



Neuroelectromagnetic Activities Across Temporal Scales*

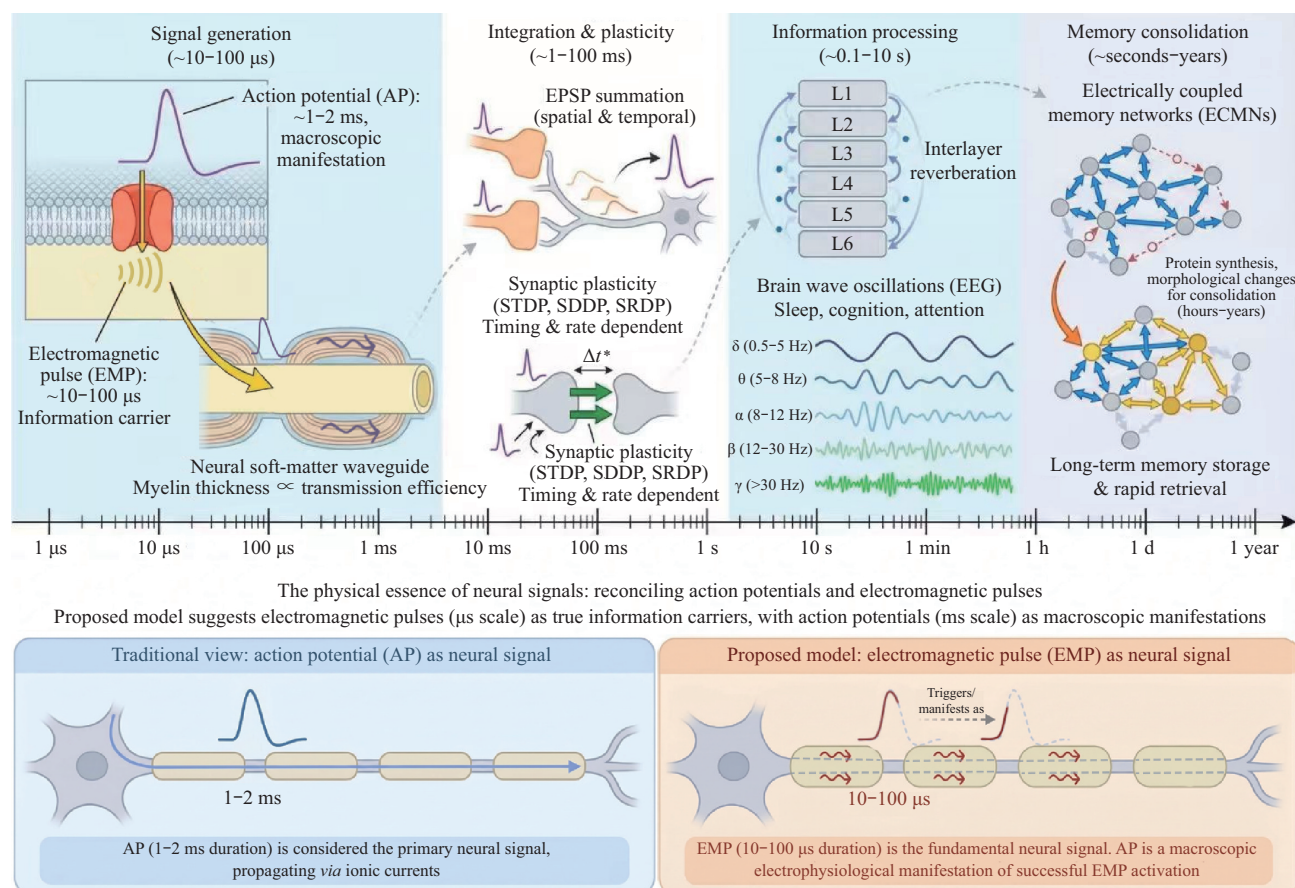
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Graphical abstract



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Abstract Although global brain science research has progressed rapidly in recent decades, several fundamental questions in neuroscience remain unresolved. In particular, the physical mechanism underlying neural signal transmission remains controversial, and the carriers responsible for neural information storage and retrieval have not yet been fully clarified. These unresolved issues motivate us to re-examine the processes of neural information generation, transmission, integration, storage, and retrieval from multiple perspectives. A key observation is that neural electromagnetic activities are closely associated with time. Their duration, temporal structure, and dynamic evolution play crucial roles in neural information processing. In this work, we analyze neural electromagnetic activities from the perspective of temporal scales (referred to here as the “time course”). By reviewing and integrating findings from previous studies, we examine the characteristic time requirements and dynamic features of neural processes occurring at different stages of information processing. These stages include neural signal generation, signal transmission along axons, synaptic integration, synaptic plasticity, and memory formation and retrieval. Based on this temporal analysis, we outline a framework describing neural electromagnetic activities across a wide range of time scales, spanning from microseconds to minutes, hours, or even longer periods associated with long-term memory, which suggests that neural information processing involves multiple physical processes operating at different time levels. Rapid electromagnetic events may occur on microsecond scales, whereas electrophysiological phenomena such as action potentials typically last on the order of milliseconds. Longer time scales are associated with synaptic plasticity and memory-related processes. From this perspective, we propose that the physical carrier of neural information may be transient electromagnetic pulses with durations on the microsecond scale. In this framework, action potentials can be interpreted as the macroscopic electrophysiological manifestation of underlying electromagnetic processes triggered by ionic currents across neuronal membranes. Rather than being the fundamental neural signal itself, the action potential may represent a measurable membrane-level response associated with the successful activation of these electromagnetic events. Moreover, we discuss a possible mechanism for long-term memory storage. Considering the apparent temporal contradiction between the millisecond-scale excitation of neurons and the long-term persistence of memories, we believe that long-term memory information may be stored within neural network topologies formed by electrical synapse coupling. Such structures, referred to as electrically coupled memory networks (ECMNs), may enable neurons within the same network to respond rapidly and synchronously to stimuli, thereby facilitating efficient memory retrieval. Overall, this study emphasizes the importance of considering the temporal organization of neural electromagnetic activities when interpreting neural signaling mechanisms. It may provide new insights into the physical nature of neural information carriers and the mechanisms of memory storage and retrieval. Furthermore, highlighting the potential role of electromagnetic interactions in neural activity may contribute to the development of new theoretical frameworks and experimental approaches in neuroscience. Such perspectives may also offer valuable references for future research on neural coding, brain function mechanisms, and neuromodulation technologies.

Key words neuroelectromagnetic activity, neural signals, information carrier, temporal scale, memory mechanism

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Time is an important reference or even determining quantity in many fields. Just as the ancient Greek philosopher Heraclitus said, “one can never step into the same river”^[1]. Time means germination, growth and aging in living organisms, but it’s more than that. From the perspectives of ecology and species, the control over time of a species basically decides the way of its perception and reaction to the outside world, which largely determines its behavior pattern and its level in the ecosystem^[2-5]. From the perspective of biological individuals, one’s ability to grasp time represents one’s speed of acquisition, processing and feedback for external information, *i. e.*, its adaptability to the environment, which usually determines the individual’s survival rate and the probability of genetic inheritance^[6-8].

There are still many unsolved mysteries in neurocommunication, or more broadly, in electrical communication which exists in most organisms, even those without a nervous system. Researchers have long found that electrical signal communication is

widely present in various living systems, not only the well-known nervous system, but also the non-neural tissues of animals, plants, and even single-celled organisms^[9-11]. Some vital issues related to bioelectrical communication, like the biological basis of consciousness and the storage and retrieval of memory in the special section “What don’t we know” raised in 2005, have not yet been fully elucidated^[12], and were emphasized in 2021 once again by *Science*^[13]. Even regarding the transmission mechanism of electrical signals, the most basic issue in biological electrical communication, there is not even an established theory, with numerous hypotheses, such as the widely accepted Hodgkin-Huxley (HH) model^[14-15] and the electromechanical soliton model developed in the same era^[16-17], the neural soft-matter waveguide model which has received increasing attention during the past decade^[18-19], and alternative hypotheses^[20-24].

Reorganizing the various processes in neural signal communication from a temporal perspective is believed to be capable of providing new insights into

bioelectrical communication, neuroscience, and related brain sciences. Most reported literature mainly focuses on the temporal characteristics of some specific behaviors, functions and physiological processes in electrical communication in biological individuals. For example, the duration of action potentials was measured as about 1–2 ms on nerves in animals^[25-26] but several seconds on plants^[27]. There is also a large body of articles devoted to discussing the importance of time scales in certain behaviors and functions, such as the determining role of temporal intervals in synaptic types^[28-31], the simultaneity requirement in synchronous responses^[32-33], the constricted time window in Venus flytrap's hunting^[34-36], and so on. Obviously, in the electromagnetic communication of living individuals, the time of information acquisition, processing and reaction has a far-reaching influence on the survival and adaptation of organisms, including animals and plants.

Taking the nervous system of animals as the example, this paper summarizes and analyzes the information processing events on various time scales (microseconds-years) and the corresponding impacts, and tries to depict the bioelectromagnetic communication framework on different time scales, in order to fully understand the physical essence of neural signal generation and transmission mechanisms.

1 Electrical communication processes on time scales

In different stages of the electromagnetic communication processes in living organisms, the time duration corresponding to a specific event varies greatly, ranging from 10 μ s to several minutes, or even years. The basic, typical but not necessarily the latest discovered electromagnetic signal communication events that occur on various time scales in the brain are summarized.

1.1 Generation of neural signals—the biological electromagnetic signals ($t=10\text{--}100\ \mu\text{s}$)

The well-accepted view is that the neural electromagnetic signals are action potentials whose duration is usually measured as 1–2 ms^[14, 37-39] and even longer, including 4 ms^[40-42], and shorter sometimes. For instance, the discharge frequency of a weak electric fish in South America was found to reach 1 500 Hz, with the duration of 0.67 ms for one action potential^[43-44]. This recognition has been gradually determined by biologists since the 1940s, due to the application of voltage clamps invented by Marmont^[45] and Cole^[46] for measuring neural

signals^[45-46]. Hodgkin and Huxley's measurement of the central axon of the squid revealed that the transmembrane voltage across the axonal membrane was reversed and lasted for about 2.0 ms when the neural signal was generated^[14], which established the idea that the action potential is the physical nature of neural signals that propagates in the form of ionic currents. Besides, several new models have been proposed in succession to complement the views of neural signals' nature. The electromechanical soliton model states that the mechanical sparse waves transmitting in membranes are truly neural signals^[17], while the emerging electromagnetic field model and neural soft-matter waveguide model demonstrated that the neural signals are electromagnetic waves with frequency higher than kHz level^[47-48], which can efficiently propagate in waveguide structures formed by the membrane and solutions on both sides.

Considering the electromagnetic signals, no matter in the form of currents or waves, travel at a speed close to that of light ($\sim 10^8$ m/s), the time consuming on the internode section can be overlooked, thus neural conduction spends most on the signal relay process at the nodes of Ranvier. With the conduction time (T) and the number of Ranvier nodes (signal relay stations) (N) on the unit length of the myelinated axons, the time for ion channels at nodes to generate a new neural signal is calculated as $\tau=T/N$. Our simulation results of ion transport after opening the sodium ionic channel also confirmed that it takes about 20 μ s for the sodium ionic channel to be activated to generate effective neural signals that can be transmitted down^[40], taking only 5% of the duration (~ 2 ms) of action potentials for neural signals to be generated^[42]. According to the above method, the time for a group of ion channels to generate a new relay signal was calculated within the range of 10–100 μ s, as listed in Table 1. This range was reported in some other work with either experimental or calculational methods^[43-44]. Considering the collection effect of thousands of channels at one node, it could be deduced to 1–10 μ s for a single ion channel and even less. The difference in this τ value across different nerves is likely dependent on the gating mechanisms and structures of different types of ion channels.

The above results were based on a large number of experimental measurement data, which did not depend on any neural communication model. In practice, this phenomenon can be explained with the neural soft-matter waveguide model very well. The mechanism of neural electromagnetic communication bears a resemblance to that of beacon communication.

Table 1 Calculation results of average signal relay time (τ) per node

Animal	Nerve name	Measurement method	Conduction speed (v)/ ($\text{m} \cdot \text{s}^{-1}$)	Internode length (L_m)/ μm	Calculated relay time (τ)/ μs
Cat ^[49]	Vagus nerve	<i>in vitro</i>	16	313	19.6
Cat ^[49]	Vagus nerve	<i>in vitro</i>	64	1 092	17.0
Cat ^[49]	Vagus nerve	<i>in vitro</i>	80	1 344	16.8
Cat ^[49]	Vagus nerve	<i>in vitro</i>	100	1 634	16.3
Cat ^[50]	Aortic nerve	<i>in vitro</i>	8	200	25.0
White rabbits ^[51]	Tibial nerve	<i>in vivo & vitro</i>	43	930	21.6
White rabbits ^[51]	Tibial nerve	<i>in vivo & vitro</i>	36.0	1 270	35.3
Mice ^[52]	Optic nerve	<i>in vitro</i>	2.64	74.39	28.2
Mice ^[52]	Optic nerve	<i>in vitro</i>	1.85	63.55	34.4
Mice ^[53]	L5 motor root	<i>in vitro</i>	50	940	18.8
Mice ^[53]	L5 motor root	<i>in vitro</i>	46	840	18.3

Sodium ion channels (groups) act as beacon towers, responsible for being stimulated by previous neural signals and generating new neural signals, which process takes 10–50 μs . It is stated in the neural soft-matter waveguide model that the downward neural signals are electromagnetic waves caused by the instantaneous influx of sodium ions, rather than the change in transmembrane potential. That is to say, the action potential is an additional semblance in the neural signals' generation process. Therefore, the generation time for neural electromagnetic signals is more likely to be 10–50 μs . The current measurement of action potential is achieved through electrophysiological techniques such as voltage clamp or patch clamp, which are designed for low-frequency (almost direct current (DC)) signal acquisition and cannot detect the electromagnetic waves generated simultaneously. There seems currently no available technology reported that can detect weak electromagnetic signals in ionic solution environments. Regarding the transmission of neural electromagnetic signals, neural soft-matter waveguides formed by the membrane and solutions on both sides serve as the pathway for efficient directional transmission of neural electromagnetic waves, as shown in Figure 1.

The advantage of the neural soft-matter waveguide model lies in the rational explanation for the difference in neural signals transmission speed on unmyelinated and myelinated nerves^[19, 54]. As electromagnetic pulse signals, the neural signals' transmission pathway in the living body is the soft-matter waveguides composed of a cell lipid membrane and its internal and external solutions. Both experimental and computational results proved that the transmission efficiency of the electromagnetic

signal is proportional to the thickness of the dielectric layer of the cell membrane in the soft-matter waveguide^[19, 55]. The thicker membrane could improve the transmission efficiency of the signal, reduce the signal attenuation speed, thus lengthen the distance of the signal relay station, *i.e.*, ion channels. As a result, the number of signal relay required in the transmission of unit length decreases, resulting in a shorter delay and the following higher propagation speed of neural signals. This model has attracted the attention of numerous research groups, and has been followed up and discussed^[55-56]. Recently, the high consistency in neural conduction characteristics between our simulation results and the biological experiments supports the neural soft-matter waveguide model indirectly^[57]. Nevertheless, it should be noted that the neural soft-matter waveguide model remains a hypothesis due to the lack of direct measurement evidence *in vivo*. This limitation is largely due to the constraints of current experimental techniques: no existing technology (such as electrophysiological recordings, optical imaging and magnetic sensing) can detect microsecond-scale electromagnetic signals in ionic environments^[58-60], while no available technology can directly resolve or isolate weak transient electromagnetic signals on the microsecond timescale within highly conductive and ion-rich biological environments. Therefore, current support for the soft-matter waveguide model is necessarily indirect, relying on the consistency between theoretical predictions, numerical simulations, and macroscopic physiological observations.

1.2 Integration of neural signals—the neural output ($t=1-100$ ms)

Although the generation of neural

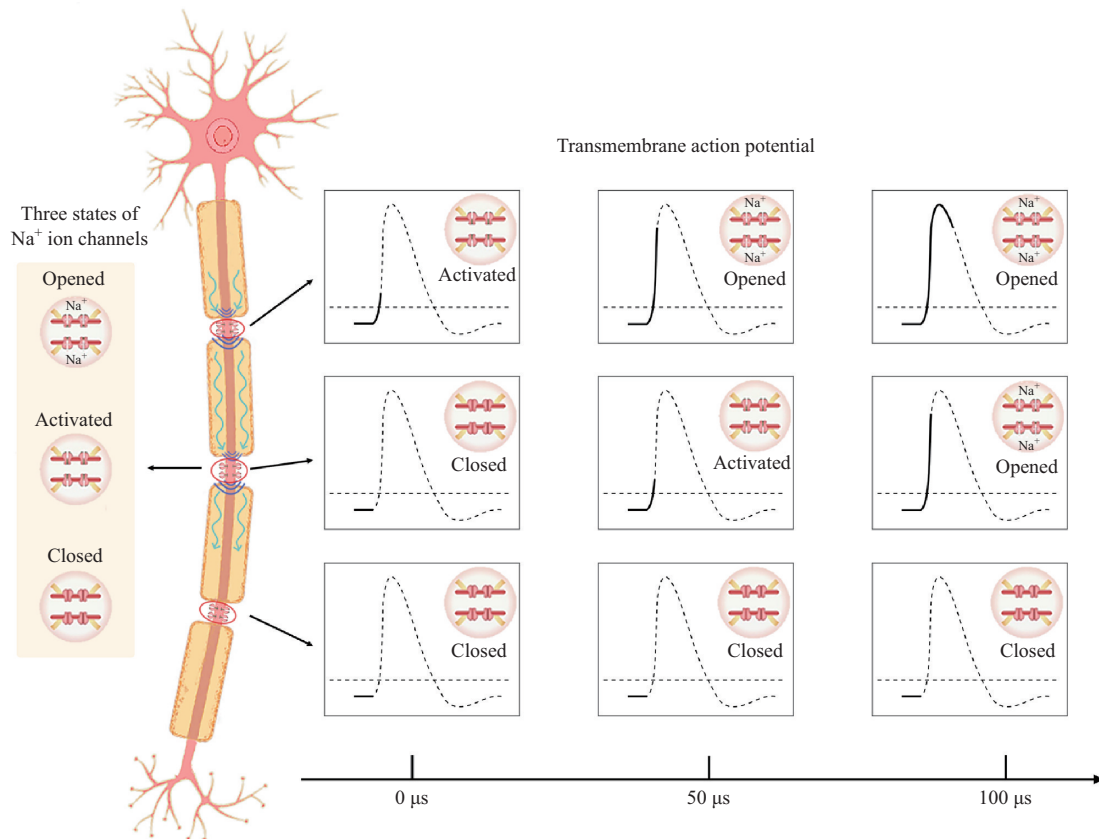


Fig. 1 The relationship and difference between neuroelectromagnetic signals and action potentials (in the proposed model)

Electromagnetic signals resulting from transmembrane ionic currents are the carrier of neural signals, while action potentials are accompanying effects in the neural signals' generation process. Action potentials remain the primary measurable electrophysiological signal, while the proposed model suggests they may be accompanied by underlying electromagnetic pulses.

electromagnetic signals only takes tens of microseconds, the processing time of the carried information is much longer than the generation time by up to three orders of magnitude. Many research results have shown that the primary information processing requires time at the level of 1–100 ms^[61–62]. Furthermore, the sensing and integration of neural information are graded in terms of energy and closely related to the integration of time and space. For instance, excitatory postsynaptic potentials (EPSPs) result from the quantal release of neurotransmitter and usually have a value of only a few millivolts, far below the amplitude of the action potential considered a neural signal^[63–64], which is the physiological basis for most neurons to perform more precise calculations. The most representative behavior is the synaptic integration, such as the synaptic integration and synaptic plasticity.

1.2.1 Synaptic integration ($t=1-10$ ms)

The dendrites of a neuron are usually the input ports of information, and the axon synapses are often the information outputs of the neuron. The received

neural signals from the dendrites determine whether there is a signal output on the axon, and the form of the output neural signal. This decision process is called synaptic integration or EPSP integration, the process by which multiple synaptic potentials combine within a single postsynaptic neuron. The result of synaptic integration determines whether the neuron outputs new signals.

EPSP summation represents the simplest form of synaptic integration in the central nervous system (CNS). There are two types of summation-spatial and temporal (Figure 2). Spatial summation is the addition of EPSPs generated simultaneously at many different synapses on a dendrite. Temporal summation is the addition of EPSPs generated at the same synapses if they occur in rapid succession. The integration window was (5.3 ± 1.8) ms ($n=12$) in the bushy cells in which the dV/dt_{thresh} was characterized, and found to be (1.4 ± 0.3) ms ($n=7$) for octopus cells^[61, 65]. This indicates the integration time window of neural signals from dendrites at the level of 1–15 ms^[28, 66–71].

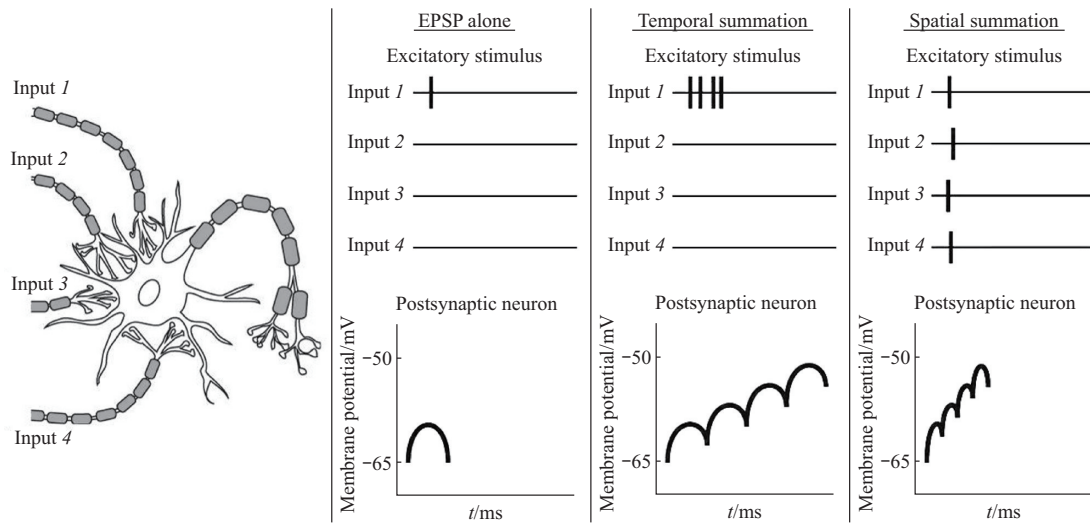


Fig. 2 The summation of excitatory postsynaptic potentials (EPSP)^[72]

Illustration of EPSP generation and summation in a postsynaptic neuron. A single presynaptic input evokes an individual EPSP (“EPSP alone”). When a presynaptic neuron fires repeatedly in rapid succession, EPSPs overlap in time, resulting in temporal summation. When multiple presynaptic inputs are activated simultaneously, their EPSPs overlap across different synaptic sites, leading to spatial summation.

1.2.2 Synaptic plasticity ($t=10\text{--}100\text{ ms}$)

EPSP summation, especially temporal synaptic integration, plays a vital role in synaptic plasticity. This influence stems from the sensitivity of synapses to spike timing. Temporary changes in synaptic transmission efficiency on a time scale of tens to hundreds of milliseconds play an important role in information encoding and processing in the brain^[73–75]. As shown in Figure 3, the following specific mechanisms are common.

Spike timing-dependent plasticity (STDP) is an embodiment of the Hebbian Learning Law. Studies on this were first proposed by Dan and Poo in 1992^[76]. STDP is a phenomenon in the neural network that regulates the weight of synaptic connections through the timing of the continuous pulses at synapses, and considered to be one of the basic principles of brain learning and memory. Neuroscientists observed STDP phenomenon in many brain regions, such as neocortical sections, hippocampal slices, cultured neurons^[77–80]. The STDP is manifested by the long-term potentiation (LTP) of synaptic connection weights observed when the presynaptic action potential is triggered approximately 50 ms earlier than the trigger of the postsynaptic action potential. The shorter the interval triggering time between the two synaptic action potentials, the stronger is the enhancement of the long-term potentiation. When the presynaptic action potential is triggered later than the trigger of the postsynaptic action potential, the

synaptic connection weights will be reduced. Similarly, the shorter the two potentials triggering interval, the more pronounced this attenuation. The process can be fitted using the following mathematical equation^[77]:

$$F(\Delta t) = \begin{cases} A_+ e^{-\Delta t/\tau_+}, & \text{if } \Delta t < 0 \\ -A_- e^{-\Delta t/\tau_-}, & \text{if } \Delta t > 0 \end{cases} \quad (1)$$

where $F(\Delta t)$ represents the time window of measured STDP for isolated spike pairs, Δt is the interval between two spikes, A is the scaling factor, τ is the time constant, “+” means LTP and “−” means long-term depression (LTD).

Markram *et al.*^[62, 81] found that the case that the pre-synaptic impulse is ahead of post-synaptic impulse within 10 ms can lead to LTP, while the case that the post-synaptic impulse be ahead of pre-synaptic impulse within 10 ms will produce long-term depression. Bi and Poo^[82] showed that the deterministic time window of STDP is $\pm 20\text{ ms}$ in cultured hippocampal neurons. There are also other experimental data for the deterministic time window. For instance, Pawlak and Kerr^[83] mentioned that the deterministic time window of $-30 - +10\text{ ms}$ in STDP when studying dopamine receptor activation and a deterministic time window of $\pm 40\text{ ms}$ in STDP has been proposed in the primary visual cortex of adult cats^[84]. Anyway, they have the timescale of tens of milliseconds.

Action potentials are the determining factors in

STDP of physiological coding events that induce synaptic plasticity. However, synaptic transmission does not necessarily trigger postsynaptic action potential, subthreshold depolarization dependent plasticity (SDDP) addresses whether transient subliminal polarization signals that do not cause long-term changes in synaptic efficacy can still induce synaptic modification^[85-88]. Studies on rat brain cortical striatal slices indicated that postsynaptic subthreshold polarization is sufficient to induce bidirectional long-term plasticity at a very high occurrence (81%) for time intervals between pre- and postsynaptic stimulations (Δt) ranging from -110 to +110 ms^[89]. SDDP greatly expands the long-term coding ability of neurons, surpassing action potentials, making neurons truly analog components. In short, postsynaptic subthreshold polarization is sufficient to induce bidirectional long-term plasticity in 100 ms, while postsynaptic action potentials play a decisive role in the strict orientation of plasticity and the accuracy of time windows with the scale of tens of milliseconds.

The synaptic weights can be regulated by the discharge frequency, which is called spike-rate-dependent plasticity (SRDP). According to the Bienenstock-Cooper-Munro (BCM) learning rule^[74], presynaptic pulses with a frequency greater than the threshold frequency θ_m will cause LTP, while presynaptic pulses below θ_m will lead to LTD. θ_m , ranging within tens of Hz^[90-93], is related to the previous synaptic activities and changes according to learning experience. For example, when Dudek *et al.*^[92] explored the patterns in the CA1 region of hippocampal slices from rats, they found that LTD was produced within the frequency range of 1–3 Hz, but no lasting change at 10 Hz, and LTP could be produced under 50 Hz pulse stimulation. Additionally, for neurons in the visual cortex of rats, θ_m is approximately 10 Hz (+10 ms), and firing (+10 ms) at frequencies of 10 Hz and above can induce LTP, with the enhancement being more pronounced at higher frequencies^[90]. This process will put the brain in an effective dynamic equilibrium state and play an important role in the brain's cognitive behavior^[94-95].

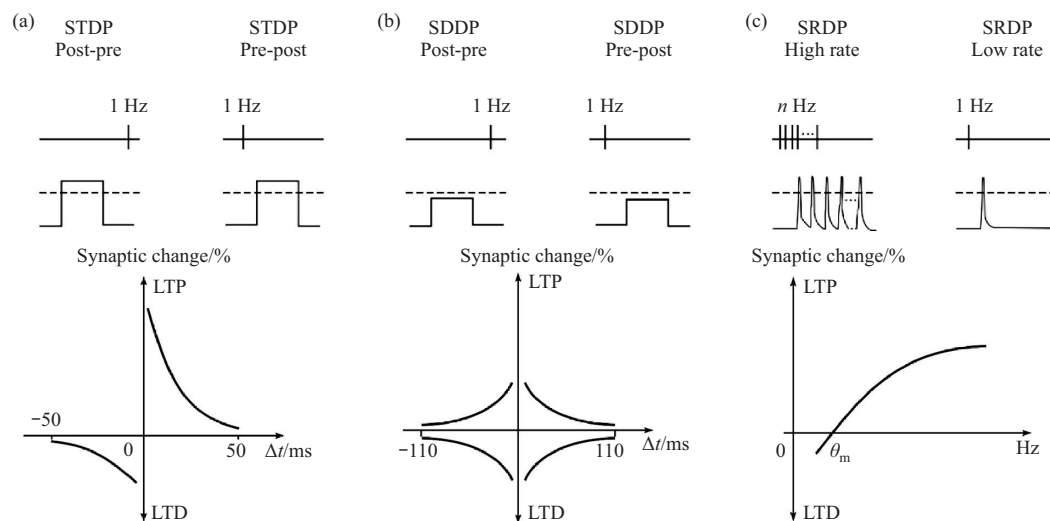


Fig. 3 Schematic representations of STDP, SDDP, and SRDP protocols and the synaptic changes they produce^[87, 89-90, 96]

(a) An illustration of post-pre and pre-post STDP protocols, its deterministic time window is within ± 50 ms. Typically, synaptic potentiation occurs when $\Delta t > 0$, while synaptic depression occurs when $\Delta t < 0$. (b) An illustration of post-pre and pre-post SDDP protocols, the deterministic time window within ± 110 ms can induce synaptic changes. (c) The protocol schematic for SRDP and the curve graph showing synaptic changes at different frequencies.

Paired-pulse facilitation (PPF) is a specific manifestation of temporal synaptic integration, that is, the postsynaptic potential caused by the posterior pulse is greater when it follows the previous pulse in a hundred-millisecond scale interval, and the shorter the interval, the greater the postsynaptic potential^[74, 97-99].

Similarly, paired-pulse depression (PPD) is another manifestation of temporal synaptic integration-synaptic inhibition^[100-101]. They are both generalized short-term synaptic plasticity phenomenon^[102-103].

1.2.3 Synfire chains

Synfire chain models describe information

transmission *via* temporally synchronized spike volleys in hierarchically organized neuron groups^[104-107]. Each layer of neurons fires nearly simultaneously upon receiving synchronous input, propagating a discharge wave. The propagation speed depends on synaptic weight distributions and input synchrony^[108]. Typically, individual synfire events last within tens of milliseconds, with interlayer delays around 1–6 ms^[109-110], enabling rapid, reliable sequential activation crucial for temporal coding.

1.3 Sustenance of neural signals—information processing ($t=0.1-10$ s)

Information consolidation manifests in the changes of synaptic type and synaptic strength. It can also be called information enhancement. This process cannot be realized during the short time like several milliseconds when one single electrical pulse come across at the synapse, because the synthesis and migration of related proteins required in information solidification takes time. It is necessary logically to increase the numbers of stimulation to change the type and/or strength of the synapses, *i. e.*, enhancing the information. The relevant process is probably related to self-oscillating behaviors like interlayer reverberation, which usually have an oscillation frequency magnitude of 0.1–10 Hz, corresponding to a single information solidification time of 0.1–10 s. Sustenance of neural signals is regarded as necessary process for short-term memory formation as well as long-term memory solidification. The well-known brain wave oscillation are to some extent the presentation of cortical current signals triggered by them^[111].

1.3.1 Predictive coding

Predictive coding theory posits that the brain continuously generates predictions of sensory inputs through a hierarchical structure and compares these predictions with actual inputs to compute prediction errors, which are then used to update internal models^[112-113]. Early studies in the cat visual cortex proposed the microcircuit basis of this theory, consisting of superficial and deep pyramidal cells as well as a common pool of inhibitory interneurons^[114]. Subsequent research revealed that intracortical connections are most frequent within the same layer, while interlaminar feedforward projections primarily run from layer 4 to layer 3, and then to layer 5^[115], consistent with the mechanism of bottom-up propagation of prediction errors and top-down transmission of predictions. Predictive coding operates across multiple timescales, with early sensory cortices processing predictions and errors within approximately 100–300 ms^[116], while higher-

order prefrontal and parietal cortices generate more abstract predictions over several seconds to tens of seconds^[117]. This hierarchical, multi-timescale predictive mechanism has been demonstrated in sensory, auditory, and language processing, underscoring its universality and significance in perception and cognition.

1.3.2 Interlayer reverberation

A new memory mechanism proposed by Xu *et al.*^[118] posits that in addition to the self-oscillation in the same neuron layer, there is also the reciprocating oscillation between layers of the cerebral cortex. As mentioned earlier, the excitation of neurons is maintained for only 1 ms which is not enough to change the connection mode between neurons. However, interlayer reverberation enables excitation to oscillate back and forth across cortical layers, prolonging effective excitation duration by increasing the number and/or frequency of excitations and promoting the formation of stronger chemical or electrical synapses^[86, 119].

The cerebral cortex has a six-layer structure in the radial direction in which external information is converted into neural electrical pulses through sensory cells and transmitted *via* axons to specific layers, forming a two-dimensional neuron array. In the present work, we define these two-dimensional neuronal assemblies that are strongly connected through electrical synapses as electrically coupled memory networks (ECMNs). ECMNs represent memory-related neural networks in which neurons are coupled through electrical synapses to form a two-dimensional topological structure capable of storing and retrieving neural information. Figure 4 illustrates the conceptual structure of such ECMNs and their potential role in memory representation and retrieval. Excitatory neuronal activity is repeatedly propagated between layers *via* vertical axons, forming temporary memory. They also mentioned that the cerebellar cortex is similarly organized into three layers from the outside to the inside: molecular layer, Purkinje cell layer and granulosal cell layer, where motor memory is formed. This is the first time that the concept of interlayer resonance has been proposed, which is worthy of further study. Recent research indicates that during memory tasks in macaques, the neuronal population activity in the frontal cortex can be decomposed into a series of subspaces for exchange, transfer, and storage^[120]. These subspaces function analogously to neuronal layers, with each subspace independently storing specific information about the activity states of prefrontal neuronal groups.

In addition to the perspective of oscillations

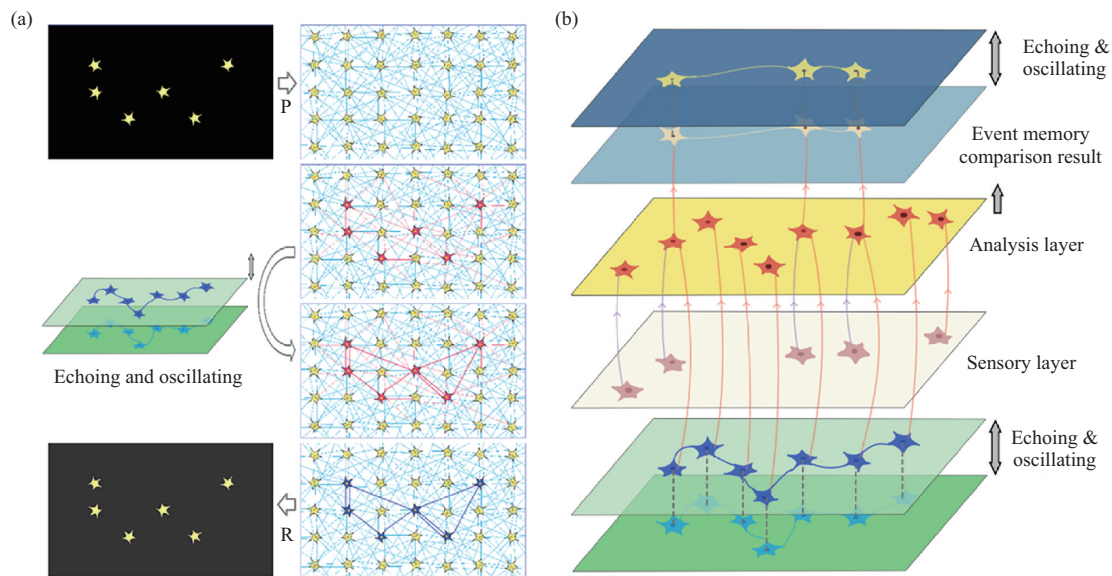


Fig. 4 Conceptual schematic of multilayered ECMNs (electrically coupled memory networks) involved in memory formation and retrieval (adapted from [118])

(a) Schematic diagram of the formation, storage, and retrieval of memory information stored in two-dimensional codes. Memory data is formed into a strongly connected neural network through interlayer projection, oscillation, and repetitive learning. Once triggered subsequently, the memory data can be retrieved. (b) During the information retrieval process, the two-dimensional code in the storage layer is projected onto the analysis layer. Comparing the data stored in the neural body pattern between the sensory layer (grey) and the memory layer (dark blue), the result can be stored in an additional memory layer. The solid curved lines with arrows represent the projection of the 2D pattern through dendrites or axons to the analysis layer.

between layers to sustain excitement, some studies have shown that the excitation of neuronal networks can be self-sustaining for a period of time^[121-124]. This is considered to be the formation stage of strong connections between neurons within the layer *via* self-sustaining oscillations that could speed up the consolidation of memory. There are even studies showing that even after a single neuron is excited, its self-sustained oscillation can be maintained for about 30 s or even longer^[125].

1.3.3 Brain wave oscillation-electroencephalogram (EEG)

Brain waves are electrical activities in the brain, and evaluate the functional activities of the brain. Researchers in Germany first observed oscillations in the visual cortex of cats^[126]. In recent years, by use of techniques such as functional magnetic resonance imaging (fMRI), synchronous oscillations have been extensively studied in terms of cognition and memory^[127-128]. Five kinds of oscillations are often discussed: delta (δ , 0.05–5 Hz), theta (θ , 5–8 Hz), alpha (α , 8–12 Hz), beta (β , 12–30 Hz), and gamma (γ , >30 Hz). The first three (delta, theta, alpha) are also called EEG slow waves and the latter two (beta, gamma) are called EEG fast waves.

EEG slow waves (usually α , θ and δ waves with

0.5–15 Hz) originate mainly from thalamic and neocortical neurons and are commonly seen during sleep. They are closely related to the consolidation of memory traces and the inhibition of cortical and subcortical structures^[129]. Delta waves occur during deep sleep, and the resulting synaptic neural oscillations can solidify information and transfer it to the neocortex. It has been shown to consolidate motor memory^[130]. Theta waves are closely associated with the interference process, and considered to provide a dynamic neural marker indicating that the memory process is encountering a higher level of interference^[131]. Alpha waves are generally thought to be related to the filtering of irrelevant information. Alpha activity produces a pulse signal repeated every 100 ms, which corresponds to pulsed inhibition^[132], reflecting cortical inhibition^[133].

EEG fast waves, including β and γ waves (15–60 Hz), are typically observed during wakefulness and rapid eye movement (REM) stage and enhance the temporal coherence of responses and the firing probability of cortical neurons^[134]. Beta waves have a generalized control related effect^[135-136]. Gamma oscillation is related to cognitive processes related to learning and memory^[137], such as sensation and perception^[138-139], information storage and retrieval,

and encoding. Gamma-synchronous oscillations play an important role in the communication across different brain regions^[140-141], and are therefore usually inactive in Alzheimer's patients. Furthermore, fast waves are probably serving as the “background refresh signals” to maintain information updating. For example, γ oscillations can be divided into spontaneous oscillations occurring in the brain without external stimuli or tasks and induced oscillations. Wherein, spontaneous γ oscillations are found to improve the coherent fluctuations of excitability in the visual cortex, ensuring faster and more reliable information transmission^[142], and transient gamma oscillations may provide a refresh mechanism to ensure coordinated memory activation^[143].

1.4 Consolidation of neural signals—long-term potentiation (LTP) (seconds–years)

The short-term potentiation of synapses is closely related to the critical computational functions of spatiotemporal information processing in biological systems, while long-term potentiation is believed to be associated with learning and memory functions. This process needs the synthesis and migration of related proteins and is regulated by chemical signals, which usually takes at least several seconds. Positive chemical signals will facilitate the formation of strong links^[74], speed up the storage of memory information,

and then enhance the strength of memory information.

Memory is a temporal segmented process in which new information is stored and consolidated^[144]. Memory is usually divided into three stages^[145-150]. The short-term memory (STM) stage lasts seconds to hours, usually independent of protein and RNA synthesis, and lacking cellular and molecular mechanisms. It is characterized by highly synchronized discharge of primary and secondary associated neurons^[151]. The second stage is long-term memory (LTM), lasting hours to days, weeks, or even months. This process typically takes 3–6 h and involves a series of specific molecular processes like synthesis and migration of RNA and proteins in the hippocampal CA1 region and its connections^[152]. The third stage is long-lasting memory, lasting years or even a lifetime.

The formation and training of both STM and LTM are immediately dependent on the integrity of AMPA, NMDA and metabotropic glutamate receptors (Figure 5)^[153-156], and their modulatory on cholinergic muscarinic, β -norepinephrine (stimulant) and GABAA receptors^[157-160]. The distinction lies in that the formation of STM does not involve gene expression and protein synthesis, whereas that of LTM includes bimodal cAMP-dependent protein kinase activity, followed by CREB phosphorylation, gene expression, and the transport of the newly

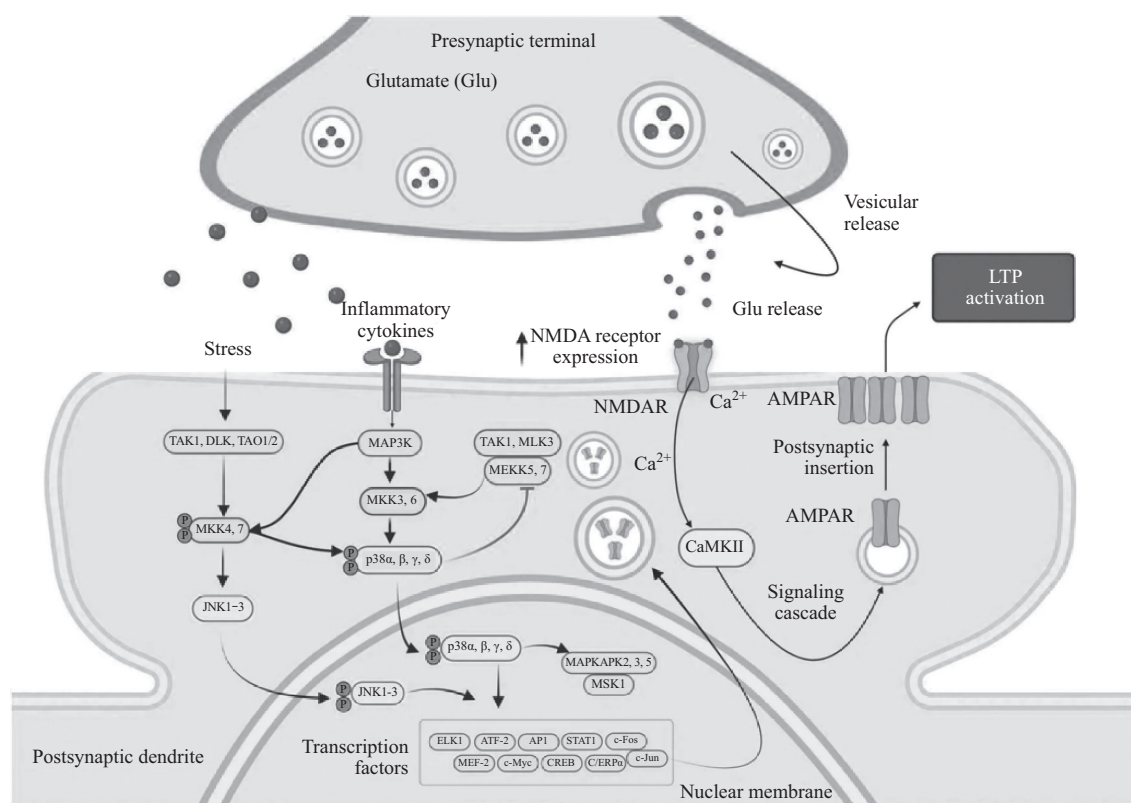


Fig. 5 The biological molecular mechanisms involved in the transformation of short-term memory to long-term memory^[156]

synthesized proteins to the appropriate location to constitute new synapses^[159, 161-162]. Therefore, there are evident synaptic morphological changes in the LTM stage, such as an increase in postsynaptic surface area, spine number, and spine density^[163]. Additionally, studies have reported changes in the distribution and quantity of synaptic vesicles and alterations in synaptic curvature associated with LTP^[164-165]. All these processes require sufficient time like several hours and even longer, as well as repetitive stimuli which may be ensured by the aforementioned

interlayer reverberation and self-sustaining in neuronal networks.

Figure 6 illustrates summarily the role of time in different levels of biological neural communication. Although derived from the mammalian neural electromagnetic communication system, the diagram is actually applicable to the electromagnetic communication behaviors in nervous and neuromorphic systems of other organisms, in line with the continuity of biological evolution.

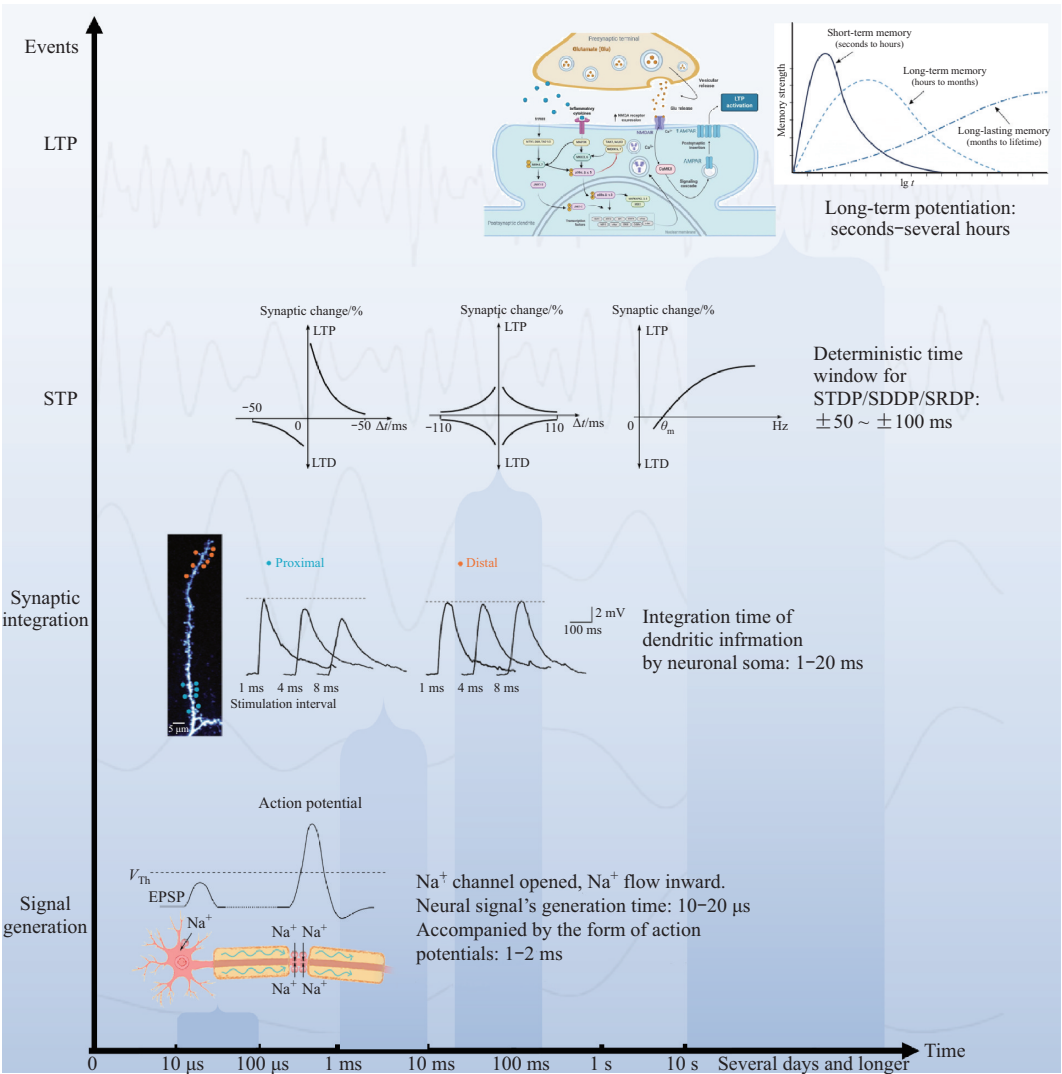


Fig. 6 Multiscale temporal hierarchy of bioelectromagnetic signaling events

The figure illustrates events in bioelectromagnetic signaling occurring on time scales ranging from several microseconds to days and even longer. Arranged in order of increasing time requirements, the events include signal generation, synaptic integration^[98], short-term potentiation^[87, 89-90, 96], long-term potentiation (LTP)^[153-154, 166].

2 Conclusion and future perspectives

2.1 Carrier of neural information

Action potentials have always been considered as

neural signals involved in information encoding and synaptic plasticity. However, the discovery and reports of some phenomena have provided different perspectives on the physical essence of neural signals.

Neural soft-matter hypothesis not only comprehensively explains the generation and transmission properties of neural signals, but also supplements the electromagnetic nature of neural information's carriers to a certain extent.

Point 1. Previous experimental studies have reported that the action potentials measured at adjacent Ranvier nodes on myelinated axons overlap highly, with the delay of only about 10 μs between adjacent action potentials^[167]. Considering that the neural electromagnetic signals propagate at nearly the speed of light, the time required for a neural signal to be generated by induction could be calculated as only about 10 μs . This indicates that, when the action potential at a certain node has not yet completed (it is actually not long after the node is activated), the new neural signal at the next node has already been generated. From this perspective, we propose that the physical essence of neural signals cannot be action potentials that last 1–2 ms.

Point 2. The reported simulation results of neural electromagnetic signal generation at the sodium ion channel exhibited that, when the channel is opened for about 10–100 μs (Figure 7), there will be a significant change of 10^6 V/m in transmembrane electric field^[47],

corresponding to a transmembrane potential increment with tens of millivolt and the rise rate of transmembrane potential with ~ 100 V/s. This is consistent with the rate of change in transmembrane potential, *i. e.*, action potential, measured by patch clamp^[168]. Based on these observations and point 1, we propose that after only 10–100 μs since the current channels were opened, the ion channels on the next node may have already sensed the neural signal and been successfully activated. This once again supports the view that the physical essence of neural signals is more likely to be electromagnetic signals caused by transient sodium ion currents.

Point 3. Synaptic integration and SDDP phenomena both show that the subthreshold depolarization can also be involved in spatial and temporal integration, and regulate synaptic plasticity. Subthreshold depolarization does not form an action potential, yet it is accompanied by changes in transmembrane electrical activity that may generate electromagnetic signals. This also indicates that compared with action potentials, neural electromagnetic signals may also serve as carriers of neural information.

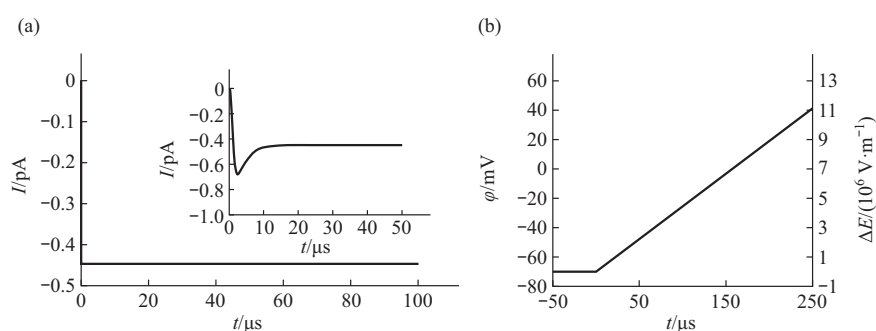


Fig. 7 The changes of transmembrane current and potential resulting from sodium ionic flux^[47]

(a) Transmembrane Na⁺ current (I) diagram. (b) Diagram of changes in transmembrane potential (ϕ) and electric field (ΔE).

In summary, a viewpoint proposed here is that the physical essence of neural signals is more likely to be electromagnetic pulses with a duration of tens of microseconds. In this framework, action potentials can be interpreted as the macroscopic electrophysiological manifestation associated with the successful activation of neural electromagnetic pulse signals, rather than the neural signal itself. This viewpoint has also been developed and discussed in theoretical work conducted by other research groups^[169]. Of course, this basically does not affect

most discoveries, inferences and applications in neuroscience that have been made using action potentials as neural signals before, because the presence or absence of action potentials usually correspond to whether the neural signal is successfully activated, which has played a significant role in evaluating the function of nerve fibers, encoding mechanisms, and so on. But this does not mean that the viewpoint that neural signals are electromagnetic pulses is meaningless. The new viewpoint is expected to lead to more efficient and

reliable brain function research and regulation technologies. For instance, it can provide the latest ideas for the exploration of neural coding mechanisms, and give a new theoretical basis for the principles and controllable use of commonly used neural regulation technologies such as light^[170-172], sound^[173], electricity^[174-175], and especially radio frequency technology^[176-177].

2.2 Hodgkin-Huxley (HH) model and the soft-matter waveguide model

The classical HH model and its extensions have provided a remarkably successful phenomenological description of action potential generation, propagation, and synaptic integration in neurons, and have underpinned decades of experimental and theoretical neuroscience. Within this framework, neural information is generally interpreted as being encoded and transmitted through ionic currents, membrane potentials, and spike timing. Importantly, the present electromagnetic pulse viewpoint in soft-matter waveguide model does not contradict these well-established electrophysiological observations; rather, it seeks to provide a deeper physical interpretation of their origin.

From this perspective, action potentials can be regarded as the macroscopic electrophysiological manifestation of underlying fast electromagnetic processes generated by transient ionic fluxes across the membrane. The HH model effectively captures the averaged, millisecond-scale dynamics of these processes, whereas the electromagnetic pulse model focuses on the microsecond-scale physical events that may trigger and shape them. At present, due to the limited temporal and electromagnetic sensitivity of available *in vivo* measurement techniques, these two descriptions cannot yet be directly reconciled experimentally. Therefore, the electromagnetic pulse hypothesis should be viewed as a complementary, physically motivated framework that extends, rather than replaces, the classical HH-based description of neural signaling.

2.3 Carriers of memory information

During the formation and storage of memory, changes in synaptic types may occur. As widely recognized, the transmission speed of electrical signals far exceeds that of chemical signals whose transmission relies on substance transport, whether it originates from diffusion under concentration gradients or drift motion under a certain electric field. There is a reported delay of 1–2 ms for chemical synapses compared to electrical synapses, which was

detected in mixed synapses (Figure 8a)^[178-179]. This conforms to the general laws of neural activities that processes like neural information conduction primarily driven by electrical signal transmission occur rapidly, while information processing and storage involving the formation of chemical synapses happen comparatively slowly. However, once information is formed and stored as memory, its retrieval is exceptionally swift, which is more like a characteristic dominated by electrical synapses. For example, the memory of the four distinct locomotion modes of *Hydra vulgaris* are stored respectively in four non-overlapping neural networks^[180]. When any neuron within a certain network is stimulated, excitations in all neurons of that network are triggered simultaneously, leading to the corresponding locomotion expression (Figure 8b). The phenomenon of neurons simultaneously excited in the same network was regarded as the behavior dominated by electrical synapses that connect neurons in the same network together^[181-183].

This aligns with our hypothesis on memory network proposed previously^[118]. Given the temporal contradiction between the extremely brief excitation time of neurons (several milliseconds) and the long-term storage of memories (lasting for hours or even decades), we conducted an extensive literature review from the perspectives of the rationality, formation conditions, and retrieval methods of neural networks connected by electrical synapses as carriers of information and memory. And we suggest that neural information is stored within neural networks joined by electrical synapses, constituting the two-dimensional topological structure, namely as ECMNs. Because electrical synapses, as opposed to chemical synapses, are better suited for ensuring that a large number of neurons in the same memory network can simultaneously respond to stimuli to recall memory scenes.

Some reports found that there are mutual conversions between electrical synapses, mixed synapses, and chemical synapses^[184-187], offering the possibility of transforming the abundant chemical synapses into electrical synapses during memory consolidation. This hypothesis is mutually supported and complemented by the interlayer reverberation mechanism in 1.3.2 that assists in the synaptic plasticity. This hypothesis requires further *in vivo* validation, such as through synchronous recording of electrosynapse-specific labeling and memory retrieval behavior.

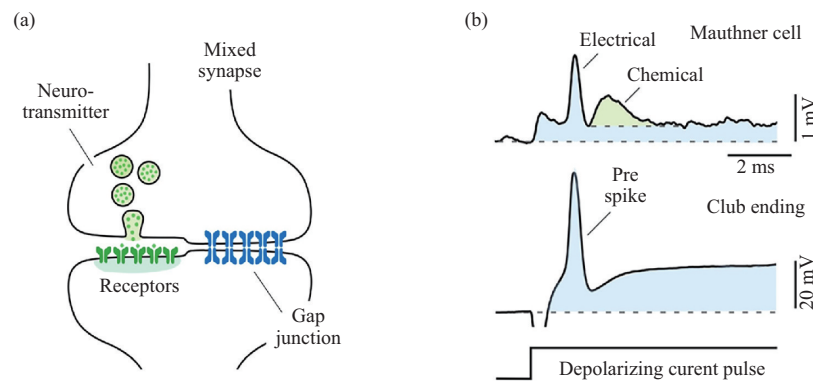


Fig. 8 Schematic diagram of mixed synapses in the goldfish brain

(a) Schematic illustration of a mixed synapse, where signals are transmitted through both neurotransmitter release and gap junction coupling^[178]. (b) Mixed synaptic transmission at club endings, showing a fast electrical response followed by a slower chemical response after presynaptic activation^[178].

2.4 Controversy between the Hodgkin–Huxley framework and the electromagnetic pulse viewpoint

In summary, this article comprehensively reviews the events occurring in the neural information processing across various time scales, ranging from 10 ms to years, including the generation of neural electromagnetic signals, the formation of action potentials, neural signal integration, short-term and long-term synaptic plasticity, and memory maintenance. We have noticed that the neural soft-matter waveguide model can comprehensively explain the phenomena and characteristics of neural signal generation and transmission, and supports that the carriers of neural signals are fundamentally more likely to be electromagnetic pulses with a duration on the order of microseconds, rather than action potentials. Here, we emphasize that the role of electromagnetics in the transmission and processing of neural signals should be highly valued, which may provide a new theoretical model for research on neural coding mechanisms and neural regulation. Nevertheless, direct experimental validation of neural electromagnetic pulse dynamics in complex biological systems remains limited, and further studies combining high-speed electrophysiological recording, advanced imaging, and multi-physics modeling are still required. In addition, how microsecond-scale electromagnetic events are coupled to millisecond neural spiking and long-term plasticity deserves deeper investigation. From an application perspective, an electromagnetic view of neural signaling may offer new insights for the optimization of neuromodulation and neural interface technologies.

References

- [1] Heraclitus. *The Art and Thought of Heraclitus: A New Arrangement and Translation of the Fragments with Literary and Philosophical Commentary*. Cambridge: Cambridge University Press, 1979: 53
- [2] Someya M, Ogawa H. Multisensory enhancement of burst activity in an insect auditory neuron. *J Neurophysiol*, 2018, **120**(1): 139-148
- [3] Liu X H, Gan L, Zhang Z T, *et al*. Probing the processing of facial expressions in monkeys *via* time perception and eye tracking. *Zool Res*, 2023, **44**(5): 882-893
- [4] Sivakumar A H, Sheldon D, Horton K G. High-latitude night shortening forces birds to accept increasingly diurnal migration to maintain flight duration. *J Avian Biol*, 2025, **2025**(4): e03405
- [5] van Dis N E, Sieperda G J, Bansal V, *et al*. Phenological mismatch affects individual fitness and population growth in the winter moth. *Proc Biol Sci*, 2023, **290**(2005): 20230414
- [6] Lee J. The effect of temporal expectation on the correlations of frontal neural activity with alpha oscillation and sensory-motor latency. *Sci Rep*, 2023, **13**(1): 2012
- [7] Xue Y, Acar M. Mechanisms for the epigenetic inheritance of stress response in single cells. *Curr Genet*, 2018, **64**(6): 1221-1228
- [8] Ekrem R K, Kokko H. Sexual conflict over phenological traits: selection for protandry can lock populations into temporally mismatched reproduction. *Evol*, 2023, **77**(3): 789-800
- [9] Li R, Yang Y, Lou H, *et al*. Electrical and calcium signaling in plant systemic defense: from local wounds to global responses. *New Phytol*, 2025, **247**(4): 1633-1642
- [10] Leal J, Shaner S, Jedrusik N, *et al*. Electrotaxis evokes directional separation of co-cultured keratinocytes and fibroblasts. *Sci Rep*, 2023, **13**(1): 11444
- [11] Jin X, Zhang X, Ding X, *et al*. Sensitive bacterial V_m sensors revealed the excitability of bacterial V_m and its role in antibiotic tolerance. *Proc Natl Acad Sci U S A*, 2023, **120**(3): e2208348120

- [12] Kennedy D, Norman C. What don't we know?. *Science*, 2005, **309**(5731): 75
- [13] Sanders S. 125 questions: Exploration and Discovery. Washington, DC, USA: Science, AAAS Custom Publishing Office, 2021:93
- [14] Hodgkin A L, Huxley A F. A quantitative description of membrane current and its application to conduction and excitation in nerve. *J Physiol*, 1952, **117**(4): 500-544
- [15] Hodgkin A L, Huxley A F. Resting and action potentials in single nerve fibres. *J Physiol*, 1945, **104**(2): 176-195
- [16] Gonzalez-Perez A, Budvytyte R, Mosgaard L D, *et al.* Penetration of action potentials during collision in the Median and lateral giant axons of invertebrates. *Phys Rev X*, 2014, **4**(3): 031047
- [17] Heimburg T, Jackson A D. On soliton propagation in biomembranes and nerves. *Proc Natl Acad Sci USA*, 2005, **102**(28): 9790-9795
- [18] Xue J, Xu S. Natural electromagnetic waveguide structures based on myelin sheath in the neural system. *arXiv* 2012. DOI: 10.48550/arXiv.1210.2140
- [19] Xu J, Xu Y, Sun W, *et al.* Experimental and computational studies on the basic transmission properties of electromagnetic waves in softmaterial waveguides. *Sci Rep*, 2018, **8**(1): 13824
- [20] Hameroff S, Penrose R. Consciousness in the universe: a review of the 'Orch OR' theory. *Phys Life Rev*, 2014, **11**(1): 39-78
- [21] Zhang X, Jiang L. Quantum-confined ion superfluid in nerve signal transmission. *Nano Res*, 2019, **12**(6): 1219-1221
- [22] Mareš J J, Špička V, Hubík P. On physical processes controlling nerve signalling. *Eur Phys J Spec Top*, 2023, **232**(20): 3561-3576
- [23] Peets T, Tamm K, Engelbrecht J. On mathematical modeling of the propagation of a wave ensemble within an individual axon. *Front Cell Neurosci*, 2023, **17**: 1222785
- [24] Abdollahi N, Prescott S A. Impact of extracellular current flow on action potential propagation in myelinated axons. *J Neurosci*, 2024, **44**(26): e0569242024
- [25] Naundorf B, Wolf F, Volgushev M. Unique features of action potential initiation in cortical neurons. *Nature*, 2006, **440**(7087): 1060-1063
- [26] Lu X, Wang Y, Liu Z, *et al.* Widefield imaging of rapid pan-cortical voltage dynamics with an indicator evolved for one-photon microscopy. *Nat Commun*, 2023, **14**(1): 6423
- [27] Armada-Moreira A, Dar A M, Zhao Z, *et al.* Plant electrophysiology with conformable organic electronics: deciphering the propagation of Venus flytrap action potentials. *Sci Adv*, 2023, **9**(30): eadh4443
- [28] König P, Engel A K, Singer W. Integrator or coincidence detector? The role of the cortical neuron revisited. *Trends Neurosci*, 1996, **19**(4): 130-137
- [29] Anisimova M, van Bommel B, Wang R, *et al.* Spike-timing-dependent plasticity rewards synchrony rather than causality. *Cereb Cortex*, 2022, **33**(1): 23-34
- [30] Morales-Weil K. The timing of excitatory and inhibitory synapses rules the cerebellar computation. *J Neurosci*, 2024, **44**(10): e1946232024
- [31] Andrade-Talavera Y, Sánchez-Gómez J, Coatl-Cuaya H, *et al.* Developmental spike timing-dependent long-term depression requires astrocyte d-serine at L2/3-L2/3 synapses of the mouse somatosensory cortex. *J Neurosci*, 2024, **44**(48): e0805242024
- [32] Leone L M, McCourt M E. Dissociation of perception and action in audiovisual multisensory integration. *Eur J Neurosci*, 2015, **42**(11): 2915-2922
- [33] Cary E, Lahdesmaki I, Badde S. Audiovisual simultaneity windows reflect temporal sensory uncertainty. *Psychon Bull Rev*, 2024, **31**(5): 2170-2179
- [34] Durak G M, Speck T, Poppinga S. Shapeshifting in the Venus flytrap (*Dionaea muscipula*): Morphological and biomechanical adaptations and the potential costs of a failed hunting cycle. *Front Plant Sci*, 2022, **13**: 970320
- [35] Burri J T, Saikia E, Läubli N F, *et al.* A single touch can provide sufficient mechanical stimulation to trigger Venus flytrap closure. *PLoS Biol*, 2020, **18**(7): e3000740
- [36] Brown W H, Sharp L W. The closing response in dionaea. *Bot Gaz*, 1910, **49**(4): 290-302
- [37] Nakanishi H, Kita H, Kitai S T. Electrical membrane properties of rat subthalamic neurons in an *in vitro* slice preparation. *Brain Res*, 1987, **437**(1): 35-44
- [38] Park K S. Electrical signal from the brain//Park K S. Humans and Electricity. Cham: Springer International Publishing, 2023: 173-197
- [39] Baloh R W. Electrophysiology in the twentieth century//Baloh R W. Brain Electricity. Cham Switzerland: Springer Nature, 2024: 303-337
- [40] Bean B P. The action potential in mammalian central neurons. *Nat Rev Neurosci*, 2007, **8**(6): 451-465
- [41] Barnett M W, Larkman P M. The action potential. *Pract Neurol*, 2007, **7**(3): 192-197
- [42] Perez R. Simulation Studies of Frequency-Dependent Action Potentials in Cell Axons[D]. Lubbock: Texas Tech University, 2023
- [43] Pappas G D, Waxman S G, Bennett M V. Morphology of spinal electromotor neurons and presynaptic coupling in the gymnotid *Sternarchus albifrons*. *J Neurocytol*, 1975, **4**(4): 469-478
- [44] Barayeu A, Schäfer R, Grewe J, *et al.* Beat encoding at mistuned octaves within single electrosensory neurons. *iScience*, 2023, **26**(7): 106840
- [45] Marmont G. Action potential artifacts from single nerve fibers. *Am J Physiol Leg Content*, 1940, **130**(2): 392-402
- [46] Cole K S. Squid axon membrane: impedance decrease to voltage clamp. *Annu Rev Neurosci*, 1982, **5**: 305-323
- [47] Wang F, Xu J, Ge Y, *et al.* Simulation of the physical process of neural electromagnetic signal generation based on a simple but functional bionic Na⁺ channel. *Chin Phys B*, 2022, **31**(6): 068701
- [48] Sergi A, Messina A, Vicario C M, *et al.* A quantum-classical model of brain dynamics. *Entropy (Basel)*, 2023, **25**(4): 592
- [49] Paintal A S. The influence of diameter of medullated nerve fibres of cats on the rising and falling phases of the spike and its recovery. *J Physiol*, 1966, **184**(4): 791-811
- [50] Paintal A S. A comparison of the nerve impulses of mammalian non-medullated nerve fibres with those of the smallest diameter

- medullated fibres. *J Physiol*, 1967, **193**(3): 523-533
- [51] Simpson A H, Gillingwater T H, Anderson H, *et al.* Effect of limb lengthening on internodal length and conduction velocity of peripheral nerve. *J Neurosci*, 2013, **33**(10): 4536-4539
- [52] Etxeberria A, Hokanson K C, Dao D Q, *et al.* Dynamic modulation of myelination in response to visual stimuli alters optic nerve conduction velocity. *J Neurosci*, 2016, **36**(26): 6937-6948
- [53] Villalón E, Barry D M, Byers N, *et al.* Internode length is reduced during myelination and remyelination by neurofilament medium phosphorylation in motor axons. *Exp Neurol*, 2018, **306**: 158-168
- [54] Gerevich Z, Kovács R, Liotta A, *et al.* Metabolic implications of axonal demyelination and its consequences for synchronized network activity: an *in silico* and *in vitro* study. *J Cereb Blood Flow Metab*, 2023, **43**(9): 1571-1587
- [55] Zhai Q, Ooi K J A, Ong C K, *et al.* Electromagnetic propagation models in nerve fibers//IEEE. 2019 IEEE 9th International Nanoelectronics Conferences (INEC). Kuching, Malaysia: IEEE, 2019: 1-4
- [56] Frede E, Zadeh-Haghighi H, Simon C. Optical polarization evolution and transmission in multi-ranvier-node axonal myelin-sheath waveguides. *IEEE Trans Mol Biol Multi Scale Commun*, 2024, **10**(4): 613-622
- [57] Xu Z, Ge Y, Wang Y, *et al.* Biophysical exploration of the neural soft-matter waveguide propagation hypothesis. *Chin J Phys*, 2026, **99**: 63-78
- [58] Brickwedde M, Anders P, Kühn A A, *et al.* Applications of OPM-MEG for translational neuroscience: a perspective. *Transl Psychiatry*, 2024, **14**(1): 341
- [59] Schröter M, Cardes F, Bui C H, *et al.* Advances in large-scale electrophysiology with high-density microelectrode arrays. *Lab Chip*, 2025, **25**(19): 4844-4885
- [60] Oikonomou E, Juli Y, Kolan R R, *et al.* A deep learning approach to real-time Markov modeling of ion channel gating. *Commun Chem*, 2024, **7**(1): 280
- [61] McGinley M J, Oertel D. Rate thresholds determine the precision of temporal integration in principal cells of the ventral cochlear nucleus. *Hear Res*, 2006, **216/217**: 52-63
- [62] Markram H, Gerstner W, Sjöström P J. Spike-timing-dependent plasticity: a comprehensive overview. *Front Synaptic Neurosci*, 2012, **4**: 2
- [63] Hunt S, Leibner Y, Mertens E J, *et al.* Strong and reliable synaptic communication between pyramidal neurons in adult human cerebral cortex. *Cereb Cortex*, 2023, **33**(6): 2857-2878
- [64] Chamberland S, Nebet E R, Valero M, *et al.* Brief synaptic inhibition persistently interrupts firing of fast-spiking interneurons. *Neuron*, 2023, **111**(8): 1264-1281.e5
- [65] Spirou G A, Kersting M, Carr S, *et al.* High-resolution volumetric imaging constrains compartmental models to explore synaptic integration and temporal processing by cochlear nucleus globular bushy cells. *Elife*, 2023, **12**: e83393
- [66] Svirskis G, Rinzel J. Influence of subthreshold nonlinearities on signal-to-noise ratio and timing precision for small signals in neurons: minimal model analysis. *Network*, 2003, **14**(1): 137-150
- [67] Magee J C. Dendritic integration of excitatory synaptic input. *Nat Rev Neurosci*, 2000, **1**(3): 181-190
- [68] Nettleton J S, Spain W J. Linear to supralinear summation of AMPA-mediated EPSPs in neocortical pyramidal neurons. *J Neurophysiol*, 2000, **83**(6): 3310-3322
- [69] Vazquez Y, Mendez B, Trueta C, *et al.* Summation of excitatory postsynaptic potentials in electrically-coupled neurones. *Neuroscience*, 2009, **163**(1): 202-212
- [70] Vladimirov N, Tu Y, Traub R D. Synaptic gating at axonal branches, and sharp-wave ripples with replay: a simulation study. *Eur J Neurosci*, 2013, **38**(10): 3435-3447
- [71] Kreeger L J, Honnuraiah S, Maeker S, *et al.* An anatomical and physiological basis for flexible coincidence detection in the auditory system. *bioRxiv*, 2024: 2024.02.29.582808
- [72] Henley C L. Postsynaptic potentials. East Lansing: Michigan State University, 2021[2026-04-20]. <https://openbooks.lib.msu.edu/neuroscience/chapter/postsynaptic-potentials/>
- [73] López Espejo M, David S V. A sparse code for natural sound context in auditory cortex. *Curr Res Neurobiol*, 2024, **6**: 100118
- [74] Deng Z, Oosterboer S, Wei W. Short-term plasticity and context-dependent circuit function: insights from retinal circuitry. *Sci Adv*, 2024, **10**(38): eadp5229
- [75] Kern F B, Chao Z C. Short-term neuronal and synaptic plasticity act in synergy for deviance detection in spiking networks. *PLoS Comput Biol*, 2023, **19**(10): e1011554
- [76] Dan Y, Poo M M. Hebbian depression of isolated neuromuscular synapses *in vitro*. *Science*, 1992, **256**(5063): 1570-1573
- [77] Froemke R C, Dan Y. Spike-timing-dependent synaptic modification induced by natural spike trains. *Nature*, 2002, **416**(6879): 433-438
- [78] Andrade-Talavera Y, Fisahn A, Rodríguez-Moreno A. Timing to be precise? An overview of spike timing-dependent plasticity, brain rhythmicity, and glial cells interplay within neuronal circuits. *Mol Psychiatry*, 2023, **28**(6): 2177-2188
- [79] McFarlan A R, Guo C, Gomez I, *et al.* The spike-timing-dependent plasticity of VIP interneurons in motor cortex. *Front Cell Neurosci*, 2024, **18**: 1389094
- [80] Martínez-Gallego I, Coatl-Cuaya H, Rodríguez-Moreno A. Astrocytes mediate two forms of spike timing-dependent depression at entorhinal cortex-hippocampal synapses. *Elife*, 2024, **13**: RP98031
- [81] Markram H, Lübke J, Frotscher M, *et al.* Physiology and anatomy of synaptic connections between thick tufted pyramidal neurones in the developing rat neocortex. *J Physiol*, 1997, **500**(Pt 2): 409-440
- [82] Bi G Q, Poo M M. Synaptic modifications in cultured hippocampal neurons: dependence on spike timing, synaptic strength, and postsynaptic cell type. *J Neurosci*, 1998, **18**(24): 10464-10472
- [83] Pawlak V, Kerr J N. Dopamine receptor activation is required for corticostriatal spike-timing-dependent plasticity. *J Neurosci*, 2008, **28**(10): 2435-2446
- [84] Yao H, Dan Y. Stimulus timing-dependent plasticity in cortical processing of orientation. *Neuron*, 2001, **32**(2): 315-323
- [85] Tomko M, Benuskova L, Jedlicka P. A voltage-based event-timing-dependent plasticity rule accounts for LTP subthreshold and

- suprathreshold for dendritic spikes in CA1 pyramidal neurons. *J Comput Neurosci*, 2024, **52**(2): 125-131
- [86] Mittermaier F X, Kalbhenn T, Xu R, *et al*. Membrane potential states gate synaptic consolidation in human neocortical tissue. *Nat Commun*, 2024, **15**(1): 10340
- [87] Fino E, Venance L. Spike-timing dependent plasticity in the striatum. *Front Synaptic Neurosci*, 2010, **2**: 6
- [88] Christie J M, Chiu D N, Jahr C E. Ca^{2+} -dependent enhancement of release by subthreshold somatic depolarization. *Nat Neurosci*, 2011, **14**(1): 62-68
- [89] Fino E, Deniau J M, Venance L. Brief subthreshold events can act as Hebbian signals for long-term plasticity. *PLoS One*, 2009, **4**(8): e6557
- [90] Sjöström P J, Turrigiano G G, Nelson S B. Rate, timing, and cooperativity jointly determine cortical synaptic plasticity. *Neuron*, 2001, **32**(6): 1149-1164
- [91] Cooper L N, Bear M F. The BCM theory of synapse modification at 30: interaction of theory with experiment. *Nat Rev Neurosci*, 2012, **13**(11): 798-810
- [92] Dudek S M, Bear M F. Homosynaptic long-term depression in area CA1 of hippocampus and effects of N-methyl-D-aspartate receptor blockade. *Proc Natl Acad Sci USA*, 1992, **89**(10): 4363-4367
- [93] Kim M H, Radaelli C, Thomsen E R, *et al*. Target cell-specific synaptic dynamics of excitatory to inhibitory neuron connections in supragranular layers of human neocortex. *Elife*, 2023, **12**: e81863
- [94] Bellingacci L, Canonichesi J, Mancini A, *et al*. Cytokines, synaptic plasticity and network dynamics: a matter of balance. *Neural Regen Res*, 2023, **18**(12): 2569-2572
- [95] Wen W, Turrigiano G G. Keeping your brain in balance: homeostatic regulation of network function. *Annu Rev Neurosci*, 2024, **47**(1): 41-61
- [96] Bi G, Poo M. Synaptic modification by correlated activity: Hebb's postulate revisited. *Annu Rev Neurosci*, 2001, **24**: 139-166
- [97] González Olmo B M, Bettles M N, DeMarsh J W, *et al*. Short-term high-fat diet consumption impairs synaptic plasticity in the aged hippocampus *via* IL-1 signaling. *NPJ Sci Food*, 2023, **7**(1): 35
- [98] Branco T, Häusser M. Synaptic integration gradients in single cortical pyramidal cell dendrites. *Neuron*, 2011, **69**(5): 885-892
- [99] Savotchenko A V, Chuyan E N. Short-term synaptic plasticity of hippocampal neural networks under experimentally-induced blood-brain barrier dysfunction. *J Evol Biochem Physiol*, 2025, **61**(4): 1200-1208
- [100] Lombardi A, Wang Q, Stüttgen M C, *et al*. Recovery kinetics of short-term depression of GABAergic and glutamatergic synapses at layer 2/3 pyramidal cells in the mouse barrel cortex. *Front Cell Neurosci*, 2023, **17**: 1254776
- [101] Neher E. Interpretation of presynaptic phenotypes of synaptic plasticity in terms of a two-step priming process. *J Gen Physiol*, 2024, **156**(1): e202313454
- [102] Vandael D, Jonas P. Structure, biophysics, and circuit function of a "giant" cortical presynaptic terminal. *Science*, 2024, **383**(6687): eadg6757
- [103] Wu W Q, Wang C F, Han S T, *et al*. Recent advances in imaging devices: image sensors and neuromorphic vision sensors. *Rare Met*, 2024, **43**(11): 5487-5515
- [104] Abeles M. Role of the cortical neuron: integrator or coincidence detector?. *Isr J Med Sci*, 1982, **18**(1): 83-92
- [105] Abeles M. *Corticonics*. Cambridge: Cambridge University Press, 1991: 232-235
- [106] Riquelme J L, Hemberger M, Laurent G, *et al*. Single spikes drive sequential propagation and routing of activity in a cortical network. *Elife*, 2023, **12**: e79928
- [107] Capek E, Ribeiro T L, Kells P, *et al*. Parabolic avalanche scaling in the synchronization of cortical cell assemblies. *Nat Commun*, 2023, **14**(1): 2555
- [108] Babadi B. Pattern propagation speed in synfire chains with excitatory-inhibitory couplings. *arXiv*, 2002. DOI: 10.48550/arXiv.physics/0202068
- [109] Miller A, Jin D Z. Potentiation decay of synapses and length distributions of synfire chains self-organized in recurrent neural networks. *Phys Rev E Stat Nonlin Soft Matter Phys*, 2013, **88**(6): 062716
- [110] Ravid Tannenbaum N, Burak Y. Shaping neural circuits by high order synaptic interactions. *PLoS Comput Biol*, 2016, **12**(8): e1005056
- [111] Bozic I. Neuro-oscillations in memory consolidation and forgotten parts of the brain: commentary on Weineret *et al.*, 2023. *Eur J Neurosci*, 2024, **59**(4): 481-482
- [112] Greco A, Moser J, Preissl H, *et al*. Predictive learning shapes the representational geometry of the human brain. *Nat Commun*, 2024, **15**(1): 9670
- [113] Gwilliams L, Marantz A, Poeppel D, *et al*. Hierarchical dynamic coding coordinates speech comprehension in the human brain. *Proc Natl Acad Sci USA*, 2025, **122**(42): e2422097122
- [114] Douglas R J, Martin K A. A functional microcircuit for cat visual cortex. *J Physiol*, 1991, **440**: 735-769
- [115] Thomson A M, Bannister A P. Interlaminar connections in the neocortex. *Cereb Cortex*, 2003, **13**(1): 5-14
- [116] Jiang Y, Komatsu M, Chen Y, *et al*. Constructing the hierarchy of predictive auditory sequences in the marmoset brain. *Elife*, 2022, **11**: e74653
- [117] Caucheteux C, Gramfort A, King J R. Evidence of a predictive coding hierarchy in the human brain listening to speech. *Nat Hum Behav*, 2023, **7**(3): 430-441
- [118] Xu S, Xu J. A Memory mechanism based on two dimensional code of neurosome pattern//BIBE. *International Conference on Biological Information and Biomedical Engineering*. Berlin: VDE, 2018: 1-5
- [119] Clausner T, Marques J, Scheeringa R, *et al*. Feature specific neuronal oscillations in cortical layers. *BioRxiv*, 2024. DOI: 10.1101/2024.07.31.605816
- [120] Tian Z, Chen J, Zhang C, *et al*. Mental programming of spatial sequences in working memory in the macaque frontal cortex. *Science*, 2024, **385**(6716): eadp6091
- [121] Hagger-Vaughan N, Kolnier D, Storm J F. Non-apical plateau potentials and persistent firing induced by metabotropic

- cholinergic modulation in layer 2/3 pyramidal cells in the rat prefrontal cortex. *PLoS One*, 2024, **19**(12): e0314652
- [122] Glasgow S D, Fisher T A J, Wong E W, *et al.* Acetylcholine synergizes with netrin-1 to drive persistent firing in the entorhinal cortex. *Cell Rep*, 2024, **43**(2): 113812
- [123] Lopez M R, Wasberg S M H, Gagliardi C M, *et al.* Mystery of the memory engram: history, current knowledge, and unanswered questions. *Neurosci Biobehav Rev*, 2024, **159**: 105574
- [124] Brahimi Y, Knauer B, Price A T, *et al.* Persistent firing in hippocampal CA1 pyramidal cells in young and aged rats. *eNeuro*, 2023, **10**(3): ENEURO.0479-ENEURO.0422.2023
- [125] Knauer B, Jochems A, Valero-Aracama M J, *et al.* Long-lasting intrinsic persistent firing in rat CA1 pyramidal cells: a possible mechanism for active maintenance of memory. *Hippocampus*, 2013, **23**(9): 820-831
- [126] Gray C M, Singer W. Stimulus-dependent neuronal oscillations in the cat visual cortex area 17. *Neuroscience*, 1987, **22**(Suppl): 434
- [127] Daume J, Kamiński J, Salimpour Y, *et al.* Persistent activity during working memory maintenance predicts long-term memory formation in the human hippocampus. *bioRxiv*, 2024: 2024.07.15. 603630
- [128] van Bree S, Levenstein D, Krause M R, *et al.* Processes and measurements: a framework for understanding neural oscillations in field potentials. *Trends Cogn Sci*, 2025, **29**(5): 448-466
- [129] Keeble L, Monaghan P, Robertson E M, *et al.* Slow-wave sleep as a key player in offline memory processing: insights from human EEG studies. *Front Behav Neurosci*, 2025, **19**: 1620544
- [130] Baena D, Gabitov E, Ray L B, *et al.* Motor learning promotes regionally-specific spindle-slow wave coupled cerebral memory reactivation. *Commun Biol*, 2024, **7**(1): 1492
- [131] Kobelt M, Waldhauser G T, Fellner M C, *et al.* Involuntary and voluntary memory retrieval relies on distinct neural representations and oscillatory processes. *PLoS Biol*, 2025, **23**(8): e3003258
- [132] Zhou Y J, Ramchandran A, Haegens S. Alpha oscillations protect working memory against distracters in a modality-specific way. *Neuroimage*, 2023, **278**: 120290
- [133] Klimesch W, Sauseng P, Hanslmayr S. EEG alpha oscillations: the inhibition-timing hypothesis. *Brain Res Rev*, 2007, **53**(1): 63-88
- [134] Bitzenhofer S H. Gamma oscillations provide insights into cortical circuit development. *Pflugers Arch*, 2023, **475**(5): 561-568
- [135] Ding Q, Lin T, Cai G, *et al.* Individual differences in beta-band oscillations predict motor-inhibitory control. *Front Neurosci*, 2023, **17**: 1131862
- [136] Lundqvist M, Miller E K, Nordmark J, *et al.* Beta: bursts of cognition. *Trends Cogn Sci*, 2024, **28**(7): 662-676
- [137] Griffiths B J, Jensen O. Gamma oscillations and episodic memory. *Trends Neurosci*, 2023, **46**(10): 832-846
- [138] Wynn S C, Townsend C D, Nyhus E. The role of theta and gamma oscillations in item memory, source memory, and memory confidence. *Psychophysiology*, 2024, **61**(9): e14602
- [139] Klaver L M F, Brinkhof L P, Sikkens T, *et al.* Spontaneous variations in arousal modulate subsequent visual processing and local field potential dynamics in the ferret during quiet wakefulness. *Cereb Cortex*, 2023, **33**(12): 7564-7581
- [140] Deng Q, Wu C, Parker E, *et al.* Mystery of gamma wave stimulation in brain disorders. *Mol Neurodegener*, 2024, **19**(1): 96
- [141] Samantzis M, Wang C, Balbi M. Gamma oscillations and their role in orchestrating balance and communication following stroke. *Neural Regen Res*, 2025, **20**(2): 477-478
- [142] Spencer K M, Nakhnikian A, Hirano Y, *et al.* The contribution of gamma bursting to spontaneous gamma activity in schizophrenia. *Front Hum Neurosci*, 2023, **17**: 1130897
- [143] Douchamps V, di Volo M, Torcini A, *et al.* Gamma oscillatory complexity conveys behavioral information in hippocampal networks. *Nat Commun*, 2024, **15**(1): 1849
- [144] Liu AA, Barr W B. Overlapping and distinct phenotypic profiles in Alzheimer's disease and late onset epilepsy: a biologically-based approach. *Front Neurol*, 2023, **14**: 1260523
- [145] McGaugh J L. Memory—a century of consolidation. *Science*, 2000, **287**(5451): 248-251
- [146] Kumar D, Li H, Das U K, *et al.* Flexible solution-processable black-phosphorus-based optoelectronic memristive synapses for neuromorphic computing and artificial visual perception applications. *Adv Mater*, 2023, **35**(28): e2300446
- [147] Gantner B N, Singh H. Short-term memory. *Nature*, 2007, **447**(7147): 916-917
- [148] Cohen-Armon M, Visochek L, Katzoff A, *et al.* Long-term memory requires PolyADP-ribosylation. *Science*, 2004, **304**(5678): 1820-1822
- [149] Berry J A, Guhle D C, Davis R L. Active forgetting and neuropsychiatric diseases. *Mol Psychiatry*, 2024, **29**(9): 2810-2820
- [150] Sridhar S, Khamaj A, Asthana M K. Cognitive neuroscience perspective on memory: overview and summary. *Front Hum Neurosci*, 2023, **17**: 1217093
- [151] Jonides J, Lewis R L, Nee D E, *et al.* The mind and brain of short-term memory. *Annu Rev Psychol*, 2008, **59**: 193-224
- [152] Bernabeu R, Bevilacqua L, Ardenghi P, *et al.* Involvement of hippocampal cAMP/cAMP-dependent protein kinase signaling pathways in a late memory consolidation phase of aversively motivated learning in rats. *Proc Natl Acad Sci USA*, 1997, **94**(13): 7041-7046
- [153] Shou T D. *Neurobiology*. 3rd ed. Beijing: Higher Education Press, 2013
- [154] Moulin T C, Schiöth H B. Excitability, synaptic balance, and addiction: the homeostatic dynamics of ionotropic glutamatergic receptors in VTA after cocaine exposure. *Behav Brain Funct*, 2020, **16**(1): 6
- [155] de León-López C A M, Carretero-Rey M, Khan Z U. AMPA receptors in synaptic plasticity, memory function, and brain diseases. *Cell Mol Neurobiol*, 2025, **45**(1): 14
- [156] Marin-Castañeda L A, Pacheco Aispuro G, Gonzalez-Garibay G, *et al.* Interplay of epilepsy and long-term potentiation: implications for memory. *Front Neurosci*, 2024, **18**: 1451740
- [157] Li H, Rodríguez-Nieto G, Chalavi S, *et al.* MRS-assessed brain GABA modulation in response to task performance and learning. *Behav Brain Funct*, 2024, **20**(1): 22

- [158] Kim S, Sohn S, Choe E S. Cofilin linked to GluN2B subunits of NMDA receptors is required for behavioral sensitization by changing the dendritic spines of neurons in the caudate and putamen after repeated nicotine exposure. *Behav Brain Funct*, 2024, **20**(1): 27
- [159] Tzilivaki A, Tukker J J, Maier N, *et al.* Hippocampal GABAergic interneurons and memory. *Neuron*, 2023, **111**(20): 3154-3175
- [160] Jami S A, Wilkinson B J, Guglietta R, *et al.* Functional and phosphoproteomic analysis of β -adrenergic receptor signaling at excitatory synapses in the CA1 region of the ventral hippocampus. *Sci Rep*, 2023, **13**(1): 7493
- [161] Das S, Lituma P J, Castillo P E, *et al.* Maintenance of a short-lived protein required for long-term memory involves cycles of transcription and local translation. *Neuron*, 2023, **111**(13): 2051-2064.e6
- [162] Yu J, Hui Y, He J, *et al.* The role of cAMP-dependent protein kinase A in the formation of long-term memory in *Bactrocera dorsalis*. *J Integr Agric*, 2024, **23**(2): 605-620
- [163] Harris K M, Kuwajima M, Flores J C, *et al.* Synapse-specific structural plasticity that protects and refines local circuits during LTP and LTD. *Philos Trans R Soc Lond B Biol Sci*, 2024, **379**(1906): 20230224
- [164] Fukaya R, Hirai H, Sakamoto H, *et al.* Increased vesicle fusion competence underlies long-term potentiation at hippocampal mossy fiber synapses. *Sci Adv*, 2023, **9**(8): eadd3616
- [165] Harris K M. Enhanced cycling of presynaptic vesicles during long-term potentiation in rat hippocampus. *J Physiol*, 2025, **603**(20): 6001-6013
- [166] Zamarreño-Ramos C, Camuñas-Mesa L A, Pérez-Carrasco J A, *et al.* On spike-timing-dependent-plasticity, memristive devices, and building a self-learning visual cortex. *Front Neurosci*, 2011, **5**: 26
- [167] Xu J J, Xu S J, Wang F, *et al.* An electromagnetic view of relay time in propagation of neural signals. *Chin Phys B*, 2021, **30**(2): 028701
- [168] Beeler G W, Reuter H. Reconstruction of the action potential of ventricular myocardial fibres. *J Physiol*, 1977, **268**(1): 177-210
- [169] Xiang Z, Tang C, Chang C, *et al.* A new viewpoint and model of neural signal generation and transmission: signal transmission on unmyelinated neurons. *Nano Res*, 2021, **14**(3): 590-600
- [170] Jiang S, Wu X, Rommelfanger N J, *et al.* Shedding light on neurons: optical approaches for neuromodulation. *Natl Sci Rev*, 2022, **9**(10): nwac007
- [171] Jin S, Li J, Jiang L, *et al.* Instant noninvasive near-infrared deep brain stimulation using optoelectronic nanoparticles without genetic modification. *Sci Adv*, 2025, **11**(24): eadt4771
- [172] Metuh P, Petersen P M, Ou Y. Recent advances in wireless optoelectronic biomedical implants. *Laser Photonics Rev*, 2024, **18**(12): 2300752
- [173] Kook G, Jo Y, Oh C, *et al.* Multifocal skull-compensated transcranial focused ultrasound system for neuromodulation applications based on acoustic holography. *Microsyst Nanoeng*, 2023, **9**: 45
- [174] Hershey A D, Lin T, Gruper Y, *et al.* Remote electrical neuromodulation for acute treatment of migraine in adolescents. *Headache*, 2021, **61**(2): 310-317
- [175] Tepper S J, Rabany L, Cowan R P, *et al.* Remote electrical neuromodulation for migraine prevention: a double-blind, randomized, placebo-controlled clinical trial. *Headache*, 2023, **63**(3): 377-389
- [176] Ellinger F. *Radio Frequency Integrated Circuits and Technologies*. Berlin, Heidelberg: Springer, 2008
- [177] Habibagahi I, Jang J, Babakhani A. Miniaturized wirelessly powered and controlled implants for multisite stimulation. *IEEE Trans Microw Theory Tech*, 2023, **71**(5): 1911-1922
- [178] Nagy J I, Pereda A E, Rash J E. On the occurrence and enigmatic functions of mixed (chemical plus electrical) synapses in the mammalian CNS. *Neurosci Lett*, 2019, **695**: 53-64
- [179] Cárdenas-García S P, Ijaz S, Pereda A E. The components of an electrical synapse as revealed by expansion microscopy of a single synaptic contact. *Elife*, 2024, **13**: e91931
- [180] Dupre C, Yuste R. Non-overlapping neural networks in *hydra vulgaris*. *Curr Biol*, 2017, **27**(8): 1085-1097
- [181] Schmitz D, Schuchmann S, Fisahn A, *et al.* Axo-axonal coupling: a novel mechanism for ultrafast neuronal communication. *Neuron*, 2001, **31**(5): 831-840
- [182] Galarreta M, Hestrin S. A network of fast-spiking cells in the neocortex connected by electrical synapses. *Nature*, 1999, **402**(6757): 72-75
- [183] Bennett M V L, Zukin R S. Electrical coupling and neuronal synchronization in the mammalian brain. *Neuron*, 2004, **41**(4): 495-511
- [184] Yu Y C, He S, Chen S, *et al.* Preferential electrical coupling regulates neocortical lineage-dependent microcircuit assembly. *Nature*, 2012, **486**(7401): 113-117
- [185] Anastasaki C, Gao Y, Gutmann D H. Neurons as stromal drivers of nervous system cancer formation and progression. *Dev Cell*, 2023, **58**(2): 81-93
- [186] Soltesz I, Losonczy A. CA1 pyramidal cell diversity enabling parallel information processing in the hippocampus. *Nat Neurosci*, 2018, **21**(4): 484-493
- [187] Wu M W, Kourdougli N, Portera-Cailliau C. Network state transitions during cortical development. *Nat Rev Neurosci*, 2024, **25**(8): 535-552

跨时间尺度的神经电磁活动*

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摘要 尽管全球脑科学研究日新月异, 但神经科学领域的一些基础性关键问题依然悬而未决。例如, 神经信号传递的机制仍存在争议, 在神经信息的载体、存储与检索等方面也尚未完全阐明。需要从多元角度, 对神经信号的生成、传递、整合、存储及检索等信息处理系列过程进行梳理。这些神经电磁活动的持续特性和动态变化模式与时间紧密相关 (简称“时程”)。本文从“时程”维度审视神经信号处理不同阶段所需的时间及相应的特征, 进而阐述对神经电磁信号传递机制, 以及神经信号载体与记忆的观点。在此基础上, 本文提出了神经信号的物理载体更可能是持续时间处于微秒量级的瞬态电磁脉冲, 而动作电位更可能是这一电磁过程在膜电生理层面的外在表现, 同时, 认为长期记忆更可能存储于由电突触连接形成的神经网络拓扑结构之中, 而非单一或某几个突触权重本身。本文强调电磁学在神经活动中的作用应被高度重视并深入探究, 这有望为神经编码与调控、大脑工作机制与记忆存储等基础及应用脑研究, 提供宝贵且具参考价值的全新视角。

关键词 神经电磁活动, 神经信号, 信息载体, 时程, 记忆机制

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