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Cooperative activation of sodium channels for downgrading the energy efficiency in neuronal information processing

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The Hodgkin–Huxley model assumes independent ion channel activation, although mutual interactions are common in biological systems. This raises the problem why neurons would favor independent over cooperative channel activation. In this study, we evaluate how cooperative activation of sodium channels affects the neuron's information processing and energy consumption. Simulations of the stochastic Hodgkin–Huxley model with cooperative activation of sodium channels show that, while cooperative activation enhances neuronal information processing capacity, it greatly increases the neuron's energy consumption. As a result, cooperative activation of sodium channel degrades the energy efficiency for neuronal information processing. This discovery improves our understanding of the design principles for neural systems, and may provide insights into future designs of the neuromorphic computing devices as well as systematic understanding of pathological mechanisms for neural diseases.

Keywords: energy efficiency, ion channel noise, action potential generation, neuronal dynamics

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Neural information transmission relies on action potentials (APs), which are generated by inward Na^+ and outward K^+ currents through coordinated opening and closing of ion channels.[1] The mechanism for generation of AP is established by the Hodgkin–Huxley (HH) model.[2] However, the classic HH model was established on several assumptions.[3] For example, it assumes an infinite number of ion channels so that the fluctuations in membrane currents caused by stochastic channel gating can be averaged out, leading to the deterministic equations. The limitation due to the assumption of infinite number of channels has been addressed by the stochastic version of the HH models that account for channel noise effects of finite membranes area.[4,5] The classic HH model also assumes that ion channels in the membrane are independent, with no direct interactions among them. This assumption was challenged recently.[6] It was argued that features of cortical neuron AP initiation, like rapid onset and variable onset potential, require cooperative channel interactions beyond the classic HH framework.[6] On the other hand, a model with cooperative sodium channel activation could reproduce these dynamics, suggesting that independent channel assumptions of the classic HH model may need revising. However, subsequent work indicates that these unusual initiation features can be explained by the back propagation of the APs initiated in the axon initial segment. Furthermore, these unusual features can be simulated with the classic HH model when the spatial geometry and spike initiation properties of cortical neurons are taken into account.[7] Thus, cortical spike generation largely agrees with the classic HH theory.[8] While this finding supports the assumption of independence activation of channels, questions remain on why neurons would favor independent over cooperative channel activation, even though mutual interaction and cooperation pervades all levels of the living world.

Evolutionary pressures may have optimized neural systems to utilize energy very efficiently for information transmission and processing.[9] Signatures of such optimization for energy efficiency in brains have been identified across multiple scales, ranging from the kinetics of individual ion channels, to the sparse coding of information and low baseline firing rates in cortical networks, to the economical wiring between brain regions, and the shift between rest and task states in functional connectivity maps during cognitive tasks.[9–11] Therefore, as a fundamental principle, energy efficiency has provided a powerful lens for understanding design and function of neural systems. We conjecture that this principle may also help us to understand the puzzling for lacking interactions among ion channels.

To this end, we first build a stochastic version of the HH neuron model with independent ion channel kinetics.[12] According to the electrical equivalent circuit for neurons with limited number of ion channels [Fig. 1(a)], the membrane potential evolution is governed by the following equation:

$$C_{\rm m}\frac{{\rm d}V}{{\rm d}t} = -[G_{\rm K}(V-V_{\rm K}) + G_{\rm Na}(V-V_{\rm Na}) + G_{\rm L}(V-V_{\rm L})] + I_{\rm sti}, \quad (1)$$

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where V is the membrane potential and $I_{\rm sti}$ the input current. $V_{\rm K}$, $V_{\rm Na}$, and $V_{\rm L}$ are the reversal potentials of the potassium, sodium, and leakage currents, respectively. $G_{\rm Na}$, $G_{\rm K}$, and $G_{\rm L}$ are the corresponding specific ion conductance, and $C_{\rm m}$ is the specific membrane capacitance. Assuming that all gates in channels open and close according to a two-state Markov process with voltage dependent opening and closing rates as demonstrated in Fig. 1(b), where the transition rates α and β are the opening and closing rates of the gates. Then the conductance for potassium and the sodium channel are given by

$$G_{\rm K}(V,t) = \gamma_{\rm K}[{\rm K}_{\rm open}]/S, \tag{2}$$

$$G_{\rm Na}(V,t) = \gamma_{\rm Na}[{\rm Na}_{\rm open}]/S, \tag{3}$$

where $\gamma_{\rm K}$ and $\gamma_{\rm Na}$ are the single-channel conductance of the potassium and sodium channels. $[{\rm K}_{\rm open}]$ refers to the number of open potassium channels and $[{\rm Na}_{\rm open}]$ refers to the number of open sodium channels at moment t. S is the membrane area of the neuron. Thus the total numbers of sodium channels and potassium channels are determined by $\rho_{\rm Na}S$ and $\rho_{\rm K}S$, where $\rho_{\rm Na}$ and $\rho_{\rm K}$ are the densities of sodium and potassium channels, respectively. In this study, we use the pulse train inputs with the pulse strength of I_0 and interpulse interval of 50 ms. All the parameters mentioned above are demonstrated in Table 1. By simulating the random gating of each ion channels in the membrane, we track the state of each gate at every moment and calculate the numbers of open sodium and potassium channels. Eventually we integrate Eq. (1) by the Euler-forward method with a time step of 0.01 ms.

Table 1. Parameters and rate functions used in the simulations.

$C_{\rm m}$	specific membrane capacitance	1 μF/cm^2
$V_{\rm K}$	potassium reversal potential	−77 mV
$V_{\rm Na}$	sodium reversal potential	50 mV
$V_{\rm L}$	leakage reversal potential	−54.4 mV
$\gamma_{\rm K}$	potassium channel conductance	20 pS
$\gamma_{\rm Na}$	sodium channel conductance	20 pS
$G_{\rm L}$	leakage conductance	0.3 mS/cm^2
$\rho_{\rm K}$	potassium channel density	20/μm^2
$\rho_{\rm Na}$	sodium channel density	60/μm^2
J	potential drift intensity	3.2 mV
α_n		$\frac{0.01(V+55)}{1-{\rm e}^{-(V+55)/10}}$
β_n		$0.125\,{\rm e}^{-(V+65)/80}$
α_m		$\frac{0.1(V+40)}{1-{\rm e}^{-(V+40)/10}}$
β_m		$4\,{\rm e}^{-(V+65)/18}$
α_h		$0.07\,{\rm e}^{-(V+65)/20}$
β_h		$\frac{1}{1+{\rm e}^{-(V+35)/10}}$

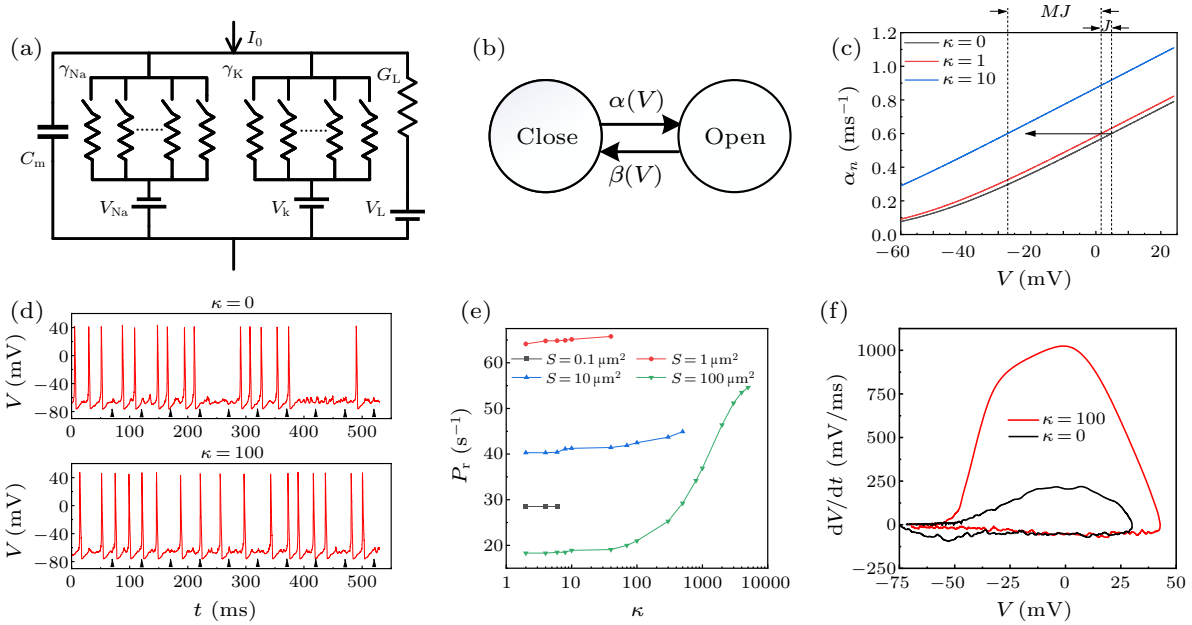


Fig. 1. Stochastic version of the HH model with or without channel coupling. (a) Electrical equivalent circuit of the HH model with consideration of each ion channel conductance. (b) Kinetics scheme of a two-state ion channel gating. (c) Modeling inter-channel cooperativity: In the cooperative activation model, the opening of neighboring channels shifts the single channel activation curve to more hyperpolarized potentials such that the transition rate of channels at a given membrane potential is increased. M ($\leq \kappa$) is the number of opening neighboring channels, and $M = 10$ in this plot. (d) Demonstration of APs of stochastic HH models with membrane area $S = 10$ cm^2 versus time under a train of pulse stimulation (marked with black triangles above the horizontal axis, pulse strength: $I_0 = 3.0$ μA/cm^2; duration: $\Delta t = 1.0$ ms; the inter-pulse-interval is 50 ms). Top panel: independent activation, $\kappa = 0$; bottom panel: cooperative activation, $\kappa = 100$. (e) The spontaneous firing rate of the stochastic HH models versus number of coupled channels κ for neurons with different membrane area. $I_0 = 0.0$ μA/cm^2. Note that κ is limited by the number of available channels, i.e., the size of membrane area. (f) Phase plot (dV/dt versus V) of the AP for the stochastic HH models with independent or cooperative activation of channels. For this plot, $S = 100$ cm^2.

Next, we introduce cooperative activation of sodium channels into the above stochastic HH model, as proposed by Naundorf *et al.*[6] In the cooperative activation model, an individual channel i is coupled to κ neighboring channels such that the opening of each of them shifts the activation curve of channel i by a voltage shift J towards more hyperpolarized values [Fig. 1(c)]. With $J = 0$ mV, the model describes statistically independent single channel activation. With $J > 0$ mV, chan-

nels are activated in a cooperative fashion. Then the activation and deactivation rates of channel i are given by

$$\alpha_i(V) = \alpha(V + \sum_j J_{ij}\sigma_j), \tag{4}$$

$$\beta_i(V) = \beta(V + \sum_j J_{ij}\sigma_j), \tag{5}$$

where $J_{ij} = J$ if channel i and j are coupled, otherwise $J_{ij} = 0$; and σ_j is a binary single channel state variable labeling channels in the open state: if channel j is open, $\sigma_j = 1$, otherwise $\sigma_j = 0$. Figure 1(d) demonstrates both the firing activities of the stochastic HH neuron with independent or cooperative activation of sodium ion channels in response to the identical pulse input trains. In the absent of external stimuli, the stochastic HH neurons would generate APs due to the fluctuations caused by the ion channel. We calculate P_r, the spontaneous firing rate that measures the number of spontaneously fired APs per second. As shown in Fig. 1(e), P_r peaks at the medium size of the membrane area (please see Fig. 4 and the explanation for the mechanism in Ref. [12]). As the number of coupled ion channels increases, the spontaneous firing rate increases as well. However, because of the limited number of available ion channels, the increase is limited for neurons with small membrane area. Figure 1(f) demonstrates the phase plots that graph the rate of change of the membrane potential dV/dt against the instantaneous membrane potential $V(t)$ during AP for both kinds of neurons. It is seen that both kinds of neurons reach the maximal velocity when the membrane potential is about 0 mV, while neurons with cooperative activation ($\kappa = 100$) show a very abrupt onset and a steep upstroke in membrane potential, and its maximal velocity is about 4 times larger than that of neurons with independent activation, suggesting that the former exhibits more rapid initiation of AP than the latter, as reported previously in Ref. [6].

We use the "direct method" to measure the entropy rate of the spike sequences in response to a specific stimulus.[13] First, we divide the spike sequence in response to set stimulus into bins of size $\Delta\tau$ with value 1 (AP) or 0 (no AP). Then we divide the train of bins into $T/\Delta\tau$ words. Considering the size of data set, the total entropy rate is calculated as

$$H_{\text{total}} = -\lim_{T\to\infty}\frac{1}{T}\sum_i P_i \log_2 P_i, \tag{6}$$

where P_i represents the probability of the i-th word. The total entropy rate measures how much information is encoded in the spike sequence of the neuron in response to the inputs. We also use the noise entropy to evaluate how much of the fine structure of the spike sequence is just noise:

$$H_{\text{noise}} = \langle -\lim_{T\to\infty}\frac{1}{T}\sum_i P_i(t)\log_2 P_i(t)\rangle_t. \tag{7}$$

The mutual information that is the difference between the total entropy and the noise entropy is used to measure how much information in the input pulse trains is encoded in the output spike t sequences of the neurons, i.e.,

$$H_{\text{info}} = H_{\text{total}} - H_{\text{noise}}. \tag{8}$$

In the calculation, the bin size $\Delta\tau$ is chosen to be 1 ms, which is small enough to keep the fine temporal structure of the spike sequences within the word size used, and yet large enough to avoid undersampling problems.

It is seen from Figs. 2(a)–2(d) that for inputs with different strengths, the total entropy and noise entropy decrease as the membrane area increases, but the mutual information is maximized around 200 μm^2. The neurons receiving stronger inputs would generate spike sequences with higher total entropy and noise entropy. They also yield spike sequences with higher mutual information, implying that the stronger inputs would make the neurons encoding more information embedded in the input pulse trains. What's more, the neurons with cooperative activation of channels exhibits increased total entropy, noise entropy, as well as the mutual information, suggesting that the cooperative activation of channels in neurons has the ability to enhance the neuronal information transmission capacity. If so, the question is: Why hasn't the neurons evolved such a mechanism?

To answer the above question, we move on to investigate how the cooperative activation of channels affects the energy efficiency of neurons in information processing. Energy plays an important role in neuronal functions.[14] In literature, there are several methods to calculate the energy consumption of spiking activities from the neuron models. One method is to estimate the total ions from the sodium influx, which is determined by integrating the sodium current during the period.[15] The alternative method to calculate the energy cost of the electrical equivalent circuit of the HH model.[13] In this study, we calculate the energy consumed by the ion channels from the electrical equivalent circuit of the stochastic HH models [Fig. 1(a)]. The electrical energy stored within a neuron with unit membrane area is accumulated in the membrane capacitor and equivalent "batteries" generated by the Nernst potentials of ions. The total energy accumulated at a given time point is[13]

$$E(t) = \frac{1}{2}C_m V^2 + E_{Na} + E_K + E_L, \tag{9}$$

where C_m and V represent the membrane capacitance and membrane potential, and the other three terms are the respective energies of the Na, K, and Cl ion channels in the battery, respectively. Hence the total derivative with respect to time of the above total energy is

$$\frac{dE(t)}{dt} = C_m V\frac{dV}{dt} + G_{Na}(V - V_{Na})V_{Na} + G_K(V - V_K)V_K + G_L(V - V_L)V_L. \tag{10}$$

Substituting Eq. (1) into Eq. (10) yields

$$\frac{dE(t)}{dt} = VI_{sti} - G_{Na}(V - V_{Na})^2$$

$$-G_{\rm K}(V-V_{\rm K})^2-G_{\rm L}(V-V_{\rm L})^2. \quad (11)$$

Then by integrating this equation over a specific time interval from T_0 to T_0+T, and then dividing by the entire duration T, we could obtain the average energy per second consumed by ion channels in a neuron with unit membrane area as[16]

$$E'_{\rm c}=\frac{1}{T}\int_{T_0}^{T_0+T}[G_{\rm Na}(V-V_{\rm Na})^2+G_{\rm K}(V-V_{\rm K})^2+G_{\rm L}(V-V_{\rm L})^2]{\rm d}t. \quad (12)$$

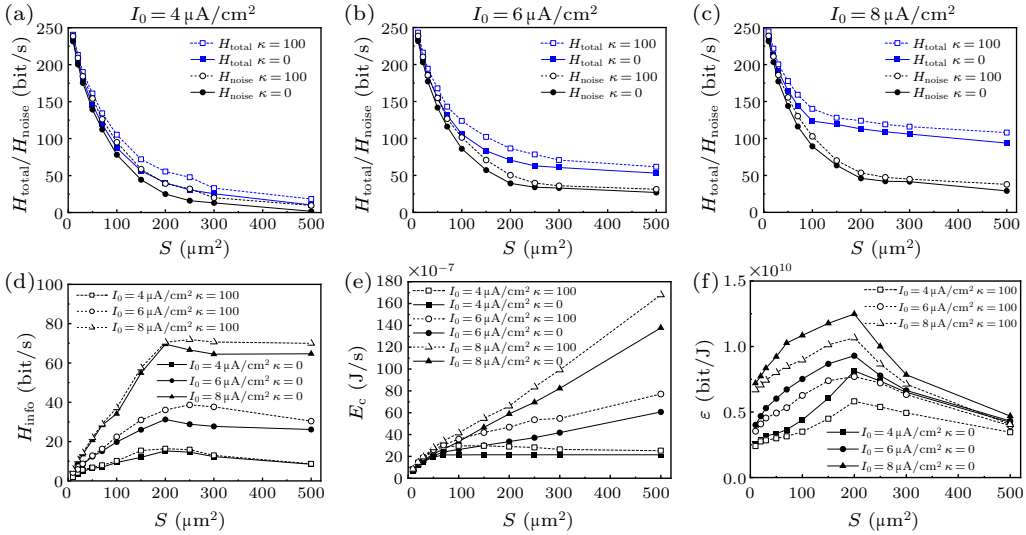


Fig. 2. Information transmission capacity, energy cost, and energy efficiency of the stochastic HH model with independent or cooperative activation. (a)–(c) The total and noise entropy rates of the stochastic HH model in response to pulse inputs as a function of membrane area in the case of independent or cooperative activation of sodium channels: (a) $I_0=4.0$ μA/cm^2, (b) $I_0=6.0$ μA/cm^2, (c) $I_0=8.0$ μA/cm^2. (d) Mutual information rate of the stochastic HH model in response to pulse inputs as a function of membrane area in the case of independent or cooperative activation of sodium channels. (e) The energy cost versus the membrane area under different pulse inputs for stochastic HH neurons with independent or cooperative activation of sodium channels. (f) The energy efficiency in coding pulse signals versus the membrane area under different pulse inputs for stochastic HH neurons with independent or cooperative activation of sodium channels.

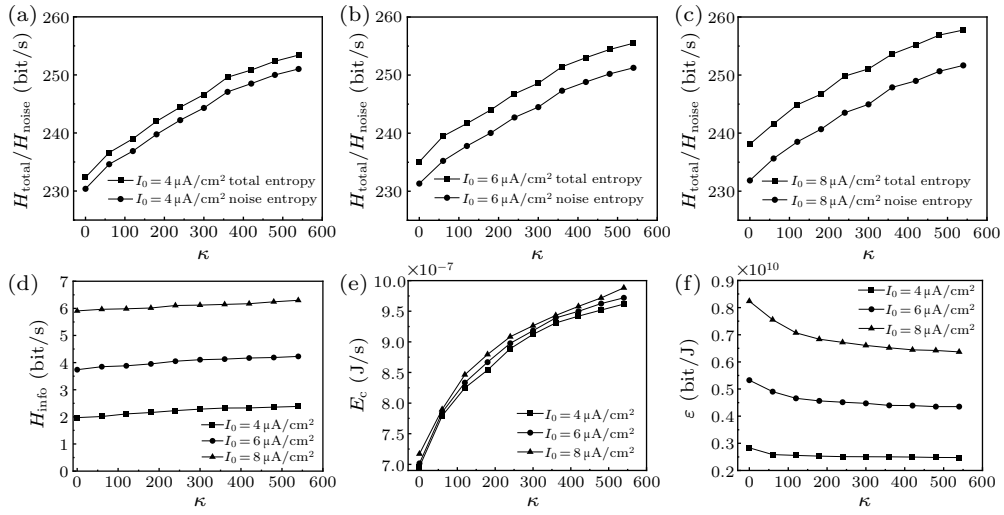


Fig. 3. Information transmission capacity, energy cost, and energy efficiency of the stochastic HH model as a function of κ, the number of cooperatively activated sodium channels. (a)–(c) The total and noise entropy rates of the stochastic HH model in response to pulse inputs as a function of κ: (a) $I_0=4.0$ μA/cm^2, (b) $I_0=6.0$ μA/cm^2, (c) $I_0=8.0$ μA/cm^2. (d) The mutual information of the stochastic HH model in response to pulse inputs as a function of κ. (e) The energy cost of the stochastic HH model as a function of κ. (f) The energy efficiency of the stochastic HH model as a function of κ. In the simulations, the membrane area of the stochastic HH model is set as $S=100$ μm^2.

Finally, for a neuron with membrane area of S, its energy consumption $E_{\rm c}$ is calculated as $E_{\rm c}=E'_{\rm c}S$. It is seen from Fig. 2(e) that the energy cost increases with the increasing membrane area, no matter whether the channels are activated independently or cooperatively. However, the neurons with cooperative activation of channels cost more energy. Therefore, the enhanced information transmission capacity due to cooperative activation of channels is at a cost of increasing energy consumption. We move on to analyze the energy efficiency of neurons in the information processing process, which is defined as the information rate over the energy consumption rate,

$$\varepsilon=H_{\rm info}/E_{\rm c}. \quad (13)$$

It is seen from Fig. 2(f) that neurons with cooperative activation of channels would be less energy efficient in their information processing process. It is also noted that the neurons, no matter whether their sodium channels are activated independently or cooperatively, exhibit a peak of energy efficiency around 200 μm^2. This optimal size for maximal energy efficiency of the HH neurons has been predicted theoretically and confirmed with simulations in our previous work.[17]

We then investigate how the number of cooperatively activated sodium channels affects the information processing capacity and energy efficiency of the neurons. As demonstrated in Figs. 3(a)–3(c), as more sodium channels are activated cooperatively, the total entropy in the spike sequences of the neurons in response to pulse inputs increases. However, since the noise entropy in spike sequences is increased as well, the increase in total entropy does not bring much contribution to the enhancement of the neuron's information transmission capacity, as indicated with mutual information in Fig. 3(d). Meanwhile, it is seen from Fig. 3(e) that, more cooperatively activated sodium channels lead to more energy consumption of the neurons in the information processing process. As a result, the energy efficiency of the neurons decreases as the number of the cooperatively activated sodium channels increases, as shown in Fig. 3(f).

To elucidate the underlying mechanism that accounts for the degraded energy efficiency due to the cooperative activation of ion channels, we investigate how it affects the response property, spontaneous activity, and the corresponding energy cost of the neurons. We define the detection rate P_c as the ratio of the number of spikes evoked by the pulse inputs to the total number of applied pulse inputs. We find that both the detection rate P_c and the spontaneous firing rate P_r are increased with the increasing number of cooperatively activated sodium channels, as demonstrated in Figs. 4(a) and 4(b). It is these two aspects of increases in neuron's output electrical signals that lead to the increasing mutual information and noise information, thus the increasing total information, as shown in Fig. 3. The energy consumption of the neurons in the information processing process can also be attributed to two parts: the energy consumption to generate APs and the energy consumption due to subthreshold membrane potential fluctuations. We also find that the energy consumption per spike (E_{spike}) increases with the increasing number of cooperatively activated channels [Fig. 4(c)]. On the contrary, the energy consumption due to subthreshold fluctuations of membrane potential, E_{subthr}, would increase if the neuron size is large, but would decrease if the size is small [Fig. 4(d)]. However, when the size of the neuron is small, the spontaneous firing rate is high so that the decrease in E_{subthr} due to channel coupling would be overcompensated by the increase in energy consumption in generating APs [Fig. 2(e)]. Therefore, we conclude that the cooperative activation of ion channels enhances the response of neuron to external inputs, but also increases the spontaneous outputs, at a cost of increasing energy consumption in generating output signals. Therefore, cooperative activation of ion channels indeed enhances the information processing capacity of the neurons but downgrades the energy efficiency of the neurons.

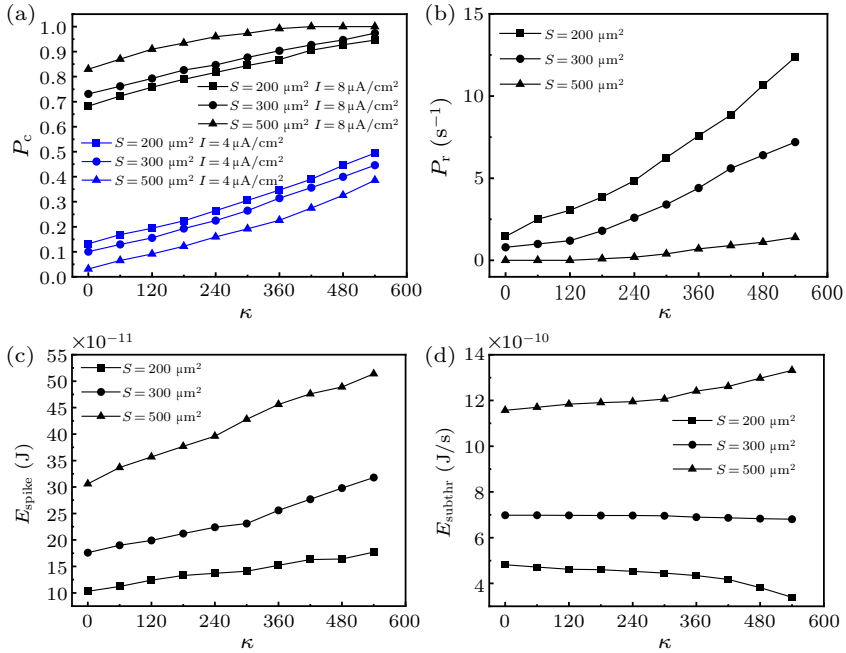


Fig. 4. Effects of sodium channel cooperative activation on the neurons' response property, spontaneous activity, and the corresponding energy cost. (a) The dependence of detection rate on the number of cooperatively activated sodium channels for neurons with different sizes and input strengths. (b) The spontaneous firing rate as a function of the number of cooperatively activated sodium channels for neurons with different size. (c) The energy cost per spike as a function of the number of cooperatively activated sodium channels for neurons with different sizes. $I_0 = 8.0$ μA/cm^2. (d) The energy consumption of subthreshold fluctuations per millisecond as a function of the number of the cooperatively activated sodium channels for neurons with different sizes.

In summary, it is argued that the evolutionary pressures have optimized neural systems for energy efficiency in information transmission and processing. The results in this study show that cooperative activation enhances information capacity but greatly increases energy consumption, degrading energy efficiency. This suggests that independent sodium channel activation may reflect an evolutionary tendency toward efficient coding given constraints on energy supply. Furthermore, since artificial neurons mimicking their biological counterparts are not constrained by energy supply,[18] we predict that their information processing capacity can be further enhanced by designing ion channels capable of cooperative activation. Meanwhile, neural models are power tool to explore and understand the pathological mechanism for many neural diseases.[19,20] Since experimental studies have reported the left-shift of activation and inactivation curves for voltage-gated sodium channels in damaged excitable cells,[21] our work implies that the possibility of similar downgraded energy efficiency may occur in these damaged cells. Further studies on energy efficiency of neurons with left-shifted channels may provide new insights into pathological mechanism of many neural diseases.

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References

[1] Kandel E, Schwartz J, Jessell T, Siegelbaum S and Hudspeth A J 2013 *Principles of Neural Science* 5nd edn. (New York: McGraw-Hill Medical)
[2] Hodgkin A L and Huxley A F 1952 *J. Physiol.* **117** 500
[3] Dayan P and Abbott L F 2001 *Theoretical Neuroscience: Computational and Mathematical Modeling of Neural Systems* (Cambridge: MIT Press)
[4] Fox R F 1997 *Biophys. J.* **72** 2608
[5] Chow C C and White J A 1996 *Biophys. J.* **71** 3013
[6] Naundorf B, Wolf F and Volgushev M 2006 *Nature* **440** 1060
[7] Yu Y, Shu Y and McCormick D A 2008 *J. Neurosci.* **28** 7260
[8] McCormick D A, Shu Y and Yu Y 2007 *Nature* **445** E1
[9] Yu L and Yu Y 2017 *J. Neurosci. Res.* **95** 2253
[10] Ding Q, Wu Y, Li T, Yu D and Jia Y 2023 *Chaos Solitons Fract.* **171** 113464
[11] Lu L, Yi M, Gao Z, Wu Y and Zhao X 2023 *Nonlinear Dyn.* **111** 16557
[12] Zeng S and Jung P 2004 *Phys. Rev. E* **70** 011903
[13] Moujahid A, d'Anjou A, Torrealdea F J and Torrealdea F 2011 *Phys. Rev. E* **83** 031912
[14] Ma J 2023 *J. Zhejiang Univ. Sci. A* **24** 109
[15] Zhu F, Wang R, Pan X and Zhu Z 2019 *Cogn. Neurodyn.* **13** 75
[16] Li X, Yu D, Li T and Jia Y 2023 *Nonlinear Dyn* **112** 2933
[17] Yu L and Liu L 2014 *Phys. Rev. E* **89** 032725
[18] Fu Y M, Wan C J, Zhu L Q, Xiao H and Wan Q 2018 *Adv. Biosyst.* **2** 1700198
[19] Liu Z, Han F and Wang Q 2022 *Nonlinear Dyn.* **108** 1849
[20] Yu Y, Fan Y, Han F, Luan G and Wang Q 2023 *Sci. China Technol. Sci.* **66** 3628
[21] Boucher P A, Joós B and Morris C E 2012 *J. Comput. Neurosci.* **33** 301