 Hz. The parameters of Eqs. (A6)–(A8) are determined experimentally and the biophysical meaning of all parameters can be found in [25,39]. For this experiment, the values of parameters are listed in Table I.

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