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Review:

A physical view of computational neurodynamics*

Jun MA†1, Zhuo-qin YANG2, Li-jian YANG3, Jun TANG4

[1]*Department of Physics, Lanzhou University of Technology, Lanzhou 730050, China*
[2]*School of Mathematics and Systems Science, Beihang University, Beijing 100191, China*
[3]*Department of Physics, Central China Normal University, Wuhan 430079, China*
[4]*School of Physics, China University of Mining and Technology, Xuzhou 221116, China*
†E-mail: hyperchaos@163.com; hyperchaos@lut.cn

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Abstract: The nervous system is made of a large number of neurons. Time-varying balance between excitatory and inhibitory neurons is important to activate appropriate modes of electrical activity. A realistic biological neuron is complex, often presenting various electrophysiological activities and diffusive propagation of ions in the cell. Therefore, the physical effects of electromagnetic induction become very important and should be considered when estimating signal encoding and mode selection. Synaptic plasticity and anatomical structure have been developed to enhance the self-adaption of neurons. Thus, the electrical mode with the most effective links and weights can be selected to benefit information encoding and signal propagation between neurons in the network. As a result, the demand for metabolic energy can be greatly reduced. In this review, neuron model setting with biophysical effects, modulation of astrocytes, autapse formation and biological function, synaptic plasticity, memristive synapses, and field coupling between neurons and networks are reviewed briefly to provide guidance in the field of neurodynamics.

Key words: Neuron; Neural networks; Autapse; Hamilton energy; Electromagnetic induction
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1 Building a neuron model

Electrophysiological processes in the cell can induce distinct bioelectricities and these charged ions are pumped to flow across the cell membrane via channels embedded in the membrane (Kawato et al., 1984; Stent, 1984; Busciglio et al., 1992). As a result, the balance of ion concentration between the inside and outside of the cell is disturbed and membrane potential becomes time-varying when an external electric stimulus is imposed on the neuron. From a physical viewpoint, static potassium and sodium can activate an electric field and the field energy can be generated by spatial distribution in the cell. Furthermore, the diffusion and propagation of these charged ions will change the energy distribution and energy propagation. In particular, the continuous current across the membrane channels will change the density distribution of charged ions, energy storage and release. Therefore, capacitance for the cell membrane can be used to estimate the membrane potential, and signal propagation will be modulated (Holmes and Loew, 2008). Biological cells are elastic and their geometry alters to change the capacitance of membranes exposed to external electromagnetic fields due to the effect of polarization and magnetization. Most previous studies seldom considered the elastic properties of cell membranes (Valverde, 1976; Pellionisz, 1989; Tomba et al., 2014) and thus the capacitance was considered fixed as a constant in biological neuron models (Fitzhugh, 1966; Hassard, 1978;

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ORCID: Jun MA, https://orcid.org/0000-0002-6127-000X

Morris and Lecar, 1981; McCormick et al., 2007; Pospischil et al., 2008). These neuron models can be simplified and rebuilt in circuits for nonlinear analysis (Tsumoto et al., 2006; González-Miranda, 2007; Storace et al., 2008; Goldwyn et al., 2011; Mao, 2017). For example, Patel and DeWeerth (1997) designed a very-large-scale integration (VLSI) circuit for producing electrical activity in a Morris-Lecar neuron, which was verified in an analog circuit by Hu et al. (2016). In this paper, we analyze the reasons for tolerance fluctuations, construct a condition-driven adaptive design method for tolerance using a hidden Markov model algorithm, and then present the results of some experiments on the precision stamping process and discuss the feasibility and effectiveness of the adaptive design method.

As the basic functional unit in the nervous system, a reliable neuron model is important to estimate the dynamic properties and predicate possible mode transition in neural activities. The propagation and pumping of ions in the cell can be very complex, but the application of a patch clamp benefits the estimation and detection of the membrane potential of neurons. As a result, implicit and auxiliary variables can be used to build reliable neuron models. Astrocytes (Dani et al., 1992; Parpura et al., 1994; Zonta et al., 2003; Navarrete et al., 2014) play an important role in regulating the concentration of calcium and inositol triphosphate (IP_3) by adjusting the neurotransmitters adenosine triphosphate (ATP) and glutamic acid. For some interneurons, an auxiliary loop is formed to develop an autapse (van der Loos and Glaser, 1972; Seung et al., 2000; Yue et al., 2017; Song et al., 2018). An autapse is a specific synapse, connecting its own body via a closed loop. It can be developed to enhance signal propagation when the axon of the neuron is injured (Wang CN et al., 2017). Thus, the anatomical structure of neurons and interaction between astrocytes and neurons should be considered. Guo et al. (2017) proposed that an astrocyte-neuron network driven by autapses can detect the possible occurrence of mode selection and firing pattern in the electrical activities. For isolated neurons, the involvement of an autapse connection can enhance the self-adaption of mode selection and response in electrical activities (Song et al., 2015; Ren et al., 2017; Uzun, 2017; Zhao and Gu, 2017; Xu Y et al., 2018a). Furthermore, setting an appropriate distribution of autapse connections in the network can induce coherence resonance (Uzun et al., 2017; Yang et al., 2017) and enhance the realization of synchronization (Ma et al., 2015b) and pattern selection (Ma et al., 2015a) in the network.

More importantly, physical effects should be considered in building reliable neuron models. From a dynamic point of view, a variety of neural circuits (Tamaševičius et al., 2015; Wei et al., 2017; Carro-Pérez et al., 2018; Wang RB et al., 2018; Bao et al., 2019) can be designed to generate different firing patterns by adjusting the parameters or applying an appropriate external stimulus. Thus, spiking, bursting, and even chaotic series can be produced to match the nonlinear properties of neural activities. In physical and mechanical systems, continuous energy supply is critical to support stable oscillation. In biological systems, sufficient metabolic energy is needed to maintain normal electrical activities. An energy model for estimating the electrical activities of neurons has recently been proposed (Wang ZY et al., 2015; Zheng et al., 2016; Wang YH et al., 2017; Wang and Wang, 2018). Results from the model are important for estimating the relation between blood flow, energy supply, and mode transition. Inspired by the Helmholtz theorem (Kobe, 1986), scale transformation is often applied to neuronal models and nonlinear circuits. The Hamilton energy (Wang Y et al., 2017; Wu et al., 2018a; Zhang et al., 2018) can be calculated to estimate the dependence of energy on oscillation modes, chaos control, and nonlinearity in the system. For example, a neuron will maintain lower Hamilton energy in a bursting and/or chaotic state than in a spiking state. The occurrence of multi-scroll attractors also presents lower Hamilton energy in chaotic systems. Most neuron models emphasize the occurrence and fluctuation of membrane potential induced by the channel current and an external electric stimulus, while the intrinsic physical field effect is missed. Potassium, sodium, and calcium are known to be kept and transmitted in and out of the cell membrane. Any slight spatial change or flow of these charged ions will induce a complex electromagnetic field in the cell and thus the successive transmission and pumping of ions will be changed to modulate electrical activity.

Therefore, Ma and Tang (2015) and Wu et al. (2017) suggested that magnetic flux can be added as

new variable to existing neuron models. The law of electromagnetic induction indicates that an equivalent induction current can be imposed to approach the effect of an induced electromotive force, which shows certain weight modulation on the membrane potential. Indeed, a memristor can be magnetic flux-controlled or charge-controlled, and the memristive function is dependent on the relation between magnetic flux and charges. At least two variables (membrane potential and current) should be used to estimate the electrical activity of a neuron. Ma and Tang (2015) introduced magnetic flux as a new additive variable to the three-variable neuron model, and the same magnetic flux was introduced into the four-variable neuron model. As a result, the effect of electromagnetic induction is estimated by supplying additive current to the membrane. The ability of a two-variable neuron model to describe the field effect resulting from electromagnetic induction can be improved by using three variables. For building a generic and simple neuron model, the effect of electromagnetic induction and radiation on neural activity can be estimated as

$$\begin{cases} C\dfrac{\mathrm{d}u}{\mathrm{d}t} = f(u, i, p) + I_{\mathrm{ext}} + i_{\mathrm{induct}}, \\ L\dfrac{\mathrm{d}i}{\mathrm{d}t} = g(u, i, p), \\ \dfrac{\mathrm{d}\varphi}{\mathrm{d}t} = k_1 u - k_2\varphi + \varphi_{\mathrm{ext}}, \\ i_{\mathrm{induct}} = \dfrac{\mathrm{d}q}{\mathrm{d}t} = \dfrac{\mathrm{d}q}{\mathrm{d}\varphi}\dfrac{\mathrm{d}\varphi}{\mathrm{d}t} = k_0\rho(\varphi)u, \end{cases} \tag{1}$$

where u represents the membrane potential, i is the channel current, q is the charge, φ represents the magnetic flux, p is the intrinsic parameter, and k_0, k_1, and k_2 are parameters associated with the media. When a neuron is considered as an excitable media, its physical parameters can be approached by using equivalent capacitance C and inductance L. t denotes time. I_{ext} denotes an external stimulus, and i_{induct} the induction current resulting from electromagnetic induction. φ_{ext} is used to describe different types of electromagnetic radiations. $\rho(\varphi)$ calculates the memductance of the memristor. This kind of electromagnetic induction can also be considered in the Hindmarsh-Rose, Hodgkin-Huxley (HH), and other neuron models (Ma and Tang, 2015; Wu et al., 2017) by including the magnetic flux variable and memristive function. In particular, when this effect is estimated in cardiac tissue, which is often described by two-variable reaction-diffusion equations, two kinds of death mechanisms (Wu et al., 2016; Ma et al., 2017) of heart tissue from electromagnetic radiation can be explained by a breakup of spiral waves and blocking of the propagation of target waves.

$$\begin{cases} \dfrac{\partial u}{\partial t} = f(u, v) + D\nabla^2 u + i_{\mathrm{induct}} + I_{\mathrm{ext}}, \\ \dfrac{\partial v}{\partial t} = g(u, v), \\ \dfrac{\partial \varphi}{\partial t} = k_1 u - k_2\varphi + \varphi_{\mathrm{ext}}, \\ i_{\mathrm{induct}} = \dfrac{\mathrm{d}q}{\mathrm{d}t} = \dfrac{\mathrm{d}q}{\mathrm{d}\varphi}\dfrac{\mathrm{d}\varphi}{\mathrm{d}t} = k_0\rho(\varphi)u, \end{cases} \tag{2}$$

where ∇^2 is the Laplace operation, and D represents the diffusion coefficient. The variables u and v often describe the activator such as membrane potential, and inhibitor such as recovery variable for current, respectively. In the cardiac tissue of a healthy heart, the sinoatrial node can emit a continuous electrical signal and maintain a stable target wave (Wu et al., 2016; Ma et al., 2017). A higher intensity of electromagnetic radiation can block the propagation of a target wave, thereby suppressing the blood pump in the heart (Qu et al., 2014). On the other hand, in the case of arrhythmia and tachycardia, when some spiral waves can be detected in the cardiac tissue, electromagnetic radiation can induce breakup of spiral waves, and ventricular fibrillation is induced leading to final rapid death of the heart. Inspired by model setting for neural activity (Ma and Tang, 2015; Wu et al., 2017), extensive studies have been carried out to investigate the collective behavior of neural networks and wave propagation in cardiac tissue in the presence of electromagnetic induction and radiation (Mvogo et al., 2017; Zhan and Liu, 2017; Ge et al., 2018b; Rostami et al., 2018; Takembo et al., 2018; Xu Y et al., 2018b; Lv et al., 2019; Mostaghimi et al., 2019).

It is accepted that an induction current can be used to estimate the effect of electromagnetic induction resulting from a time-varying concentration of charged ions under transmission and exchange. Also,

by generating memristive currents it can give helpful clues to help understand the function of memristive synapses. Therefore, when two neurons are connected by a memristive synapse (Park et al., 2015; Covi et al., 2016; Azghadi et al., 2017; Xu F et al., 2018), the synapse current for the coupled neurons is estimated by

$$I_{\mathrm{m}} = k\rho(\varphi)(u_1 - u_2), \tag{3}$$

where u_1 and u_2 describe the membrane potential for each of the two neurons respectively, $\rho(\varphi)$ is dependent on the synapse property, and k is the coupling intensity. According to the difference in function mechanism, an electrical synapse is often considered as a gap junction coupling between neurons, while chemical synapse coupling is activated between neurons by the release of neurotransmitters. From a physical viewpoint, electrical synapse coupling could account for voltage coupling via a resistor, while chemical synapse coupling can be recognized as field coupling (Perea et al., 2009; Ma SY et al., 2019; Wu et al., 2019) because any release of neurotransmitter can induce a time-varying electromagnetic field and a change in the distribution of charged ions in the cell. The field variable is not presented in this model, though its field effect is considered by using an induction current. Therefore, a variable E is introduced to describe the effect of the electric field in the neuron, that is, the continuous exchange of ions across the membrane channels can be considered as placing a certain distribution of charges on the cell membrane. For simplicity, a two-variable nonlinear circuit is used and the intrinsic electric field E of the capacitor is estimated. As a result, its generic form and the relations of physical variables are estimated as follows (Ma J et al., 2019):

$$\begin{cases} C\dfrac{\mathrm{d}V}{\mathrm{d}t} = f(V, i, p), \\ L\dfrac{\mathrm{d}i}{\mathrm{d}t} = g(V, i) + rE, \\ \dfrac{\mathrm{d}E}{\mathrm{d}t} = ki + E_{\mathrm{ext}}, \end{cases} \tag{4}$$

where V and E describe the membrane potential and inner and outer electric fields of the cell membrane, respectively. C, L, r, k, and p are normalized parameters for equivalent capacitance, inductance, the size of the membrane and intrinsic properties of the media. E_{ext} is the external static or the time-varying electric field, and the propagation of an ion flow will be regulated to modulate the channel current. When more than two neurons are exposed to the external electric field, the electric field between the neurons will be activated to adjust the synapse coupling. As suggested in open problems (Ma SY et al., 2019), the Hindmarsh-Rose and HH neuron models can be used to describe the nonlinearity in Eq. (4). Thus, field coupling-induced synchronization and pattern formation can be further investigated. From physical and chemical viewpoints, the neuron cell can be regarded as an excitable media in which the inner propagation of charged ions and external electromagnetic field will change the distribution of magnetic and electrical fields, as shown in Fig. 1. As a result, the transient distribution and flow of ions across the membrane will have a distinct impact on membrane potential. Therefore, it is useful to include appropriate variables to estimate the membrane potential, channel current, magnetic field, and electrical field. Wu et al. (2019) proposed a new neuron model with four physical variables: membrane voltage, current, charge number on the membrane, and magnetic flux. Their paper includes a detailed description of the dynamical response induced by electromagnetic radiation.

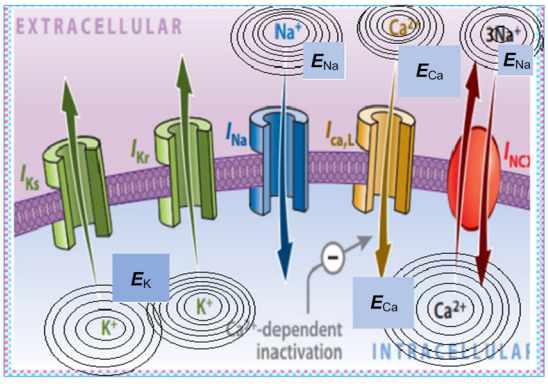

Fig. 1 Ion propagation in a neuron cell. Calcium, potassium, and sodium ions can trigger spatial distribution of electric fields E_{Ca}, E_{K}, and E_{Na}

Continuous movement and propagation of ions can induce a time-varying magnetic field which can be described by magnetic flux

2 Contribution of astrocytes

Astrocytes were thought to be passive elements playing merely nutritional and structural roles in the central nervous system of mammals. However, new evidence suggests that they can have a great effect on neuron function (Perea et al., 2009). It was suggested that astrocytes implement a feedback control to neural activity through synapses by regulating neurotransmitter release (Volterra and Meldolesi, 2005; Wang et al., 2009; Halassa and Haydon, 2010; Henneberger et al., 2010; Khakh and Sofroniew, 2015; Poskanzer and Yuste, 2016). This feedback may involve different biochemical pathways (de Pittà et al., 2012), among which, the astrocytic calcium modulation pathway is most involved. Astrocytes are not electrically excitable cells, but they can sense and respond to synaptic activity by adjusting their Ca^{2+} concentration. When the neurotransmitter glutamate is released from a presynaptic neuron, the astrocyte Ca^{2+} concentration will be increased. Furthermore, astrocytes can release some active gliotransmitters which can modulate the excitability and synaptic plasticity of pre- and postsynaptic neurons (Araque et al., 2014). Indeed, astrocytes are connected via gap junctions, and any increase of Ca^{2+} concentration in one astrocyte may induce a Ca^{2+} wave in nearby astrocytes (Newman and Zahs, 1997). As a result, neurons connected to those astrocytes will be excited and may have epileptic activity in the whole neuron-astrocyte network.

Due to the limitations of experimental technology, most studies on astrocyte Ca^{2+} signaling are performed in vitro (Bezprozvanny et al., 1991; Höfer et al., 2002; Sloan and Barres, 2014; Manninen et al., 2018). Thus, the reliability of these results may be dependent on the method applied and the context selection. Therefore, a reliable computational method is often required to understand the complexity of different astrocyte Ca^{2+} signals and estimate the dynamics of astrocytic Ca^{2+}. The most popular astrocyte Ca^{2+} models are the DeYoung-Keizer (de Young and Keizer, 1992) and Li-Rinzel (Li and Rinzel, 1994) models, and the Höfer model (Höfer et al., 2002), which includes a calcium (Ca^{2+})-induced Ca^{2+} release (CICR) mechanism (Höfer et al., 2002). The Höfer model was proposed specifically to simulate astrocytes, and comprises the dynamics of the cytoplasmic Ca^{2+} concentration ($C_{Ca^{2+}}$), endoplasmic reticulum (ER) store Ca^{2+} concentration ($C_{Ca^{2+},ER}$), IP_3 concentration (C_I), and active fraction of IP_3R (C_R). The nonlinear relation between these variables can be calculated by

$$\begin{cases} \dfrac{\partial C_{Ca^{2+}}}{\partial t} = v_{rel} - v_{SERCA} + v_{in} - v_{out} \\ \qquad\qquad + D_{Ca}\left(\dfrac{\partial^2 C_{Ca^{2+}}}{\partial x^2} + \dfrac{\partial^2 C_{Ca^{2+}}}{\partial y^2}\right), \\ \dfrac{\partial C_{Ca^{2+},ER}}{\partial t} = \beta(v_{SERCA} - v_{rel}), \\ \dfrac{\partial C_R}{\partial t} = v_{rec} - v_{inact}, \\ \dfrac{\partial C_I}{\partial t} = v_{PLC\beta} + v_{PLC\delta} - v_{deg} \\ \qquad\qquad + D_{IP_3}\left(\dfrac{\partial^2 C_I}{\partial x^2} + \dfrac{\partial^2 C_I}{\partial y^2}\right), \end{cases} \tag{5}$$

where v_{rel}, v_{SERCA}, v_{in}, and v_{out} represent the Ca^{2+} release from the ER store, Ca^{2+} pump into the ER store, Ca^{2+} transmembrane influx, and transmembrane outflow, respectively. v_{rec} and v_{inact} are the recovery and inactivate rates, respectively, of the IP_3 receptor. $v_{PLC\beta}$ and $v_{PLC\delta}$ are the activation rates of IP_3 mediated by PLCβ and PLCδ, respectively, and v_{deg} is the IP_3 degradation rate. The diffusion of Ca^{2+} and IP_3 inside the astrocyte cell is described by the last diffusive terms, where D_{Ca} and D_{IP_3} are the diffusion coefficients. The Höfer model assumes that IP_3 is the key messenger that mediates information communication between cells. Based on this model, many studies of Ca^{2+} signaling in astrocytes (Lavrentovich and Hemkin, 2008; Zeng et al., 2009; Toivari et al., 2011) have been discussed. The DeYoung-Keizer and Li-Rinzel models, in which the neurotransmitter is taken into account at the same time (Gibson et al., 2007; Bennett et al., 2008; Chander and Chakravarthy, 2012; Witthoft and Karniadakis, 2012; Hadfield et al., 2013; Witthoft et al., 2013; Kenny et al., 2018), have also been selected for detecting signal propagation in and between cells.

A pioneering model named the ‘dressed neuron’ model, which includes a neuron and an astrocyte, was proposed by Nadkarni and Jung (2003). They adapted

the HH model (Hodgkin and Huxley, 1952) to simulate the action of a neuron, and the Li-Rinzel model to simulate the Ca^{2+} dynamics in an astrocyte. When the neuron is triggered to produce an action potential, neurotransmitter is released into the synaptic cleft and binds to the transmitter receptor on the astrocyte. Thus, the intracellular IP_3 is released as follows (de Young and Keizer, 1992; de Pittà et al., 2012):

$$\frac{\mathrm{d}C_{\mathrm{IP_3}}}{\mathrm{d}t}=\frac{1}{\tau_{\mathrm{IP_3}}}(C^{*}_{\mathrm{IP_3}}-C_{\mathrm{IP_3}})+r_{\mathrm{IP_3}}\Theta(v-50\ \mathrm{mV}), \quad (6)$$

where $C_{\mathrm{IP_3}}$ represents the concentration of IP_3 in the astrocyte cell, and $C^{*}_{\mathrm{IP_3}}$ is the equilibrium value of IP_3. $\tau_{\mathrm{IP_3}}$ determines the time scale of chemical transmission of IP_3. A step function $\Theta(x)$ is used to simulate the IP_3 release triggered by action potential, and $r_{\mathrm{IP_3}}$ represents the strength of coupling between the neuron and the astrocyte. The feedback of astrocytes to the HH neuron is estimated by introducing the current I_{astro}, which can be obtained by fitting the experimental data (de Young and Keizer, 1992; de Pittà et al., 2012):

$$\begin{aligned} I_{\mathrm{astro}}&=2.11\Theta(\ln y)\ln y,\\ y&=(C_{\mathrm{Ca^{2+}}}-196.69)\ \mathrm{nmol/L}. \end{aligned} \quad (7)$$

Note that the time scale of the action potential of the neuron is millisecond, whereas the Ca^{2+} dynamics simulated by the Li-Rinzel model is on a much slower time scale of seconds. Some results confirmed that with a stronger coupling strength, i.e. a high value of $r_{\mathrm{IP_3}}$ that is related to the density of mGlu receptors on the astrocyte membrane, seizure-like oscillations emerge without an external stimulus (Nadkarni and Jung, 2003). Therefore, Nadkarni and Jung (2003) suggested that higher expression of mGlu receptors may be one of the physiological reasons for epilepsy. Based on a similar modeling scheme, Tang et al. (2013, 2016) obtained a tripartite synapse model and discussed in detail the information transmission between neurons. They found that the presence of an astrocyte facilitates the occurrence of episodic spikes (ESs) in both presynaptic and postsynaptic neurons. Furthermore, the noise, originating from random open-close transitions of calcium ion channels in the endoplasm reticulum membrane of the astrocyte, can change the firing patterns of two neurons and facilitate the occurrence of ESs in both neurons during neuronal information transmission.

In addition to the HH model, other neuron models, such as the Pinsky-Rinzel Model (Nadkarni and Jung, 2007), FitzHugh-Nagumo model (Postnov et al., 2007), and leaky integrate-and-fire model (Wade et al., 2012; Nazari et al., 2017), can be effective for examining information transmission between neurons and astrocytes. These models can reproduce the phenomena of hyperexcitability, plasticity, and Ca^{2+} oscillation observed in experiments. Other evidence has indicated that astrocytes can be organized into networks (Halassa and Haydon, 2010), interconnected through gap junction channels. These are regulated by extracellular and intracellular signals that enable the effective exchange of information. For exploring two networks, the concept of 'astroglial networks' was suggested in a recent review paper (Giaume et al., 2010). Network models including multiple neurons and astrocytes have been developed to study information transmission in the cortex (Allegrini et al., 2009; Liu and Li, 2013; Chan et al., 2017; Tang et al., 2017), hippocampus (Amiri et al., 2012a, 2013; Mesiti et al., 2015), and other parterres in the brain (Amiri et al., 2012b, 2012c; Yang and Yeo, 2015). The main issues discussed in those models are synchronization, information transfer, and hyperexcitability. For example, Amiri et al. (2012a, 2013) constructed a neural network model to study the effect of astrocytes on synchronization reach. It is thought that astrocytes are capable of changing the threshold value of transition from synchronous to asynchronous behavior among neurons. Tang et al. (2017) constructed a chain-type neuron-astrocyte network model to study the correlation between an astrocytic calcium wave and seizure-like behavior in a neuron network. They concluded that calcium wave propagation in astrocytes dominates the propagation of seizure-like discharges (SDs) in coupled neurons (Tang et al., 2017). As reviewed by Manninen et al. (2018), calcium signaling models in astrocytes can be categorized into four groups: isolated astrocyte models, astrocyte network models, neuron-astrocyte synapse models, and neuron-astrocyte network models.

3 Synaptic plasticity

Elastic media, such as cardiac tissue and muscle, can capture and slow down external mechanical pressure by inducing an appropriate deformation orientation. Complex electrophysiological activity occurs in the cardiac tissue and nervous system, and an appropriate response will be triggered when an external electric stimulus is applied. Synapses behave synchronously as receptors and transmitting terminals. In particular, the input signal will be encoded by a synapse and then converted to an equivalent transmembrane potential. Furthermore, intracellular and extracellular ion exchange will be regulated to induce a variety of firing patterns. External stimuli, including a signal from the post-synapse from an adjacent neuron, can change the polarization properties of a synapse. As a result, the impendence of the synapse will be changed under an external stimulus. When two or more neurons are coupled via synapses, the time-varying exchange of charged ions and fluctuation of membrane potentials can propagate electric signal along the axon to emit changeable signals to the pre-synapse of another neuron. Therefore, the impendence of the coupling synapse is changed to present different intensities. The nervous system has distinct self-adaption due to synaptic plasticity (Zucker, 1989; Bliss and Collingridge, 1993; Abraham and Bear, 1996; Abbott and Nelson, 2000; Zucker and Regehr, 2002). The connection intensity of synapses can be changeable, and the property or phenomenon of relatively permanent alteration in the morphology and function of synapses is confirmed. That is, the synapse current can be enhanced by an increase in involvement in the processing electrical activities, while its intensity is reduced by a decrease in involvement in signal encoding.

Synaptic plasticity can be short-term or long-term. Potentiation, depression, and facilitation are the main aspects of short-term synaptic plasticity. Short-duration synaptic plasticity (Salin et al., 1996; Buonomano, 2000; Junge et al., 2004; Pan and Zucker, 2009; Tutkun et al., 2010) is an important form of synaptic plasticity and plays an important role in activating the normal function of nervous systems. Synaptic short-duration plasticity can strengthen the certainty of synaptic transmission, and regulate the balance between the cortical excitation and inhibition. As a result, a switch between excitation and inhibition enables neurons to select the most suitable response and firing patterns. In a word, its biological function is to form the temporal and spatial characteristics of neural activities, and to enhance and regulate the synchronous oscillation of the cortical thalamic network. Synaptic short-duration plasticity is involved in the realization of higher nervous system functions such as attention, priming effect, sleep rhythm, learning, and memory. Short-duration synaptic plasticity can be divided into short-duration enhancement and short-duration inhibition. Long-term synaptic plasticity (Bear and Malenka, 1994; Engert and Bonhoeffer, 1999; Nestler, 2001; Yuste and Bonhoeffer, 2001; Trachtenberg et al., 2002) is characterized by long-term potentiation (LTP) and long-term depression (LTD). Long-term potentiation is also called the long-term gain effect, and is a persistent potentiation phenomenon in signal transmission resulting from synchronous stimulation of two neurons. This is one of several phenomena associated with synaptic plasticity, the ability of synapses to change strength (Bliss and Collingridge, 1993). Since memory is thought to be encoded by changes in synaptic strength, LTP is widely regarded as one of the major molecular mechanisms underlying learning and memory.

In 1966, LTP was discovered in the hippocampus of rabbits by Terje Lomo and has long been a hot topic of research. Many modern LTP studies attempt to better understand its biological rationale, while others aim to explore the causal relationship between LTP and behavioral learning. Some researchers are developing ways to improve learning and memory by enhancing LTP, for example, by injecting drugs. LTP is also the subject of clinical research in areas such as Alzheimer's disease and addiction medicine. LTP has several characteristics, including input specificity, relevance, synergy, and persistence. LTD is known as long-term depression and long-term depotentiation, which refers to the inhibitory behavior of nerve synapses lasting for several hours to several days. Strong synaptic stimulation (cerebellar Purkinje cells) or long-term weak synaptic stimulation (hippocampus) can lead to long-term inhibition, which is thought to be induced by changes in postsynaptic receptor density. However, changes in presynaptic release may also have an effect on inhibition. Long-term inhibition

of the cerebellum is assumed to play an important role in motor learning, and long-term suppression of the hippocampus can be effective in erasing past memories. LTD of the hippocampus/cerebral cortex is controlled by the N-methyl-D-aspartate (NMDA) receptor, mGluR, or the endocannabinoid.

The coupling intensity and equivalent synapse current are dependent on the involvement of synapses such that external input-induced polarization can be balanced and less energy can be consumed. As a result, the synchronization between neurons and model selection can be controlled completely. For example, Wang JY et al. (2018) constructed a modular neuronal network with modified Oja's learning rule, and used it to eliminate the pathological synchronized rhythm of interacting bursting neurons. They found that synaptic plasticity with a high learning rate can effectively suppress bursting synchronization among strongly synchronous neurons in a modular neural network by applying a specific range of coupling intensity. Based on an Izhikevich neuron within a subthreshold excitatory population, these individual neurons can exhibit noise-induced bursts with increasing coupling intensity. The neuronal population has adaptive dynamic synaptic strength governed by spike-timing-dependent plasticity (STDP), and the neuron cannot fire spontaneously without noise. Kim and Lim (2018) investigated the effect of additive STDP on stochastic burst synchronization (SBS) by changing the noise intensity in a Barabási–Albert scale-free network (SFN). They explained a Matthew effect in synaptic plasticity which occurs due to a positive feedback process. Furthermore, perfect burst synchronization (with a high bursting measure) improves with LTP of synaptic strength, while non-perfect burst synchronization (with a low bursting measure) deteriorates with LTD. Tarai et al. (2019) discussed the neurobiological mechanisms of stress and mood disorders with the aim of enhancing the pharmacological effects of antidepressants and mood stabilizers. They found that regulation of neurotrophic factors can blockade stress and enhance neuronal survival, even though limbic regions can be paralyzed. Neurotrophic factors and molecular agents also adjust behavioral and synaptic plasticity in addiction and stress disorders. Short-term synaptic depression mainly reveals the depletion of the readily releasable pool (RRP) of quanta. Bui and Glavinović (2013) used patterned stimulation on the Schaeffer collateral fiber pathway and model-fitting of the excitatory postsynaptic currents (EPSCs) recorded from CA1 neurons in rat hippocampal slices. Ursino et al. (2018) implemented new synaptic learning rules to take into account the role of partially shared features and distinctive features with different saliency. The trained network handled word recognition and task naming tasks in an effective way, and the different roles of salient versus marginal features in concept identification were exploited. Li (2014) studied dendritic and synaptic integration with different spatial distributions of synapses on the dendrites of a biophysically-detailed layer 5 pyramidal neuron model. They found that temporally synchronous and spatially clustered synaptic inputs make dendrites perform a highly nonlinear integration. Lu et al. (2019) analyzed the propagation and fidelity of a subthreshold EPSC signal in a feed-forward neural network composed of five layers by using the spike timing precision and power norm and the EPSC signal imposed on the Hodgkin–Huxley neurons of the first layer. They found that background noise contributed to the propagation of subthreshold EPSC signal in the feed-forward neural network and the fidelity between the system's response and subthreshold EPSC signal was preserved. Sun et al. (2019) discussed the dependence of signal detection on coupling strength and network topologies in small-world neuronal networks. They confirmed that the shorter the average path length, the better the signal detection under intermediate coupling strengths.

Synaptic plasticity, involving changes in synaptic strength observed in vivo or in vitro after learning, is one of the mechanisms underlying memory storage. Long-lasting forms of synaptic plasticity, including both LTP that synaptic strength increases (Bliss and Gardner-Medwin, 1973; Bliss and Lømo, 1973), and LTD that synaptic strength decreases (Ito, 1989), are the cellular bases of learning and memory (Bliss and Collingridge, 1993), which are fundamental mental processes critical for adaptation and survival.

Quantitative computational models have become important for obtaining a deep understanding of complex networks of interacting pathways with convergence, divergence, and positive and negative feedback loops. Some evidence has revealed that a large number of molecules, and complex interactions between them, underlie plasticity (Collins et al., 2005;

Coba et al., 2009). Presynaptic release of glutamate and postsynaptic depolarization are the two crucial features of most induction protocols at excitatory synapses. Biophysical models involving both electrophysiological properties and biochemical reactions (signaling pathways) have been developed to understand the pre- and postsynaptic events in LTP and LTD (Kotaleski and Blackwell, 2010; Manninen et al., 2010). For example, a framework for computational models of signaling pathways was proposed to understand the molecular mechanisms underlying synaptic plasticity of glutamatergic synapses (Kotaleski and Blackwell, 2010). Some computational postsynaptic signal transduction models have been developed to investigate the dependence of synaptic plasticity on species and interactions (Manninen et al., 2010).

An elevation in intracellular calcium in the postsynaptic neuron is crucial for LTP or LTD (Bliss and Collingridge, 1993; Malenka and Bear, 2004), and shows some differences from the molecular mechanisms leading to synaptic plasticity. Ca^{2+} can activate protein kinases and phosphatases for inducing phosphorylation–dephosphorylation cycles, LTP and LTD. There are various pre- and postsynaptic mechanisms of changes in synaptic strength, in which cytosolic Ca^{2+}/calmodulin-dependent signals play an important role in synaptic plasticity (Lisman and Goldring, 1988a, 1988b). Most mechanistic models typically confirm the role of calcium in synaptic plasticity by detecting calcium dynamics in electrical activity (Schiegg et al., 1985; Gamble and Koch, 1987; Holmes and Levy, 1990). It is claimed that the amplitude of calcium elevation depends on the frequency of synaptic stimulation, while kinases are not directly implicated in plasticity. It remains open why some stimulation protocols produce depression and others produce potentiation, because Ca^{2+} can activate multiple processes and enzymes. The interaction of multiple signaling pathways at multiple points leading to kinase activation, as well as neuromodulators, is nonlinear processing, which makes a quantitative understanding more difficult. Indeed, further computational modeling of signaling pathways is needed to investigate these complex interactions and predict important molecular mechanisms, and then to guide researchers to the most valuable experiments.

It is assumed that the number of molecules in deterministic modeling is large enough to be represented as a concentration. However, many subcellular compartments are so small as to contain finite molecules, which results in stochastic fluctuation in molecule numbers and changes the outcome of signaling pathways. Stochastic simulations have revealed that signaling pathways, such as positive feedback loops with bistable switches modeled deterministically, are no longer bistable. For example, taking an extremely long time to spontaneously switch states resembles the bistability mechanism, and suggests that synapses can exhibit multiple stable states (Hayer and Bhalla, 2005). These spontaneous transitions cause thresholds to be located in some ranges and so switches become either less sensitive to signals or more sensitive to noise (Bhalla, 2004; Hayer and Bhalla, 2005). Deterministic simulations (Bhalla and Iyengar, 1999; Bhalla, 2002; Ajay and Bhalla, 2004) have revealed several emergent properties of a global network of interacting pathways that are not present in individual pathways. Consequently, stochastic effects should be considered when modeling a system.

Spatial gradients of signaling molecules are known to be prominent in neurons with elongated dendritic structures, and the spatial aspect of cell signaling should be estimated. For example, synaptic inputs in one part of the dendrite induce the generation and diffusion of secondary messengers to other parts of the dendrite (Blackwell and Jedrzejewska-Szmek, 2013). When all synapses of a neuron are potentiated in response to synaptic stimulation, the neuron will respond not only to previously learned patterns, but also to any arbitrary spatial pattern of synaptic input from environmental stimuli (Irvine et al., 1994). Modeling this spatial aspect of neurons is a relatively new approach to investigating neuronal plasticity (Ajay and Bhalla, 2007; Neves et al., 2008). Although there are many non-spatial models of signaling pathways, it is necessary to focus on models incorporating significant morphological features of neurons, such as a soma with an elongated dendrite or a dendrite with spines (Blackwell and Jedrzejewska-Szmek, 2013). A more comprehensive, deterministic spatial model of signaling pathways (including mitogen-activated protein kinase (MAPK), protein kinase A (PKA), calcium/calmodulin-dependent protein kinase II (CaMKII), and protein kinase C (PKC)) needs to incorporate a multi-compartmental, multi-channel electrical model of a dendrite.

In summary, computational models of synaptic plasticity have taken into account the involvement of molecules in synaptic plasticity and that some particular molecular mechanisms are responsible for experimental observations. Most models at the molecular level are far from complete due to a lack of knowledge of the biology and exact setting for kinetic parameters. This also raises some challenges for understanding the role of the stochasticity induced by sometimes only dozens of copies of a certain protein in a synaptic spine, and how to estimate the effect of spatial inhomogeneity remains an open question. The scarcity of complete models of synaptic plasticity reflects the complexity of the underlying mechanisms resulting from insufficient information on the quantity and subcellular localization of critical enzymes. Therefore, applying a synthetic and integrative systems-level approach to model setting facilitates a deeper understanding of nonlinear processes of multiple interactions in information processing and memory storage in neurons.

4 Collective behavior in neural networks

Due to the application of nanotechnology, feasible micro circuits and artificial synapses can be designed to build intelligent neuron processors. In a practical sense, the collective responses of neural processors and artificial neurons are worthy of further investigation so that signal propagation and information encoding in nervous systems can be understood. In this way, the occurrence and emergence of neural disease could be predicted, enabling possible suppression or curing. Neurons can be soaked in potassium, sodium, calcium, and even chloride ion solutions. A concentration gradient of ions can be activated to enhance the exchange and pumping of charged ions, thereby changing the membrane potential to trigger a variety of firing modes (Gu and Chen, 2014; Gu et al., 2014a, 2014b; Gu and Pan, 2015a, 2015b). In the nervous system, 20% of neurons can be inhibitory while 80% are kept in excitatory states. Therefore, it is important to consider the balance between excitability and inhibition (Zhao and Gu, 2015; Xiao et al., 2016) of neurons in estimating collective responses and pattern selection in neural networks. The collective behavior of networks depends mainly on the local kinetics of node and topological connection (Ma et al., 2016b; Mei et al., 2016, 2018; Xu et al., 2016; Wei et al., 2018), and even initial setting (Ma et al., 2016a). For example, synchronization transition between neurons can be induced by resetting parameters and initial values (Gu et al., 2015). Multistability emerges in memristive systems involved with memristor-based functions. Therefore, synchronization stability between memristive systems (oscillators) is dependent on the initial values (Wu et al., 2018b, 2018c; Liu Y et al., 2019). As a result, resetting the initial values will induce different types of synchronization.

In biological neurons, chemical and electrical synapses are activated to receive and encode signal inputs and wave propagation between neurons. An electric synapse can couple neurons via a gap junction while a chemical synapse can connect neurons by release of a neurotransmitter. From a dynamic viewpoint, the parameters of many nonlinear circuits or systems can be modulated to trigger quiescent, spiking, bursting, and even chaotic series, which can be consistent with modes of electrical activity in the membrane potential of biological neurons. Any electrical devices such as resistors, capacitors, induction coils, and memristors can be used to connect the nonlinear circuits, and appropriate setting of parameters (resistance, capacitance, inductance, and memductance) for the coupling device can realize phase synchronization and/or complete synchronization. Indeed, gap junction coupling shows properties similar to those of direct voltage coupling via a resistor by consuming a certain Joule heat. This kind of coupling is often used as direct variable coupling. As explained by Liu ZL et al. (2019a) and Xu et al. (2019), capacitor coupling provides effective electric field coupling by balancing and pumping the energy from the connected circuits. The energy pumping in the coupling device is estimated from $H=0.5C(V_1-V_2)^2$, where C is the capacitance of the coupling device, and V_1 and V_2 are the output voltages of the circuits. On the other hand, inductor coupling (Yao et al., 2019) bridges magnetic field coupling by balancing the energy estimated as $H=0.5LI^2$, where L is the inductance and I the current across the coupling induction coil. A biological tissue such as a synapse can present complex physical properties such as resistance, capacitance, and inductance. Ma J et al. (2019) used a resistor, capacitor,

and induction coil to investigate synchronization realization between memristive circuits. Therefore, hybrid synapses can be more reliable in processing information and signal propagation than a sole chemical or electrical synapse. Liu ZL et al. (2019b) combined a capacitor, resistor, and induction coil to design hybrid coupling devices. By connecting these electrical devices in parallel or series, they found that parallel connection was more effective in stabilizing synchronization than connection in series, and that the intensity threshold via resistance coupling could be reduced and power consumption greatly reduced in the coupling device. For example, Fig. 2 is a diagram representing two artificial neurons connected via a hybrid synapse.

These hybrid synapses bridge the same output ends by balancing the energy flow and energy consumption. Nonlinear circuits can draw out many output ends, and thus more than two coupling channels can be opened for signal and energy exchange. For an isolated neuron, low frequency, high frequency signals and even different kinds of noise can be imposed synchronously, and the intrinsic properties of excitable media account for mode selection and dynamical response in the electrical activities. From a physical viewpoint, continuous or intermittent release of a neurotransmitter can change the propagation and distribution of charged ions. As a result, an electromagnetic field is induced to propagate signals between neurons. Therefore, this kind of field coupling gives physical evidence for understanding the biological function of chemical synapses. In realistic and biological nervous systems, neurons show diversity in excitability and inhibition, and can be considered in different clusters and layers in the networks. As a result, the collective response in hybrid networks with cluster connections and a layered distribution is worthy of investigation by activating multiple channel coupling and hybrid synapses. In estimating the degree of spatial regularity and synchronization, the statistical synchronization factor (Qin et al., 2014; Wang and Ma, 2018) R is calculated using mean field theory, as follows:

$$F=\frac{1}{N}\sum_{i=1}^{N}x_i,\quad R=\frac{<F^2>-<F>^2}{\frac{1}{N}\sum_{i=1}^{N}(<x_i^2>-<x_i>^2)},\tag{8}$$

where x_i is any detectable variable of node i in a network composed of N nodes, and <*> represents the average value over time with a transient period. $R\approx1$ indicates perfect synchronization and the network will show a homogeneous distribution, while $R\approx0$ indicates the occurrence of non-perfect synchronization and a regular spatial pattern is formed in the network. In previous studies with the preferred chemical synapse between neurons, the coupling effect was estimated by adding an equivalent forcing current to each neuron. In our view, any winding or facing between synapses of two neurons can be approached by estimating the field coupling effect. Firstly, the gap junction can be thought of as an equivalent capacitor coupling in which a time-varying electric field is induced before reaching complete synchronization in the series for membrane potential. The interaction between synapse ends can be estimated by the induction current as follows:

$$i_C=\pm C'\left(\frac{\mathrm{d}v_1}{\mathrm{d}t}-\frac{\mathrm{d}v_2}{\mathrm{d}t}\right),\tag{9}$$

where v_1 and v_2 are the membrane potentials of two neurons (output voltages from the same output end of

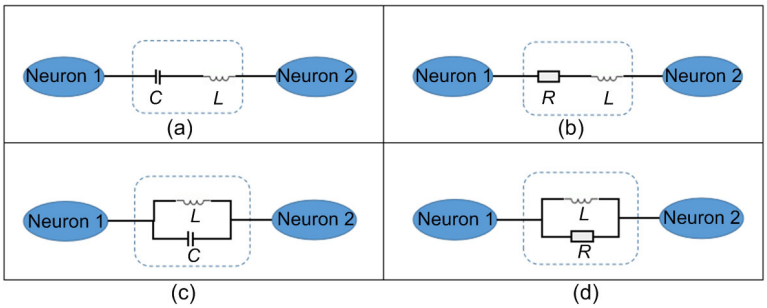

Fig. 2 Neuron connections bridged via hybrid synapses: (a) capacitor connecting inductor in series; (b) resistor connecting inductor in series; (c) capacitor connecting inductor in parallel; (d) resistor connecting inductor in parallel

the neural circuits), and C' is the equivalent capacitance for the coupling synapse (or gap junction). The symbols "+" and "–" denote the current term included in the two neurons. As a result, a time-varying electric field is generated in this gap junction and energy is pumped between the two neurons. Considering the random flow and exchange of ions in the cell, a magnetic field also can be generated due to the flow of ions, and the neuron cell has a certain inductance. In this way, a magnetic field coupling can be considered when synapses are twisted together, then an induced electromotive force and induction current will be generated to balance the two neurons. As a result, the induced electromotive force ε modulates the membrane potential by imposing the induction current as follows:

$$i_L = \pm\frac{1}{L}\int (v_1 - v_2)\mathrm{d}t, \tag{10}$$

where L is the equivalent inductance for the coupling synapse, and v_1 and v_2 denote the membrane potentials of the neurons (also the output voltages from the output ends of the neural circuits). According to Eqs. (9) and (10), the capacitance and inductance of the coupling terms enables the ability to pump energy between neurons, and thus the membrane potential to be regulated for reaching synchronization. Furthermore, when the interaction between synapses is considered as field coupling, the winding and twisting between synapses can be explained as building an effective energy harvester, which can capture field energy from external electromagnetic radiation. For example, when the neurons (or neural circuits) are exposed to electromagnetic radiation, the coupling devices or coupled synapses can capture energy as follows:

$$\begin{cases} H_C = 0.5C'(v_1 - v_2)^2 + c_1 H_{\text{ext}}, \\ H_L = 0.5Li_L^2 + c_2 H_{\text{ext}}, \end{cases} \tag{11}$$

where H_C and H_L represent the energy in the coupling capacitor and induction coil, respectively. H_{ext} represents the energy flow from electromagnetic radiation, and c_1 and c_2 are coefficients for energy absorption. These coefficients are associated with the intrinsic properties of the media and neurons. A schematic diagram of this kind of field coupling between neurons is shown in Fig. 3.

From a physical viewpoint, each neuron of a multi-layer network is embedded into a certain field node superposed by other neurons, and continuous flow between intracellular and extracellular ions will induce coexistence of a magnetic field and an electric field. Therefore, it is a challenge to discuss the collective behavior and regularity of a network when magnetic and electric field couplings between neurons are activated with different ratios. Recent studies of signal processing and communication between neurons suggest that synchronization stability and transition become more attractive when synaptic plasticity and memristive synapse function are considered (Zhang and Liao, 2017; Xu Q et al., 2018; Sharma et al., 2019; Zayer et al., 2019).

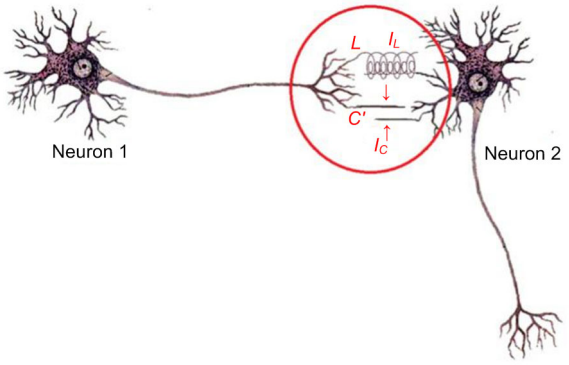

Fig. 3 Magnetic field coupling and electric field coupling between neurons whose synapses are twisted together and/or placed in parallel

5 Open problems

It is well known that the nervous system has a large number and diversity of neurons and these neurons can have different biological functions. However, most neuron models consider only a single biological function and the external stimulus is often handled as an equivalent current. Neural activities are often described by a series of membrane potentials and the anatomical structure and functional connectivity can be estimated by supplying appropriate additive current to the membrane potential. From a physical viewpoint, the electromagnetic field is often described by the electric field and magnetic field,

while many neuron models seldom involve the electromagnetic field variables directly. Therefore, it is difficult to estimate the electrical activities of neurons from a physical viewpoint in these neuron models. For an artificial and biological neuron model, auditory, visual, and perceptual effects should be included to describe the responses to acoustic, optical, or piezoelectric signals. In this way, multiple channel inputs can be processed to guide the body morphology and movements. Furthermore, when synapse coupling is explained as field coupling, it is interesting to investigate the collective responses of neural networks and the weight of the contribution of the electromagnetic field of each neuron is kept open. Noise is known to play an important role in mode transition and information encoding of neural activities. From a physical viewpoint, external noise in nonlinear and neural circuits can be considered as a stochastic disturbance resulting from electromagnetic radiation, which can be estimated by low frequency, high frequency, Gaussian white noise, and color noise. Based on our neuron model (Ma and Tang, 2015) with electromagnetic induction, electromagnetic radiation can be described by adding an appropriate magnetic flux function to the dynamical equation for magnetic flux (Lu et al., 2017; Ge et al., 2018a; Jin et al., 2019). That is, the external magnetic field can change the magnetic flux across the membrane and then the induction current can be used to estimate the electromagnetic induction. However, the physical electric field cannot be estimated directly when the magnetic field is changed.

In a summary, building a reliable neuron model with biophysical effects is critical for estimating the mode transition and coexistence of multiple modes of electrical activity. Considering the difference in perceptive function, the photoelectric, piezoelectric, and acoustoelectric conversion can be estimated to build a multifunctional neuron model. Thus, a stimulus of light, sound, or mechanical stress can be included for designing intelligent neuron sensors and processors. Also, the dependent and independent relations between different sense functions should be recognized. From the viewpoint of complex networks, path optimization in one-layer, multi-layer, and cluster networks should be reconsidered so that signal can be propagated in the most effective way, while minimizing energy consumption. The involvement of field coupling in multiscale networks gives new insights and guidance for exploring neurodynamics.

Contributors

Jun MA designed the research and presented the discussion on model setting, collective behavior of neural networks, and open problems. Zhuo-qin YANG presented the discussion on synaptic plasticity. Li-jian YANG and Jun TANG presented the discussion on astrocytes. Jun MA revised and edited the final version.

Conflict of interest

Jun MA, Zhuo-qin YANG, Li-jian YANG, and Jun TANG declare that they have no conflict of interest.

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中文概要

题　目： 从物理学角度认知计算神经动力学

目　的： 基于物理学基本原理解释神经元电活动过程中存在的物理效应，解释突触生物功能活化过程的物理机制，以及分析神经元建模中的电磁场效应（图 1）。探讨神经元建模、胶质细胞调控、突触可塑性和神经元群体电活动的网络效应。

创新点： 1. 论证荷控和磁控忆阻器非线性函数在物理神经元模型构建中的作用。2. 提出神经元突触耦合的物理机制就是电场和磁场耦合（图 3）。3. 研究神经元电路混合突触耦合的物理实现（图 2）以及能量存储与泵浦。

方　法： 依据物理学电磁感应定律和赫姆霍兹定理论证神经元电活动过程产生的电磁感应效应以及能量输运过程。基于忆阻器物理特性和量纲一致原理来构建物理神经元模型，从物理角度解释突触功能实现过程的物理机制。

结　论： 在神经元电活动过程中需考虑电磁感应效应；场耦合可以调控神经元突触耦合作用；在神经元网络中信号传递需考虑物理场耦合过程。

关键词： 神经元；神经网络；自突触；哈密顿能量；电磁感应