Modelling working memory in neuron-astrocyte network

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***Abstract*— Working memory is one of the most intriguing brain function phenomena that permits to store and recognize several information patterns simultaneously in the form of coherent activations of specific brain circuitries. These patterns can be recalled and, if physiologically (cognitively) significant, they can be further transferred to long term storages by cortical circuits. In the paper we show how the working memory can be effectively organized by multiscale network model composed of spiking neurons accompanied by astrocytic network. The latter serves as the temporal storage of information patterns that can be manipulated (relearned, retrieved, transferred) at the time scale of astrocytic calcium activation. In turn, the activation of the astrocyte network is possible, when coherent firing occurs in corresponding sites of the neuronal layer. We study the role of interplay the astrocyte-induced modulation of signal transmission in neural network and the Hebbian synaptic plasticity in the working memory organization. We show that modulation of synaptic communication caused by astrocytes does not exclude, but rather complements Hebbian synaptic plasticity, and they may well act in parallel. We believe this model to be a significant step in confirming the importance of non- neuron species (e.g. astrocytes) in the formation and sustainability of cognitive functions of the brain.**

Keywords*— *Astrocyte, Working Memory, Neural Network, Neuron-Astrocyte Interaction, Hebbian Synaptic Plasticity

I. Introduction

The effects of Hebbian learning in neural networks [1], consisting of various types of neurons [2] are studied in many works. Hebbian learning rule is the basis of many neural network training methods. According to this rule, learning occurs as a result of increase in the strength of connection(synaptic weight) between simultaneously active neurons. On this basis, connections often used in network are amplified, which explains the phenomenon of learning through repetition. Recent studies have revealed that an important role in the information processing play glial cells and the extracellular matrix [3,23]. A lot of research has been focused on the role of astrocytes in synaptic transmission between neurons [4,5].Experimental and theoretical studies [6–8] have shown that astrocyte can determine level of spatio-temporal coherence inactivity of accompanying neural network and be a spatio-temporal integrator of this activity. This integration leads to long-term changes in synaptic functionality of neural network. We propose a new approach to the consideration of astrocytic modulation of synaptic transmission from the point of view of the mechanism of short-term memory in neural networks. Most of the existing work on short-term memory in neural networks uses formal neurons. Such models show excellent computational performance and large memory capacity, but are not biologically relevant. The second group of works includes models of spiking neural networks, in which learning is carried out using the mechanisms of synaptic and neural plasticity (Hebbian fast synaptic plasticity, short-term synaptic plasticity, facilitation,

etc.). Such models reproduce network dynamics observed in experimental studies. Such systems can also be used in the context of working memory when using non- or slightly overlapping patterns. When using patterns with large overlaps, networks of this type are prone to the appearance of chimeras, and the mechanism of modulation of synaptic transmission by astrocytes, proposed in this paper, is one of the solutions to this problem.

In this article, we continue to develop our previous short-term memory model [9–11] and show that astrocyte-induced modulation of synaptic transmission complements Hebbian synaptic plasticity. In our model, working memory is associated with item-specific patterns of synaptic facilitation induced by astrocytes. We show that a neuron-astrocyte network is capable of loading, storing and retrieving several patterns with large overlaps.

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II. Model and Architecture of the Neuron-Astrocyte Network

The neuron-astrocyte network consists of three layers: the first layer of excitatory neurons (dimension W×H (79×79)), the second layer of inhibitory neurons (dimension $W_1 \times H_1$ (40×40)), and the third layer of astrocytes (dimension M×N (26×26)). Each astrocyte (m, n) interacts with N_a = 16 (4×4 ensemble) neurons of the first layer with an overlap in one row and one column (Fig. 1).

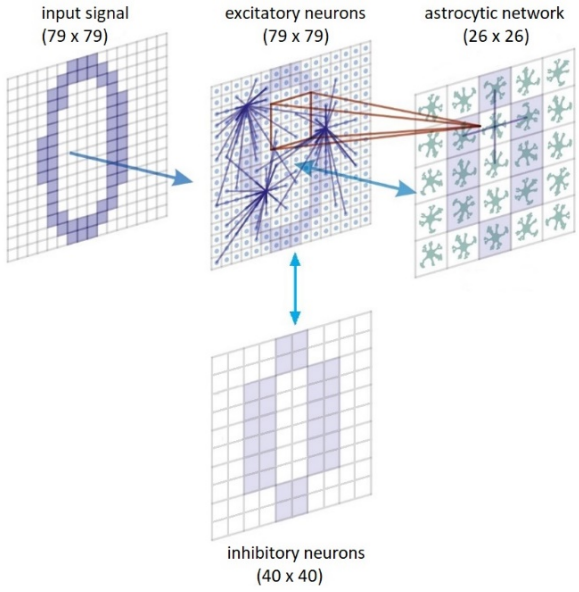

Fig. 1. The structure of the neuron-astrocyte network model.

A. Neural layers dynamics

Out of many existing dynamical models of a single neuron, we chose the Izhikevich model [13] due to its biological relevance and high computational efficiency. The membrane potential of each neuron in our network is described by:

$$\begin{cases} \frac{dV_{i,j}}{dt} = 0.04{V_{i,j}}^2 + 5V_{i,j} + 140 - U_{i,j} + I_{app_{i,j}} + I_{syn_{i,j}} \\ \frac{dU_{i,j}}{dt} = a\left(bV_{i,j} - U_{i,j}\right) \end{cases}$$
$$If\ V_{i,j} \geq 30\ mV, then\ V_{i,j} \rightarrow c,\ U_{i,j} \rightarrow U_{i,j} + d$$

where a = 0.1; b = 0.2; c = – 65 mV; d = 2. I_{app} – input signal, I_{syn} – total synaptic current.

The neurons of the first layer are connected with each other by random excitatory synaptic connections (designation of the connection: E → E, the number of output connections for each neuron: $N_{c\ E\rightarrow E}$ = 200). In addition, excitatory synaptic connections emanate from the neurons of the excitatory layer activating the neurons of the inhibitory layer (connection type: E → I, the number of output connections for each neuron: $N_{c\ E\rightarrow I}$ = 5). Output inhibitory synapses couple neurons of the inhibitory layer with neurons from the excitatory one (connection type: I → E, the number of output connections for each neuron: $N_{c\ I\rightarrow E}$ = 2000). The inhibitory neurons of the second layer are not connected.

The postsynaptic neuron is sampled from a radial exponential distribution centred in a presynaptic neuron:

$$f_R(R) = \begin{cases} 1/\lambda\ (e^{-R/\lambda}), & R \geq 0 \\ 0, & R < 0 \end{cases}$$

where λ = 15 for connection E → E, λ = 2 for connection E → I, λ = 80 for connection I → E. R – distance between pre- and postsynaptic neurons. The angle of the vector of connection R is chosen randomly.

The synaptic current for each neuron (i, j) of the excitatory layer is calculated as the sum of the total excitatory synaptic current from all its presynaptic neurons of the excitatory layer and the total inhibitory synaptic current from all its presynaptic neurons from the inhibitory layer. Thus, the synaptic current is determined by the expression [14]:

$$I_{syn}(i,j) = I_{syn\ E\rightarrow E} + I_{syn\ I\rightarrow E}$$

where:

$$I_{syn\ E\rightarrow E} = \sum_{k=1}^{N_{c\ E\rightarrow E}^{i,j}} W_{gsyn\ E\rightarrow E}(k) \cdot S(k) \cdot \left(E_{syn\ E} - V_{(i,j)}\right)$$
$$I_{syn\ I\rightarrow E} = \sum_{k=1}^{N_{c\ I\rightarrow E}^{i,j}} W_{gsyn\ I\rightarrow E}(k) \cdot S(k) \cdot \left(E_{syn\ I} - V_{(i,j)}\right)$$
$$S(k) = 1 / (1 + e^{-\frac{V_{pre}(k)}{k_{syn}}})$$

$E_{syn\ E}$ = 0 mV for excitatory connections, $E_{syn\ I}$ = –90 mV for inhibitory connections, k_{syn} = 0.2 mV, V_{pre} is the membrane potential of the presynaptic neuron, $W_{gsyn\ E\rightarrow E}$ and $W_{gsyn\ I\rightarrow E}$ – synaptic weights.

The synaptic current for each neuron (i^*, j^*) of the inhibitory layer is equal to the total excitatory synaptic current from all its presynaptic neurons of the excitatory layer. Thus, the synaptic current is determined by the expression:

$$I_{syn}(i^*, j^*) = I_{syn\ E\rightarrow I}$$

where:

$$I_{syn\ E\rightarrow I} = \sum_{k=1}^{N_{c\ E\rightarrow I}^{i,j}} W_{gsyn\ E\rightarrow I}(k) \cdot S(k) \cdot \left(E_{syn\ E} - V_{(i^*,j^*)}\right)$$

$$S(k) = 1 / (1 + e^{-\frac{V_{pre}(k)}{k_{syn}}})$$

$E_{syn\ E}$ = 0 mV for excitatory connections, k_{syn} = 0.2 mV, $V_{pre(k)}$ is the membrane potential of the presynaptic neuron, $W_{gsyn\ E\to I}$ – synaptic weight.

At the beginning of each session, the weights of synaptic connections between neurons of the excitatory layer ($W_{gsyn\ E\to E}$) and the weights of inhibitory connections ($W_{gsyn\ I\to E}$) have zero values. The weights of connections between excitatory and inhibitory neurons ($W_{gsyn\ E\to I}$) are constant throughout the session and equal to 0.1.

The training of connections between excitatory neurons is described as follows:

$$\frac{d(W_{gsyn\ E\to E})}{dt} = \delta \cdot \Delta gsyn_{EE},$$

$$\delta = \begin{cases} 1, if\ V_{pre} > 25\ mV\ and\ V_{post} > 25\ mV \\ 0, else \end{cases},$$

$$W_{gsyn\ E\to E} = [0, gsyn_{EE}]$$

If pre- and postsynaptic neurons are simultaneously active when stimulated with a training pattern (V_{pre} > 25 mV and V_{post} > 25 mV), then the weight increases by $\Delta gsyn_{EE}$ = 0.007. The maximum weight of a synaptic coupling is limited by the $gsyn_{EE}$ = 0.5. Thus, the weight of the exciting connection ($W_{gsynE\to E}$) can range from 0 to $gsyn_{EE}$. That is, if two neurons belong to the same learning pattern, then the weight of the connection between them increases by $\Delta gsyn_{EE}$. If two neurons belong to different learning patterns, then the weight of the connection between them does not change.

The training of inhibitory connections is carried out as follows: the weights of the inhibitory connections ($W_{gsyn\ I\to E}$) spanning from active neurons of the inhibitory type to those neurons of the excitatory layer that were not active during the presentation of the training pattern are increased by $\Delta gsyn_{IE}$ = 0.007. To do this, we introduce the function of the instantaneous frequency of the neuron of the excitatory layer:

$$\frac{df}{dt} = \delta A - \beta_2 f$$

where:

$$\delta = \begin{cases} 1, if\ V_{post} \geq 25\ mV \\ 0, else \end{cases}$$

$\beta_2 = 200$, $A = 0.5, V_{post}$ – membrane potential of the postsynaptic excitatory neuron. The instantaneous spiking rate is limited by an upper threshold of 1.

The dynamics of the inhibitory synaptic weight is described by the differential equation:

$$\frac{d(W_{gsyn\ I\to E})}{dt} = \delta \cdot \Delta gsyn_{IE}$$

$$\delta = \begin{cases} 1, if\ f < 0.3\ and\ V_{pre} \geq 25\ mV \\ 0, else \end{cases},$$

$$W_{gsyn\ I\to E} = [0, gsyn_{IE}]$$

Thus, the weight of the inhibitory synaptic coupling can range from 0 to $gsyn_{IE}$ = 0.05.

B. Dynamics of astrocytic layer

The astrocytic network has a dimension of 26×26. Each astrocyte is associated with the four nearest astrocytes in the network by diffusion through calcium ions (Ca^{2+}) and molecules of inositol 1,4,5-trisphosphate (IP_3).

To model the dynamics of each astrocyte, we used the Li-Rinzel model [15]. As a reduction of the biophysical De Young-Keizer model [16], this two-variable system provides low computational costs to reproduce astrocytic calcium signalization observed in experiments. The model also retains the most important dynamic features of the original system.

The dynamics of the intracellular calcium concentration in the astrocyte (m, n) is described by equations:

$$\begin{cases} \frac{dIP_3^{(m,n)}}{dt} = \frac{IP_3^* - IP_3^{(m,n)}}{\tau_{IP3}} + J_{PLC\delta}^{(m,n)} + J_{glu}^{(m,n)} + diff_{IP3}^{(m,n)} \\ \frac{dCa^{(m,n)}}{dt} = J_{ER}^{(m,n)} - J_{pump}^{(m,n)} + J_{leak}^{(m,n)} + J_{in}^{(m,n)} - J_{out}^{(m,n)} + diff_{Ca}^{(m,n)} \\ \frac{dh^{(m,n)}}{dt} = a_2\left(d_2 \frac{IP_3^{(m,n)} + d_1}{IP_3^{(m,n)} + d_3}\left(1 - h^{(m,n)}\right) - Ca^{(m,n)} h^{(m,n)}\right) \end{cases}$$

where:

$$J_{ER} = c_1 v_1 Ca^3 IP_3^3 h^3 \frac{c_0/c_1 - \left(1 + \frac{1}{c_1}\right) Ca}{((IP_3 + d_1)(IP_3 + d_5))^3}$$

$$J_{leak} = c_1 v_2 (c_0/c_1 - (1 + 1/c_1) Ca)$$

$$J_{pump} = \frac{v_3 Ca^2}{k_3^2 + Ca^2}$$

$$J_{PLC\delta} = v_4 \frac{Ca + (1 - \alpha) k_4}{Ca + k_4}$$

$$J_{in} = \frac{v_6 IP_3^2}{k_2^2 + IP_3^2}$$

$$J_{out} = k_1 Ca$$

$$diff_{IP3}^{(m,n)} = d_{IP3} (\Delta IP_3)^{(m,n)}$$

$$diff_{Ca}^{(m,n)} = d_{Ca} (\Delta Ca)^{(m,n)}$$

$$(\Delta Ca)^{(m,n)} = (Ca^{(m+1,n)} + Ca^{(m-1,n)} + Ca^{(m,n+1)} + Ca^{(m,n-1)} - 4Ca^{(m,n)}$$

$$(\Delta IP_3)^{(m,n)} = (IP_3{}^{(m+1,n)} + IP_3{}^{(m-1,n)} + IP_3{}^{(m,n+1)} + IP_3{}^{(m,n-1)} - 4IP_3{}^{(m,n)}$$

$IP_3^{(m,n)}$ – inositol 1,4,5-trisphosphate concentration, $Ca^{(m,n)}$ – intracellular concentration of calcium ions Ca^{2+}, $h^{(m,n)}$ – the proportion of non-inactivated calcium channels on the intracellular calcium storage.

The values of biophysical parameters used:

$c_0 = 2.0; c_1 = 0.185; v_1 = 6.0; v_2 = 0.11, v_3 = 2.2; v_4 = 0.3; v_6 = 0.2; k_1 = 0.5; k_2 = 1.0; k_3 = 0.1; k_4 = 1.1; d_1 = 0.13; d_2 = 1.049; d_3 = 0.9434; d_5 = 0.082; IP_3{}^* = 0.16; \frac{1}{\tau_r} = 0.14; \alpha = 0.8;\ d_{Ca} = 0.05;\ d_{IP3} = 0.1.$

III. Bidirectional Neuron-Astrocyte Interaction

In our model, we also try to implement a biologically inspired approach to reproduce bidirectional neuron-astrocyte interaction. Spiking neuronal activity induces the release of glutamate from the presynaptic terminal into the synaptic gap. The released glutamate binds to the metabotropic glutamate

receptors (mGluRs) on the astrocyte membrane and triggers the production of IP_3 in astrocytes. The amount of glutamate that diffuses from the synaptic gap and reaches the astrocyte is described by the following equation [17–18]:

$$\frac{dG^{(i,j)}}{dt} = -\alpha_{glu} G^{(i,j)} + k_{glu}\Theta\left(V^{(i,j)} - 30mV\right)$$

$$\text{if: } \frac{1}{N_a}\sum_{(i,j)\in N_a}\left[G^{(i,j)} > G_{thr}\right] > F_{act}$$

$$\text{then: } J_{glu} = \begin{cases} A_{glu}, if\ t_0 < t \le t_0 + t_{glu} \\ 0, else \end{cases}$$

where: α_{glu} = 50 s^{-1}; k_{glu} = 600 μM s^{-1}; Θ – the Heaviside function, N_a = 16; G_{thr} = 0.7; F_{act} = 0.5.

When a neuron spikes, the concentration of the neurotransmitter glutamate G increases by a constant amount and then decreases exponentially over time. More than half of the neurons associated with this astrocyte with a high glutamate concentration (exceeding the threshold value G_{thr}) produce current J_{glu} in the astrocyte. This, in turn, initiates the calcium activity of the astrocyte through the IP_3 molecules. J_{glu} is a rectangular pulse with amplitude A_{glu} = 5 μM s^{-1} and duration t_{glu} = 60 ms.

Astrocytic synaptic potentiation consists of NMDA-dependent generation of postsynaptic slow incoming currents (SICs) [19] and mGluR- dependent heterosynaptic facilitation of presynaptic glutamate release [20]. Thus, the feedback of astrocytes in our neuron-astrocyte network model simulates the neural activity of the postsynaptic neurons of the excitatory layer increasing the weights of the input connections. This occurs when two factors coincide: a calcium event in the astrocyte and the presence of spikes in at least half of the neurons associated with this astrocyte in the last 10 ms [20]. That is, taking into account astrocytic modulation, the weights of excitatory couplings between neurons in the excitatory layer can be calculated as follows:

$$W_{gsyn\,E\to E} = \eta(1 + \nu_{Ca}),$$

where: $\eta = [0 - gsyn_{EE}]$, $\nu_{Ca} = \nu^*_{Ca}\Theta(Ca^{(m,n)} - Ca_{thr})$

ν^*_{Ca} = 2 is the strength of modulation of synaptic weight modulated by astrocytes, Θ – the Heaviside function, Ca_{thr} = 0.15 μM – the threshold concentration of calcium in an astrocyte. The feedback duration is fixed and equal to τ_{astro} = 20 ms.

IV. Experiment Scheme

At the beginning of the session, we pre-trained the network as follows: 40 patterns (black-and-white binary images with a dimension of 79×79 pixels) were fed to the layer of excitatory neurons as an input signal. Each black pixel was fed to the corresponding neuron as a rectangular pulse with an amplitude of 80 μA and a duration of 0.5 ms. In the case of a white pixel, the input signal was not fed to the neuron. The interval between the presentation of the training patterns was 10 ms. The network was stimulated 10 times by each pattern with the addition of a random 5% noise ("salt and pepper" type). During the training of the network, the training patterns have been changing the weights of synaptic connections according to the Hebbian learning rule. After pre-training, the resulting synaptic weights were recorded and stored until the end of the experiment. At this stage, astrocytes did not monitor the activity of neurons.

Next, we trained the neuron-astrocyte network with 7 patterns, which were randomly selected from 40 patterns memorized by the neural network. Starting from this stage, astrocytes began monitoring the activity of neurons. The network was stimulated 10 times by each pattern with the addition of a random 5% "salt and pepper" noise. Then, after 700 ms, we trained the network for a new pattern, which was randomly selected from the remaining 33 patterns. To train the neuron-astrocyte network for a new pattern, we fed the network a 5%-noisy image 10 times as an input signal. Then the network was stimulated by the above set of 7 test patterns with 20% noise in random order. Each test pattern was applied to the input by a single rectangular pulse with an amplitude of 8 μA and a duration of 20 ms. The time interval between test patterns was 50 ms. Next, the network was stimulated by training series of 10 pulses of one more pattern from the remaining 32. The test was conducted with 7 images (6 were randomly selected from the 7 images tested during the previous step + one testing pattern that the network learned just before the last test). The procedure was repeated iteratively. Thus, there were always 7 images in the so-called "testing room", and during each test consisting of 7 patterns one random pattern would "leave the room" and one random new pattern would "enter" it. Each test sequence consisting of 7 patterns and separated from the others by a learning pattern is referred to as the "test cycle". The second part of the experiment is shown schematically in Fig. 2.

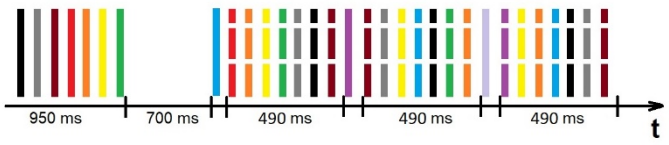

Fig. 2. Scheme of the experiment. The solid line denotes the presentation of a training packet consisting of 10 presentations of the training pattern with a random 5% noise (different every time). Different colours denote corresponding patterns. Test patterns with 20% noise are indicated by the dotted lines.

V. Results

Fig. 3 shows the images after they were processed by the neuron-astrocyte network and the corresponding correlation values of each pattern with its target pattern during 5 test cycles (the noise level in the test images was 20%).

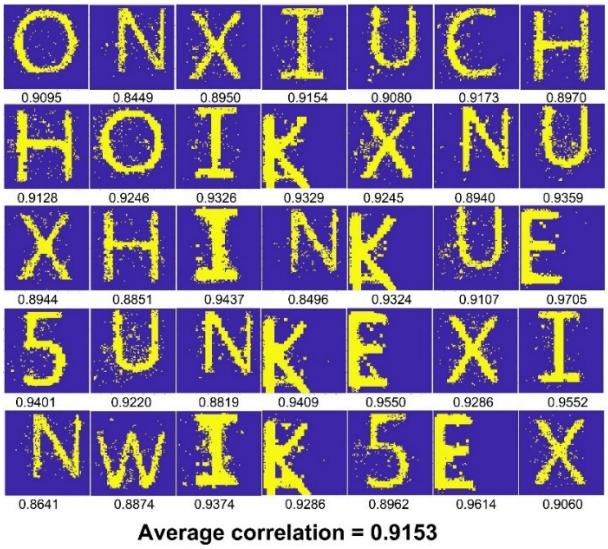

Fig. 3. The resulting images, upon presentation of test patterns with 20% noise. Neurons with a spiking rate exceeding 66 Hz during the test are shown in yellow, the rest – in blue.

To assess the contribution of astrocytes to the mechanism of working memory in the network and Hebbian pre-training of synaptic connections, we conducted this experiment with pre-training on 40 images and 20 images, with and without astrocytic modulation of synaptic signalling. The mean correlation of the resulting images with their target patterns at different noise levels in the test patterns is shown in Fig. 4.

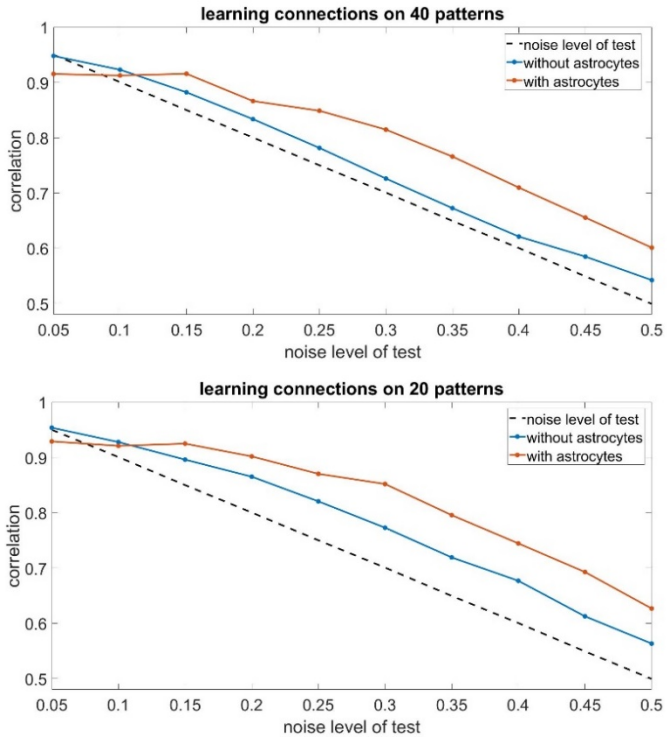

Fig. 4. Average correlation value relative to different noise levels of test patterns. The red graph corresponds to the correlation of the obtained images with the target patterns in the network with astrocytic modulation of synaptic transmission. Blue graph – without modulation of synaptic transmission by astrocytes ($\nu_{Ca}^{*} = 0$).

An increase in the correlation level of patterns with their target images is caused by the Hebbian pre-training of synaptic connections and astrocytic modulation of synaptic weights.

VI. Conclusion

In this paper, we continue to study the mechanism of theworking memory phenomenon in the neuron-astrocyte network.We have shown that two mechanisms of synaptic plasticity:astrocytic modulation of synaptic transmission between neuronsand Hebbian learning of synaptic connections work perfectlytogether, complementing each other. Using mathematicalmodeling methods we here confirmed the hypothesis thatastrocytes can be involved in the organization of workingmemory. This hypothesis emerges from numerous recentexperimental evidences about the astrocytic contributions to thecognitive functions and impairments [3, 19–22].

Acknowledgement

This research was supported by the RFBR grants No. 20-32-70081, 18-29-10068, 19-32-60051, by the Ministry of Science and Higher Education of the RF grant No. 0729-2020-0061, by the Grant of the President of the RF No. NSh-2653.2020.2.

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