Research Article

Simulations of Learning, Memory, and Forgetting Processes with Model of CA1 Region of the Hippocampus

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The aim of this paper is to present a computational model of the CA1 region of the hippocampus, whose properties include (a) attenuation of receptors for external stimuli, (b) delay and decay of postsynaptic potentials, (c) modification of internal weights due to propagation of postsynaptic potentials through the dendrite, and (d) modification of weights for the analog memory of each input due to a pattern of long-term synaptic potentiation (LTP) with regard to its decay. The computer simulations showed that CA1 model performs efficient LTP induction and high rate of sub-millisecond coincidence detection. We also discuss a possibility of hardware implementation of pyramidal cells of CA1 region of the hippocampus.

1. Introduction

Hippocampus is a neural structure located in brain in medial temporal lobe, under the cerebral cortex. Being a part of limbic system, hippocampus plays main role in cortical memory [1–4] navigation [5–10] and conditioning [11–13]. In basic hippocampal circuit, a series of narrow zones could be distinguished, part of which are Cornu Ammonis (CA) areas filled with pyramidal cells. CA1 and CA3 are proven to be areas of highest significance [14–17].

Most of reviewed models are associated with memory and imply that hippocampus is working as a homogenous network [18]. These models do not assume any differentiation among CA1 and CA3. Among numerous hippocampal models only a few specify a role for CA1; however there are many examples of synaptic integration among pyramidal cells in CA1 area presenting no connection with their basic function.

Scientific research was primarily concentrated on the potential of CA3 areas, mostly the ability of cells to autoassociate [19, 20] or to associate activity in sequences [21, 22]. Treves and Rolls [19] suggested that CA3 requires recording into a stronger code and takes benefits from this process. Otherwise, McClelland and Goddard [20] presented slightly different point of view, in which CA3 are too strong for direct association. As a result, the invertible code might cause confusion among superficial and deep layers of entorhinal cortex.

Following Marr’s inspiration [23], Treves and Rolls improved a precise and successful hippocampus memory model, in which CA3 is area associated with recurrent collaterals and memory recall [19, 24–27]. In this model possible functions of CA1 are also mentioned. It is suggested that CA1 are responsible for insurance of effective information transmission and reduction of CA3 excessive activity [28–31].

However, O’Reilly and McClelland [32] presented a slightly different expertise in which CA1 areas are required to solve the problem of associating CA3 activity with the primal entorhinal activity. McClelland and Goddard [20] developed a model, in which CA1 cells contact EC cells and have direct connections to them. Another point of view suggesting connection between CA3 cells and dentate gyrus is given by Lisman [33]. Nevertheless, Lisman and Otmaikhova [22] declared that storage of new information in hippocampus requires activation of dopamine receptor which enables temporoammonic input activity. Dopamine has ability to inhibit reaction caused by temporoammonic stimulation and simplify the induction of early LTP in the Schaffer collateral [34] without interfering in their response [35].

Implementation of CA1 presented in model given by Haselmo and Schnell [36] imputes a crucial role to acetylcholine,
which is presented as the main agent performing suppression [37]. Otherwise, Hasselmo et al. submit implication for CA1 in which the theta rhythm pertains separate phases of storage and recall in CA1 and CA3 [38].

Another model, presented by Levy, concentrates on more general or predicted function of CA1 and might not be compatible with proven activity of hippocampus [39]. A temporoammonic input activity is suggested to take place in CA1 and is associated with activity in CA3 through the Schaffer collaterals. Furthermore, it could be possible that that temporoammonic input blocks Schaffer collateral vividness in order to determine which active CA1 cells can be connected with active CA3 cells. In this model CA1 are viewed as a decoder of CA3 activity, like subiculum and entorhinal cortex, while CA3 recurrent collaterals simplify the preservation of sequences [40]. The prediction of existing dependent on time plasticity in the Schaffer collaterals is examined and supported by Nishiyama et al. [41].

On the contrary, Lorincz and Buzsaki [18] suggest that the mismatch between the current input and events recalled by hippocampus is calculated in the entorhinal cortex. The contribution to CA1 is observed during activity by using delta rule [42]. Those hypothetical learning rules based on mathematics are given by Lorincz [43]; however in the previous version of this theory there is no precise input to CA1 [44].

The hippocampus is an area in human brain, which becomes activated in order to process temporal orders of events. CA1 involve this region in the memory of objects, odors, and, what is more important, their sequences [45]. Another promising conclusion might be a relation of temporal delays in the neural circuitry of the medial entorhinal cortex to temporal adjustment process, which could result in the various volume of spatial grids found in the medial entorhinal cortex [46]. Various types of neuron cells’ firing rates are high at different times, within a trial or delay period [47–49].

The role of hippocampus in contextual learning of objects recognition must be also mentioned [50]. And even simply models of hippocampal circuits could prove new explanations in human pathology such as Alzheimer disease or drugs art of work. It is well established that the connections from entorhinal cortex layer 2 to hippocampus play a crucial role in development of CA1. Cannabionoids disrupt memory encoding by functionally isolating hippocampal CA1 from CA3 [52].

In Section 2, we introduce mathematical model of CA1 region of the hippocampal microcircuit and discuss the methods simulation CA1 (Sections 2.1 and 2.2). Additionally, in Sections 2.3–2.5, we describe mathematical presentations: pyramidal, basket, and O-LM cells of CA1 region, while, in Sections 2.6–2.7, we present CA1 network inputs and synaptic properties glutamate and GABA receptors. In Section 3, we present the results of the paper. In Section 4, we fully discuss our results. Section 5 summarizes the conclusions.

2. Materials and Methods

2.1. The Model Description. The CA1 microcircuit is presented of Figure 1. Our simulations of the hippocampus are based on computational models from previous studies [53, 54]. There are four pyramidal cells (P1, P2, P3, and P4), two basket (B1, B2), one O-LM cell (inhibitory interneurons), and 3 independently programmed theta rhythm generators. Such sparse network with strictly topographically related connections is very similar to the CA1 net used by Hasselmo and Cutsuridis [55]. And in our opinion it has more biological plausibility as compared to previous Cutsuridis network with 100 pyramidal cells and nearly to all interconnections [56].

We have used the theta oscillation in our previous studies [53, 54], which were based on faster gamma-frequency oscillations [1, 57–60], spatial information [61–63], in-time locking cell activities [64], and regulation of learning facilities [1, 65–67].

The MS-DBB (Medial Septum-Diagonal Band of Broca) has been classically viewed as the hippocampal theta rhythm generator [57, 64, 68]. However, the role of the MS-DBB in hippocampal theta oscillations must be revised in light of recent discovery that the hippocampus itself can generate a theta-frequency rhythm independent of the MS-DBB [69]. Huh et al. suggest that the MS-DBB is one of several extrinsic rhythm generators that amplify and regulate intrinsic theta generators within the hippocampus [70]. Hence, the hippocampal theta rhythm recorded in vivo may be a product of several interacting intrinsic and extrinsic theta generators working in concert. It remains to be elucidated what role glutamatergic, GABAergic, and most importantly cholinergic MS-DBB neurons [71] play in these interactions; the understanding of these matters will bring new insights into the mechanisms underlying functions such as spatial learning and memory. In our model we have employed the most simple theta generators, which depict the basic Wang [68] suggestions, but we have not considered the proposals of Huh et al. suggest that the MS-DBB is one of several theta-frequency rhythm independent of the MS-DBB [69]. The T1, T2, and T3 theta generators send the series of 8 bursts every second; it means 8 Hz theta frequency. Each burst consists of 5 spikes at 100 Hz; for T2 and T3 we have a faze delay of 10 and 20 milliseconds for burst activity.

2.2. Art of Work. The mathematical description of equations and parameters that we used in our simulations was based on our previous studies on single neuron model [53] and sparse CA3 network model [54]. All mathematical descriptions of CA1 model neurons are presented in Table 1.

2.3. CA1 Pyramidal Cells. Every CA1 pyramidal cell consists of 16 compartments in which each dendrite has an excitatory or inhibitory synapse. There are glutamate receptors for excitatory inputs: AMPA - E (k, i), NMDA - M (k, i). GABA receptors are for inhibitory inputs: I (k, i) while k is the number of dendrite compartments and i is the number of area register table. There are dendrites constructed within a course of compartments. Each CA1 cell receives somatic synaptic inhibition from CA1 basket cells, proximal excitation from CA1 pyramidal cells, mid-dendritic excitation from Mossy Fibers, and distal apical excitation from the layer 3 entorhinal cortex [73, 74].
2.4. CAI Basket Cells. Every CAI basket cell consists of 16 compartments in which each dendrite has an excitatory or inhibitory synapse. There are glutamate receptors for excitatory inputs: AMPA - E (k, i), NMDA - M (k, i). GABA receptors are for inhibitory inputs: I (k, i) while k is the number of dendrite compartments and i is the number of area register table. There are dendrites constructed within a course of compartments. Each basket cell receives somatic synaptic inhibition from the medial septum (theta oscillations) and neighboring basket cells in their soma. Excitatory connections are received to their distal dendrites form layer 3 entorhinal cortex and to medium dendrites from both CAI pyramidal cells and CA3 Schaffer collaterals.

2.5. CAI O-LM Cells. Every CAI O-LM cell consists of 16 compartments in which each dendrite has an excitatory or inhibitory synapse. There are glutamate receptors for excitatory inputs: AMPA - E (k, i), NMDA - M (k, i). GABA receptors are for inhibitory inputs: I (k, i) while k is the number of dendrite compartments and i is the number of area register table. There are dendrites constructed within a course of compartments. Each O-LM cell receives excitatory and inhibitory connections. First ones were received from active CAI cells, whereas second ones were received from the medial septum (theta oscillations: T1, T2, and T3).

2.6. Model Inputs. According to Witter sources of inputs to CA1 are Mossy Fibers and entorhinal cortex layers III [75] as well as disinhibitory theta input from medial septal area. Every CA1 pyramidal cell input from Mossy Fibers was presented as the firing at an average frequency of 44 Hz and from layer 3 entorhinal cortex of 24 Hz. Each CA1 basket cell input from CA3 Schaffer collaterals was modeled as firing at an average frequency of 50 Hz and from the medial septum at 8 Hz theta rhythm. All initial parameters of microcircuit model of CA1 network are presented in Table 2. Pyramidal cells received somatic synaptic inhibition from CA1 basket cells (B1, B2), proximal excitation from CA1 pyramidal cells (P1, P2, P3, and P4), mid-dendritic excitation from Mossy Fibers (MF), and distal apical excitation from the layer III entorhinal cortex (EC). Basket cell received somatic synaptic inhibition from the medial septum (theta oscillations: T1, T2, and T3). Excitatory connections are received from both CA1 pyramidal cells (P1, P2, P3, and P4) and CA3 Schaffer
Table 1: The most important mathematical issues of the model cells of CA1 region.

<table>
<thead>
<tr>
<th>Name of functions</th>
<th>Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synaptic</td>
<td>[ SF(t) = \begin{cases} 0 &amp; t = t_{sd} \ \frac{A_{\text{MAX}}}{t_d} (t - t_{sd}) &amp; t_{sd} &lt; t \leq t_r \ \frac{A_{\text{MAX}}}{t_d} \left( t_d - (t - (t_r + t_{sd})) \right) &amp; t_r &lt; t \leq t_d \end{cases} ]</td>
</tr>
<tr>
<td>Summarized potential</td>
<td>[ S(k, i + 1) = \text{ReP} + \sum_{m=1}^{\text{NE}} (E(m, i) - \text{ReP}) \inf (m, k) ]</td>
</tr>
<tr>
<td>Memory</td>
<td>[ \text{MEM}(k, i) = 1 + \ln \frac{C(k, i + 1)}{6c\log} ]</td>
</tr>
<tr>
<td>Power function</td>
<td>[ \text{power} = \text{powerA} (M(k; i) - \text{ReP}) ]</td>
</tr>
<tr>
<td>Time of memory duration</td>
<td>[ C(k, i + 1) = C(k, i) + e^{\text{power} - 1} ]</td>
</tr>
<tr>
<td>Summarized postsynaptic potential in neuron</td>
<td>[ \text{PSP}(i) = \text{ReP} + \sum_{m=1}^{\text{NE}} W(k) (E(m, i) - \text{ReP}) + \sum_{m=1}^{\text{NI}} (I(m, i) - \text{ReP}) ]</td>
</tr>
<tr>
<td>Threshold function for action potential</td>
<td>[ \text{out} = \begin{cases} 0 &amp; \text{PSP} &lt; \text{threshold} \ 1 &amp; \text{PSP} \geq \text{threshold} \end{cases} ]</td>
</tr>
</tbody>
</table>

Table 2: Initial parameters microcircuit model of CA1 network.

<table>
<thead>
<tr>
<th>CA1 cells</th>
<th>LTP (Memory)</th>
<th>NE</th>
<th>NI</th>
<th>Threshold</th>
<th>ReP</th>
<th>LSW</th>
<th>CaMT</th>
<th>EPSPd</th>
<th>IPSPd</th>
<th>FQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyramidal cell (P1)</td>
<td>on</td>
<td>13</td>
<td>3</td>
<td>-50</td>
<td>-80</td>
<td>0.2</td>
<td>-68</td>
<td>4.5</td>
<td>-6</td>
<td>10</td>
</tr>
<tr>
<td>Pyramidal cell (P2)</td>
<td>on</td>
<td>13</td>
<td>3</td>
<td>-50</td>
<td>-80</td>
<td>0.2</td>
<td>-68</td>
<td>4.5</td>
<td>-6</td>
<td>10</td>
</tr>
<tr>
<td>Pyramidal cell (P3)</td>
<td>on</td>
<td>13</td>
<td>3</td>
<td>-50</td>
<td>-80</td>
<td>0.2</td>
<td>-68</td>
<td>4.5</td>
<td>-6</td>
<td>10</td>
</tr>
<tr>
<td>Pyramidal cell (P4)</td>
<td>on</td>
<td>13</td>
<td>3</td>
<td>-50</td>
<td>-80</td>
<td>0.2</td>
<td>-68</td>
<td>4.5</td>
<td>-6</td>
<td>10</td>
</tr>
<tr>
<td>Basket cell (B1)</td>
<td>off</td>
<td>13</td>
<td>3</td>
<td>-50</td>
<td>-80</td>
<td>1</td>
<td>-68</td>
<td>4</td>
<td>-4.5</td>
<td>10</td>
</tr>
<tr>
<td>Basket cell (B2)</td>
<td>off</td>
<td>13</td>
<td>3</td>
<td>-50</td>
<td>-80</td>
<td>1</td>
<td>-68</td>
<td>4</td>
<td>-5.5</td>
<td>10</td>
</tr>
<tr>
<td>O-LM cell</td>
<td>off</td>
<td>13</td>
<td>3</td>
<td>-50</td>
<td>-80</td>
<td>0.6</td>
<td>-68</td>
<td>4</td>
<td>-4</td>
<td>10</td>
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</table>

collaterals (SC). O-LM cells receive excitatory and inhibitory connections. First ones are received from active CA1 cells (P1, P2, P3, and P4), whereas second ones are received from the basket cells (B1, B2). Hippocampal formation of CA1 microcircuit connections is presented in Table 3. As compared with the previous model of CA3 microcircuit, we have inputs from layer III of entorhinal cortex instead of those from layer II (*). The Mossy Fibers (**) subsequently diminish in range; instead we have intermingled input from pyramidal cells with each other through the Schafer collaterals (Table 3).

2.7 Synaptic Properties. The mathematical description of the glutamate receptors AMPA, NMDA, and the GABA receptor was based on our previous studies [53, 54]. The real value of postsynaptic potential is estimated by using functions from Table 1. During these studies all pyramidal cells had the same LSW parameter (0.2). However during our previous studies on CA3 microcircuit, the pyramidal cells used LSW ranging from 0.6 to 0.7 [53, 54].

3. Results

Two simulations were used, one without LTP induction and one with LTP induction. For LTP induction pyramidal cells (P1, P2, P3, and P4) were strongly excited on inputs 7, 8, and 9 by stimulation at 100 Hz for 400 ms. Such approach was inspired by the well-known phenomenon where during the
environmental activity the firing rate at particular hippocampal connections increases rapidly [76]. Firing histograms of the 3 cells’ groups and theta oscillation including stimulation with and without LTP are given in Figures 2 and 3. Those stimulations refer to Bliss and Lomo research work [77, 78].

The induced LTP protocol has a specified time of duration. The solution could be the LTP related algorithm. This algorithm functions on a dendrite level, independently of each compartment in compatibility with canonical form of sp. The induced LTP protocol has a specified time of duration.

CA1 pyramidal cells LTP network (called Memory) is dependent on duration of simulation in both cases, connected with LTP induction and without it. In those simulations positive and statistically relevant correlation between time of simulation and average value Memory f of 4 pyramidal cells were received (Figure 5). Score without stimulation was R=0.97 (p=0.0001) and with LTP stimulation R=0.89 (p=0.0001). Furthermore, in both cases statistically valid increase of pyramidal cells memory and spikes value was observed (R=0.98, p=0.0001).

After arrival of action potential at a synapse the subsequent change of synaptic potential remains disposable for further computation. For EPSP this time of duration I pyramidal cells is 15 ms. The amount of interspike intervals combination relies on the number of received action potentials arriving at one synapse at the same time. Making an assumption that the difference of one clock step at one interval is essential for further computing, it could be estimated that there are 6272 combinations from one spike to dense impulse of 8 spikes. If we assume that 1 ms is a significant distinction then we achieve 623 combinations. A total amount of variation for 16 inputs (synapses of pyramidal cells) can be calculated as 627216.

Coworkers of Kasabov presented SPAN which allows recognizing over 200 synapses’ spike patterns during 200 ms stimulation [79]. SPAN is a spiking neuron capable of learning connections of arbitrary spike trains in a controlled fashion which enables process of spatiotemporal information encoding in the accurate timing of spikes.

4. Discussion

In biological neurons the precision might be less advanced due to the fact that diverse membranes current need time to raise potential. However, the mathematical models of

<table>
<thead>
<tr>
<th>CA1 cells</th>
<th>Inputs</th>
<th>1</th>
<th>2</th>
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<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
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<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyramidal cell (P1)</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>MF++</td>
<td>MF++</td>
<td>P2</td>
<td>P2</td>
<td>P2</td>
<td>P3</td>
<td>P3</td>
<td>P4</td>
<td>P4</td>
<td>P4</td>
<td>B1</td>
<td>B1</td>
<td>B2</td>
</tr>
<tr>
<td>Pyramidal cell (P2)</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>MF++</td>
<td>MF++</td>
<td>MF++</td>
<td>P3</td>
<td>P4</td>
<td>P4</td>
<td>P4</td>
<td>P3</td>
<td>P1</td>
<td>B1</td>
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<tr>
<td>Pyramidal cell (P3)</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>MF++</td>
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<td>MF++</td>
<td>P1</td>
<td>B1</td>
<td>B2</td>
<td>B2</td>
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<tr>
<td>Pyramidal cell (P4)</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>EC1+</td>
<td>MF++</td>
<td>MF++</td>
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<td>P1</td>
<td>B1</td>
<td>B2</td>
<td>B2</td>
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<tr>
<td>Basket cell (B1)</td>
<td>P2</td>
<td>P2</td>
<td>P2</td>
<td>P2</td>
<td>P2</td>
<td>P1</td>
<td>P1</td>
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<td>T1</td>
<td>T2</td>
<td>T3</td>
<td></td>
</tr>
<tr>
<td>Basket cell (B2)</td>
<td>SC</td>
<td>SC</td>
<td>SC</td>
<td>SC</td>
<td>SC</td>
<td>P3</td>
<td>P3</td>
<td>SC</td>
<td>SC</td>
<td>SC</td>
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<td>SC</td>
<td>SC</td>
<td>P4</td>
<td>T1</td>
<td>T2</td>
<td>T3</td>
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</table>

Table 3: Connections of hippocampal CA1 microcircuit.
Figure 2: Basic 10-second real-time simulation of pyramidal cells, basket cells, and OL-M cell without LTP inducing protocol. On the left, time course of summarized postsynaptic potential for all cells of CA1 region. On the right, output spikes train. Time (s), forecast time of duration LTP at the end of simulation.
Figure 3: Basic 10-second real-time simulation of pyramidal cells, basket cells, and OL-M cell with LTP inducing protocol. On the left, time course of summarized postsynaptic potential for all cells of CA1 region. On the right, output spikes train. Time (s), forecast time of duration LTP at the end of simulation.
Figure 4: Temporal relationship between theta oscillations, pyramidal cells, basket cells, and OL-M cell. On the left, temporal relationship without LTP inducing protocol. On the right, temporal relationship with LTP inducing protocol. Histograms show the firing probability rates of all cells CA1 region.

dendrites present the capacity to perform sub-millisecond compatibility detection.

The presence of chaos process inside brain network is acknowledged and there is no possibility for precise prediction of multiple inputs data and finding any analytical solution seems to be rather impossible. The Izhikevich model, similarly to many other models, consists of accessible differential equations with only a few parameters which appears to be easy to define [80]. Every constructed model of biological feasibility needs to be introduced with an efficient algorithm. Although there is a precise set of internal parameters for mature living neuron cells, some differences might appear in various brain areas. We have already determined the number of excitatory and inhibitory inputs with their exact location on dendrites. Then, we have estimated the parameters of postsynaptic potential, amount of time needed for threshold
and refraction, and finally two fundamental physiological values: resting potential and synaptic reversal potential. Thanks to those parameters, we have ability to present for model any algorithm weight change (learning) for the inputs (synapses). In order to inaugurate the simulation a signal for all excitatory impulses must be determined and in the Izhikevich equation it is named as “I.”

We performed more than a thousand simulations, using not only single pyramidal cells, but also a small network of ten neurons connected like in CA1 hippocampal area. Any

Figure 5: Comparison of memory during the simulation without and with LTP inducing protocol. On the left, relationship between memory function without and with LTP inducing protocol. On the right, histograms reflect number of firing spikes during 10s simulation without and with LTP inducing protocol.
changes appearing in initial values or input patterns were the reason for further alterations of the interspike intervals (ISI) time series on the output. The real time of course simulations was even 10 or 20 seconds. This is the fundamental proof that the CA1 model has chaotic, dynamic characteristics.

There are some notions for the stochastic resonance phenomenon, which were firstly observed in 1950 by Bernard Langenbeck [81]. Nowadays such diagnostic methods are commonly used; however the physical white noise signal extends the capacity of inner ear receptors to react.

Undoubtedly, certain emotions such as curiosity or fear strengthen the capability to learn and memorize new models and patterns. In this process pyramidal neurons located in the cerebral cortex or hippocampus get supplementary inputs from the excited emotion areas such as amygdaloid body, which might be perceived as an indirect supervised learning algorithm. The elementary mechanism for long-term potentiation induction requires presence of NMDA channels and removal of magnesium ions blockade to enable calcium ions influx through them [82, 83]. This is accomplished by depolarization of postsynaptic region; however instant depolarization is dependent on history of input patterns. This information may lead to a conclusion that any accessory input of all possible characteristics could potentially enlarge learning capability and should be considered as an unlike equivalent of the stochastic resonance phenomenon.

5. Conclusions

The most influential conclusion of all our studies seems to be the prospect of extracting pure information processing algorithm from biological backgrounds as channels and membranes. The fundamental concept of our pyramidal neuron model was derived precisely from the theory of transistors with floating gates capacitor coupling, computer language, and usual models of all biological details measured in hitherto models of neurons. We did not use any of Hodgkin-Huxley, integrate and fire, or spike timing dependent plasticity formalisms. The received outcome is a mathematical circuit which matches major apparent features of living nervous cells. Moreover, we are able to repeat the Bliss and Lomo trial for induction of long-term synaptic potentiation in the rabbit hippocampus carried out in 1973, which is presented in Figures 2 and 3 from our previous work [54].

The circuit model within shift registers working as memory buffer for any synapse is believed to have a great potential to future development of spatiotemporal computing. Such an accessible mathematical model can become a starting point for constructing biologically inspired processors which could be slightly implemented in hardware like Neuron-MOS Transistor of Shibata or Ohmi, which nowadays are arousing great interest [84, 85].

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The author declares that they have no conflicts of interest.

Supplementary Materials

CA1 network simulation: the video screen captures file hippocampal formation CA1 microcircuit from the simulation. On the top four pyramidal cells and on the bottom two basket cells and OL-M cell. 10-second real-time simulation of hippocampal cells with LTP inducing protocol. CA1 linear chart simulation: 10-second real-time simulation of pyramidal cells, basket cells, and OL-M cell with LTP inducing protocol showing linear chart. On the top four pyramidal cells and on the bottom two basket cells and OL-M cell (in the middle). Linear chart shows spikes and firing hippocampal cells formation of CA1 microcircuit. (Supplementary Materials)

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J. L. McClelland and N. H. Goddard, “Considerations arising


