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Working memory capacity: the role of parameters of spiking neural network model

N. S. Kovaleva, V. V. Matrosov, M. A. Mishchenko

National Research Lobachevsky State University of Nizhny Novgorod
23, Gagarin ave., Nizhny Novgorod 603022, Russia
E-mail: natazh29@yandex.ru, matrosov@rf.unn.ru, mischenko@neuro.nnov.ru
Correspondence should be addressed to Natalia S. Kovaleva, natazh29@yandex.ru
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The **purpose** of this work is to study a computational model of working memory formation based on spiking neural network with plastic connections and to study the capacity of working memory depending on the time scales of synaptic facilitation and depression and the background excitation of the network. **Methods.** The model imitates working memory formation within synaptic theory: memorized items are stored in form of short-term potentiated connections in selective population but not in form of persistent activity. Integrate-And-Fire neuron model in excitable mode are used as network elements. Connections between excitatory neurons demonstrates the effect of short-term plasticity. **Results.** It is shown that the working memory capacity increases as calcium recovery time parameter grow up or the capacity increases with neurotransmitter recovery time parameter becomes lower. Working memory capacity is found to decrease to zero with decrease of the background excitation as a result of lower values of both the mean and the variance of the external noise. **Conclusion.** Working memory capacity was studied as a function of time scales of synaptic facilitation and depression and background excitation of the network. Estimated working memory capacity is shown to be possibly larger than classical experimental estimations of four items. But capacity strongly depends on intrinsic parameters of neural networks.

Key words: working memory, memory capacity, spiking neural network, delayed activity, short-term synaptic plasticity.

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Introduction

Working memory (WM) is a system for short-term storage and manipulation of information [1–3]. WM is capable of handling information on time scales of several seconds up to minute and plays important role in mental reasoning, planning and, for instance, also calculating [4]. WM stands at the crossroads between memory, attention, and perception [1, 5].

A simple example of WM task is remembering a sequence of words spoken by your interlocutor in dialogue. Working memory is also important for performing and holding in mind a string of new information or a series of movements [6]. In visuospatial working memory, subject is holding for a short time, for example, locations, color or orientation of presented visual stimuli [7, 8]. Working memory experiments quantify the precision of memory recall. Typically, in such experiments, subjects are briefly presented with sensory inputs, which are then removed. After a delay the subjects are asked to estimate from memory some feature of the input.

Neural circuits of the prefrontal cortex (PFC) of the brain are assumed to be responsible for WM implementation [9, 10]. In primates, visual WM has been studied in delay tasks, which require a memory to be held during a brief delay period lasting for several seconds [11]. Recordings in the monkeys' PFCs during the delay task showed that some neurons displayed persistent and stimulus-specific delay-period activity [12–15]. Delay activity is considered the neural correlate

of WM [10, 16]. This implies that information in WM is represented by self-sustaining activity states. Delay activity in specific neural ensembles reflects keeping the memorized item «online».

The neural mechanisms of WM remain unclear and discussive. Electrophysiological recordings of neural activity during WM tasks demonstrate that some PFC neurons remain active during delay period. This "persistent activity" is hypothesized as neural correlate of memorized stimulus hold in WM [17–19]. This concept became classical and has its experimental proofs and mathematical models [20–23].

However, it is important to note that persistent spiking observed in experiments could be the result of classical approach of averaging spiking over time and across trials. In this case, persistent activity to be artifact of this averaging, even though, in real time, e.g., on single trials, recorded spiking of neuronal ensembles is sparse [24].

Other studies, however, find pieces of evidence for a different mechanism to store information in WM [24, 25]. Some researchers have hypothesized that the information in WM could be represented in form of complex sequences of different activity patterns, so called transient trajectories [26–29]. Brief, sparse, bursts of spiking were registered in WM tasks rather than persistent spiking. Information about memorized items are held between bursts by spiking-induced changes in synaptic weights, "impressions" left in the network [30, 31]. Wang, Goldman-Rakic, and colleagues showed that spiking in the PFC can produce fast synaptic enhancement that lasts hundreds of milliseconds [32] In fact, the enhancement depends on sparse, bursty spiking. Because the time spent by stimulus specific neural population in active state is kept to a minimum, the WM items are less prone to disruption from, e.g., a new sensory input applied to another neural population. Multiple items can be simultaneously held in WM in form of silent but enhanced neural population. Brief reactivations of populations multiplexed in time refreshes the synaptic enhancements and allows to held items in memory for a longer time [24].

Another interesting dynamical mechanism of WM formation is proposed in [33]. Gordleeva et. al. discovered that astrocytes operating at a time scale of a dozen of seconds can successfully store traces of neuronal activations corresponding to information patterns. In the retrieval stage, the astrocytic network selectively modulates synaptic connections in the spiking neural networks leading to successful recall.

It is not clear which dynamical mechanisms actually underlies the neuronal implementation of WM because both persistent activity and transient dynamics hypothesis have experimental validation [8]. This fact keeps the WM neural mechanisms as an open problem.

Working memory capacity is severely limited, as we can see from everyday experience, restricted to just a few items [34], and recall accuracy decay when a set of items to be memorized is too large [35]. The brain is often hypothesized to possesses a specialized buffer called «focus of attention», where memory items can be temporarily stored for short periods of time; therefore, WM capacity corresponds to the size of this buffer [36–38].

WM capacity in the brain is not easy to determine experimentally because multiple mechanisms retain information. The experiments should be carefully designed to prevent or control processing strategies (for example, silent rehearsal of items to be memorized or unite multiple items into single memory item by any mnemonic rule) [1, 36, 39]. Another way to determine WM capacity in psychological experiment is the running-span procedures, when the participant does not know when the presented set will end and has to recall as much items from the end of the list as possible [40]. The experimental estimation of WM capacity is still a problem to be solved [41].

In the mathematical modelling the WM capacity is also a widely discussed. The maximal capacity of persistent activity WM model depends on the characteristics of the network, but it does not seem to have the fundamental upper limit [22]. Lisman and Idiart [21] suggested that the WM capacity is estimated as a ratio of gamma and theta frequencies, which is compatible

with earlier psychophysical estimates [34]. In the framework of synaptic theory of WM proposed in [30] the WM capacity was analyzed in terms of basic synaptic parameters of the network [42].

In this paper we develop results of earlier studies in the framework of synaptic theory of WM [30] and present the results of a study of WM capacity in a spiking neural network as a function of synaptic plasticity and background excitation parameters. We have performed scrupulous computational analysis of spiking neural network dynamics and the effect of parameters on dynamic modes.

1. Materials and Methods

Mongillo et. al. in [30] proposed that memorized object is maintained in the WM by short-term enhancement of strength of connections between neurons that code for this item. Information about memorized items are held in WM by changes in synaptic weights, «impressions» left in the network after stimulus presentation. Because leak of residual calcium that facilitate synaptic transmission is a relatively slow process, the memory can be held for about one second without persistent spiking activity. While connections between neurons remains facilitated even weak stimulus or even noise could reactivate coding neurons and extract memorized item.

Most models of WM formation both persistent and transient use recurrent network architecture. In the synaptic theory of WM the recurrent neural network consists of excitatory and inhibitory neurons connected in a probabilistic way. Low probability of connections (about 20%) allows to form sparsely connected network. Some excitatory neurons are randomly grouped in subnetworks called clusters that are selective to specific stimulus (Figure 1). Connections between neurons in one cluster are stronger than connections between different clusters, mimicking the prior neural circuits formation [43] or dynamic long-term formation of network with adaptive connections [44]. The clusters mimic groups of neurons with, for example, similar receptive fields. Inhibitory neurons are forming a nonspecific pool connected to the excitatory clusters in a nonstructured way, resulting in dynamic competition between different selective populations (Figure 1).

The whole network dynamics is formed as a result of interplay between excitation and

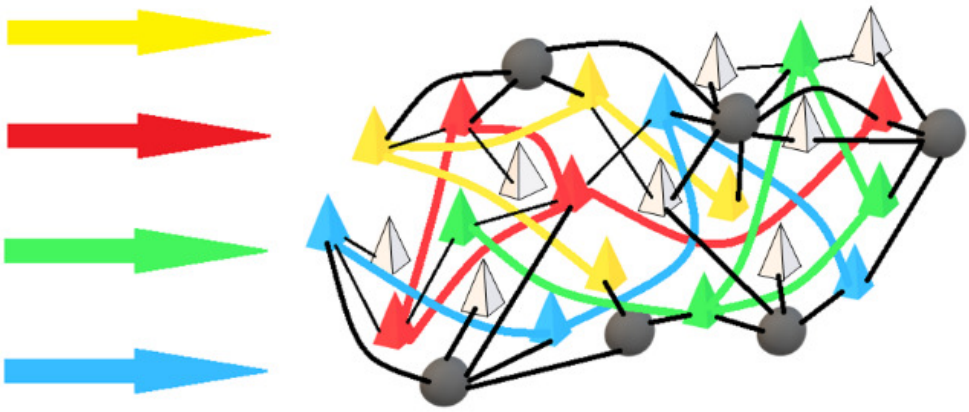

Рис. 1. Network architecture: Colored triangles are excitatory neurons that code for different memories. Light triangles are nonselective excitatory neurons. Black circles are inhibitory neurons with nonstructured connections to the entire network.

inhibition. When some neurons of the same cluster emit spikes nearly simultaneously (as a result of local stimulation or noise-induced spontaneous activity), they excite both other neurons of the clusters through strong couplings and some of inhibitory neurons, that inhibits activity of other neurons of the network. When the activity in the excited cluster decays the activity of inhibitory neurons decays too and «releases» other neurons from suppression.

The network consists of N_E excitatory and N_I inhibitory Integrate-and-Fire neurons. Their subthreshold dynamics is described by equation (1):

$$\tau_m \dot{V}_i = V_r - V_i + I_i^{(rec)}(t) + I_i^{(ext)}(t), \tag{1}$$

where $i = [1, N_E + N_I]$ – is a neuron number, τ_m — refers to membrane time constant, $I_i^{(ext)}$ – is an external current provided by distant brain areas. Membrane resistance has been absorbed into the definition of the currents. Every time, the membrane potential reaches a fixed threshold θ, neuron emits a spike and becomes refractory for a time τ_{arp}, after which resumes from subthreshold reset potential V_r [45]. Recurrent current $I_i^{(rec)}(t)$ is a sum of postsynaptic currents from all the other neurons connected to neuron i is described by equation (2):

$$I_i^{(rec)}(t) = \sum_j \widehat{J_{ij}}(t) \sum_k \delta(t - t_k^{(j)}), \tag{2}$$

where $\widehat{J_{ij}}(t)$ – is the instantaneous efficacy (time dependence is due to short-term synaptic dynamics) of the synapse connecting neuron j to neuron i; the sum on k is over all the emission times, $t_k^{(j)}$ of presynaptic neuron j. For simplicity, we neglect rise and decay times of the postsynaptic currents. In a single neuron case, the dynamics is totally defined by applied external current $I^{(ext)}$. If applied current is relatively weak, membrane potential V_i in model (1) does not reach the threshold θ and spike is not generated. As the external current increases, the membrane potential V_i reaches the threshold and a spike is generated. In this case, the generation frequency increases as the external current increases.

Synapses between excitatory neurons demonstrates the effect of short-term plasticity. There are two types of short-term plasticity: depression and facilitation. Synaptic depression is caused by the depletion of neurotransmitters, used for signal transmission on presynaptic neuron, while facilitation is caused by inflow of calcium ions into axonal terminal right after spike generation that increase the probability of neurotransmitter release. All excitatory-to-excitatory connections display facilitating transmission, described by a phenomenological model of short-term plasticity [46]. Short-term synaptic plasticity is described by equations (3)-(4):

$$\dot{u_j}(t) = \frac{U - u_j(t)}{\tau_F} + U[1 - u_j(t)] \sum_k \delta(t - t_k^{(j)}), \tag{3}$$

$$\dot{x_j}(t) = \frac{1 - x_j(t)}{\tau_D} + u_j(t) x_j(t) \sum_k \delta(t - t_k^{(j)}), \tag{4}$$

where u – is a fraction of available neurotransmitter to be released during the synaptic transmission, x – is an available neurotransmitter resource, τ_F and τ_D – are temporal parameters of short-term synaptic plasticity. For facilitating synapses $\tau_F > \tau_D$, and vice-versa $\tau_F < \tau_D$ for depressing synapses. In PFC synapses demonstrates facilitation and temporal scale of τ_F is up to several seconds and for τ_D is about several hundreds of milliseconds [42].

Instant synaptic efficacy $\widehat{J_{ij}}(t)$ in equation (2), also known as synaptic weight, is described by equation (5):

$$\widehat{J_{ij}}(t) = J_{ij} u_j(t) x_j(t), \tag{5}$$

Parameters of neurons	**Excitatory**	**Inhibitory**
θ – spike threshold	20 mV	20 mV
V_r – reset potential	16 mV	13 mV
τ – membrane time constant	15 ms	10 ms
τ_{arp} – absolute refractory period	2 ms	2 ms
N – number of neurons	800	200
Parameters of short-term synaptic dynamics		
U – baseline utilization factor	0.1	
J_{IE} – synaptic efficacy $I \to E$	-0.6 mV	
J_{EI} – synaptic efficacy $E \to I$	0.2 mV	
J_{II} – synaptic efficacy $I \to I$	-0.6 mV	
J_b – baseline level of $E \to E$ synapses	0.02 mV	
J_p – potentiated level of $E \to E$ synapses	2.7 mV	

Таблица 1. Model parameters.

where J_{ij} – is absolute synaptic efficacy of connection between neurons j and i.

All the other synapses between inhibitory and excitatory and inhibitory neurons demonstrates linear synaptic transmission i.e. $\widehat{J_{ij}}(t) = J_{ij}$.

External currents are modelled as Gaussian white noise (6):

$$I_i^{(ext)}(t) = \mu_{ext} + \sigma_{ext}\eta_i(t) \tag{6}$$

with $< \eta_i(t) >= 0$, $< \eta_i(t)\eta_j(t') >= \delta_{ij}\delta(t - t')$, so that μ_{ext} and σ^2_{ext} are respectively the mean and the variance of the external currents.

Network dynamics are fully described by equations (1) – (6). Numerical simulations have been conducted using Euler-Maruyama scheme. The biologically relevant values of the neuron parameters are the same as in the paper [42]. Model parameters are presented in Table 1.

We have also observed the network with overlapping populations. In such network some neurons from one selective population have strong (on potentiated level) connections with another selective population.

We have modeled neural network of 1000 neurons. Network contains 8 selective populations of 70 excitatory neurons (neurons 1-560), 200 neurons are inhibitory (neurons 801-1000) and the rest 240 neurons are excitatory non-selective. Probability of connection between any two neurons is 20%.

After 5 seconds of spontaneous activity from start of simulation every selective population (or cluster) is consequently stimulated by external current of 30 mV for 0.3 seconds. This external stimulation represents a process of loading 8 items into memory. The dynamics of the network is shown on raster plot (Figure 2), where every dot represents a spike.

The population activity increases for the duration of the selective input, changing the internal state of the synaptic connections. The connections demonstrate both depression (reduced resource x) and facilitation (increased sensitivity u), with depression dominant on the shorter time scale of τ_D and facilitation dominant on longer time scale of τ_F. As long as the intracluster connections remain facilitated, the object can be recalled from WM by reactivation of cluster activity in response on presentation of a weak nonspecific excitatory input to the whole network or by the internal noise activity, even though the neural activity is at the spontaneous level [30]. Reactivation of cluster activity is expressed as a population spike (PS), where almost every neuron in the population fires a spike within a short interval of time.

In Figure 2, seven of eight populations demonstrate spontaneous reactivations that corresponds

to seven items maintained in network memory. Every population spike refreshes the memorized item by increasing the facilitation u of synapses in the population.

The number of items that could be simultaneously stored in working memory refers its capacity. In the studied model, the working memory capacity is determined as a number of clusters that emit population spikes in 5 seconds after external stimulation stopping and after 1 second for every cluster stimulus to avoid transient processes. Moreover, the neural network should not have spontaneous population spikes before external stimulation. For instance, network simulated in Figure 2 has capacity of seven items. Results of 10 simulations for a fixed set of parameters have been averaged to estimate the working memory capacity.

2. Results

Let us show the role of the network parameters in working memory formation and its capacity. The model (1)-(6) has a huge number of parameters: synaptic weights, time scales of synaptic facilitation and depression, mean and the variance of the external currents, network and cluster sizes etc. All these parameters play significant role in network dynamics and respectively, in working memory formation. For example, the balance of network excitation and inhibition is crucial for population spikes formation and concurrency between different clusters. We have studied the role of temporal parameters of short-term synaptic plasticity τ_F and τ_D and the total network activity defined by mean and the variance of nonspecific external currents applied to every neuron in network.

2.1. Role of synaptic parameters. We have studied the role of synaptic parameters on working memory capacity in spiking neural network model. Capacity of the network's memory was estimated for different temporal parameters of short-term synaptic plasticity τ_F and τ_D and

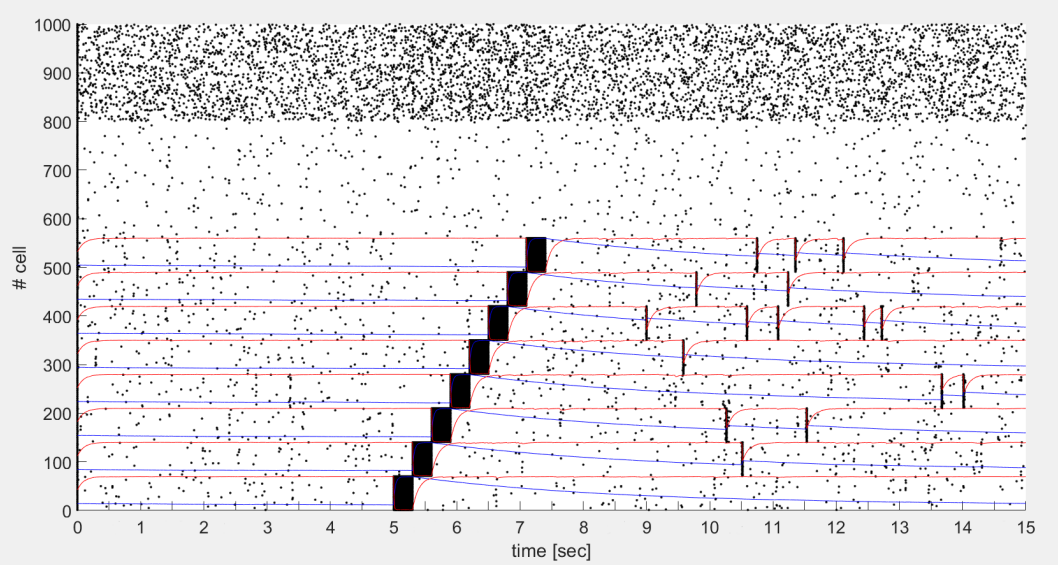

Рис. 2. An example of network simulation, demonstrating a pre-stimulus spontaneous activity and consequent loading eight items into WM. Each dot represents a spike of one of 1,000 neurons arranged in order such that the first 560 neurons are encoding eight items stored in the network. For each cluster, graphs of changes in the average values of synaptic efficiency u in clusters are shown in blue, and changes in the average values of synaptic resource x in clusters are shown in red. Parameters are as follows: $J_p = 2.7$, $\mu_{ext} = 10$, $\sigma^2_{ext} = 0.12$, $\tau_F = 3.6$, $\tau_D = 0.1$.

synaptic weight of connections between excitatory neurons belonging to same cluster. It should be noted, that the existence of population spikes and concurrency between different clusters are possible only with a balance of synaptic weight parameters and external excitation. Sufficient deviation from the balance of the parameters can lead to a lack of population spikes, appearance of global asynchronous activity in the network or the appearance of population spikes prior to memory load by items presentation. Working memory capacity as a function of τ_F and τ_D obtained with numerical simulations of equations (1) – (6) are presented in Figure 3 for two values of potentiated intracluster synaptic weights $J_p = 2.3$ (Figure 3a) and $J_p = 2.7$ (Figure 3b).

We found, that working memory capacity increases as synaptic facilitation timescale τ_F grow up. Synaptic depression timescale τ_D shows opposite role in our simulations: for a fixed τ_F the capacity increases with τ_D becomes lower. These results partly contradict to the estimations obtained by Mi et. al., as they noted that "surprisingly, even though the WM trace in the model is maintained by synaptic facilitation, the derived expression shows that WM capacity is chiefly increasing with the time constant of synaptic depression and only weakly increasing with the time constant of facilitation" [42]. This contradiction looks surprising, but the decrease of time constant of synaptic depression allows cluster to generate next population spike earlier that refreshes the memory trace of this cluster. Population spikes rate grows up and memory traces remain on higher levels increasing the capacity.

The potentiated intracluster synaptic weights J_p play scaling role for working memory capacity as a function of τ_F and τ_D: as J_p decrease from 2.7 (Figure 3b) to 2.3 (Figure 3a) the capacity diagram scales down but overall dependence on τ_F and τ_D remains the same.

2.2. Role of background excitation. We have studied the role of background excitation parameters on WM capacity. Background excitation is nonspecific input applied to every neuron in the network modeled as Gaussian white noise with mean μ_{ext} and variance σ^2_{ext}. WM capacity is estimated for different combinations of mean and variance of white noise (Figure 4).

Working memory capacity is found to decrease to zero with decrease of global network activity as a result of lower values of both the mean μ_{ext} and the variance σ^2_{ext} of the noise. The variance seems to play more significant role for the capacity. The level of background excitation enables the WM to be efficiently «tuned» to the desired capacity; in particular, reducing the background below the critical value make neural network unable to produce population spikes, hence, removes memorized items from WM to make room for new inputs [42].

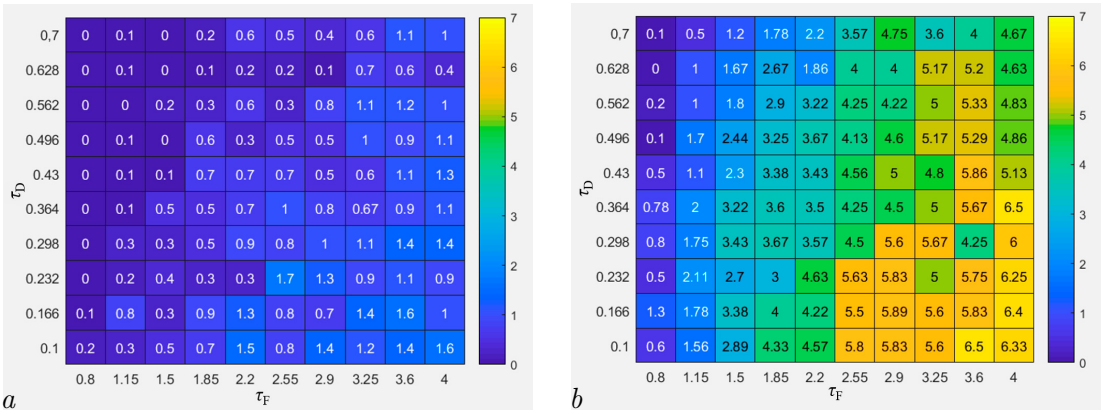

Рис. 3. Working memory capacity as a function of τ_F and τ_D obtained with numerical simulations of equations (1) –(6) for two values of potentiated intracluster synaptic weights $J_p = 2.3$ (a) and $J_p = 2.7$ (b). $\mu_{ext} = 10$, $\sigma^2_{ext} = 0.12$.

2.3. Working memory in a network with overlapping clusters. Another unrealistic feature of the model concerns the absence of overlaps between representations of different memory items. The model was modified to make some excitatory neurons have potentiated connections with neurons of several clusters i.e., memory representations are overlapping. Network dynamics in a case of two items memorized in the network is shown in Figure 5.

The network in Figure 5 contains two clusters of excitatory neurons that have 4 neurons belonging to both clusters (1,4% of cluster size). For each cluster averaged values of short-term synaptic plasticity variables u and x are shown in blue and red respectively. After 5 seconds of spontaneous activity from start of simulation every cluster is consequently stimulated by external current of 30 mV for 0.3 seconds. After loading two items into working memory, they are represented as a temporal facilitation of connections in clusters and population spikes. Despite the presence of overlapping in clusters, the distinction between clusters is preserved, and population spikes of one cluster does not activate the neurons of another cluster through overlapping connections.

We have added 5,5% overlapping of clusters into model with eight items loaded into

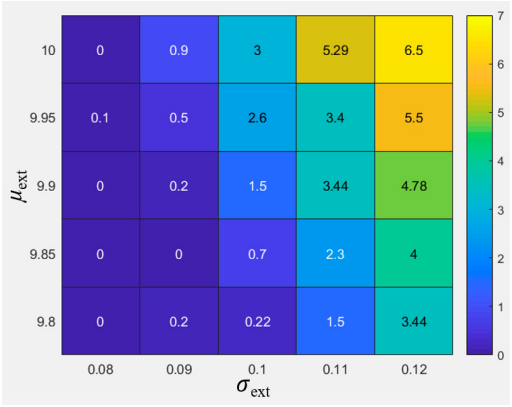

Рис. 4. . Working memory capacity as a function of background excitation parameters μ_{ext} and σ^2_{ext} for $\tau_F = 3.6$, $\tau_D = 0.1$.

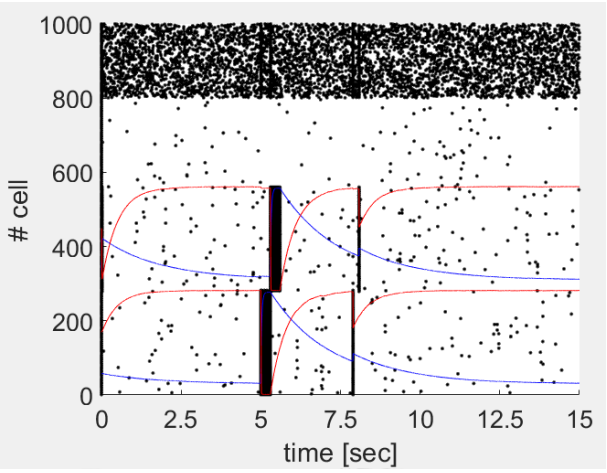

Рис. 5. An example of network with overlapping clusters simulation, including spontaneous activity and WM triggered by loading two stimuli. Spikes of 600 neurons are shown as dots; neurons are arranged in order such that the first 560 neurons are encoding two items stored in the network. Grey shaded area marks the 4 neurons belonging to both clusters. Parameters are as follows: $J_p = 2.3$, $\mu_{ext} = 9.63$, $\sigma^2_{ext} = 0.12$, $\tau_F = 3$, $\tau_D = 0.6$.

working memory and studied its capacity. Estimated capacity as a function of τ_F and τ_D is obtained for overlapping clusters with $J_p = 2.3$ (Figure 6).

Working memory capacity for overlapping clusters is very similar to estimations obtained without overlapping (Figure 3). Thus, the observed model is robust and does not require rigid delimitation of clusters. Therefore, all the results for non-overlapping memory representations could be adapted to overlapping ones.

3. Discussion

We studied a computational model of working memory formation based on spiking neural network. The model imitates working memory formation within synaptic theory: memorized items are stored in form of short-term potentiated connections in selective population but not in form of persistent activity. Short-term potentiation of connections modelled as short-term synaptic plasticity. The recurrent neural network consists of excitatory and inhibitory neurons connected in a probabilistic way. Some excitatory neurons are randomly connected in clusters selective to specific stimulus. Connections between the neurons in cluster are stronger than connections between different clusters, mimicking the prior neural circuits formation or dynamic long-term formation of network with adaptive connections [44]. Inhibitory neurons are forming a nonspecific pool connected to the excitatory clusters in a nonstructured way, resulting in dynamic competition between different selective populations. As long as the intracluster connections remain facilitated, the object can be recalled from WM by reactivation of cluster activity in response on presentation of a weak nonspecific excitatory input to the whole network or by the internal noise activity, even though the neural activity is at the spontaneous level. Reactivation of cluster activity is expressed as a population spike (PS), where almost every neuron in the population fires a spike within a short interval of time.

The number of items that could be simultaneously stored in working memory refers its capacity. In the studied model, the working memory capacity is determined as a number of clusters that emit population spikes after external stimulation stopping. These population activity is the same activity recorded in electrophysiological experiments in PFC that hypothesized to be neural basis of WM [20, 25].

We have studied the working memory capacity as a function of time scales of synaptic

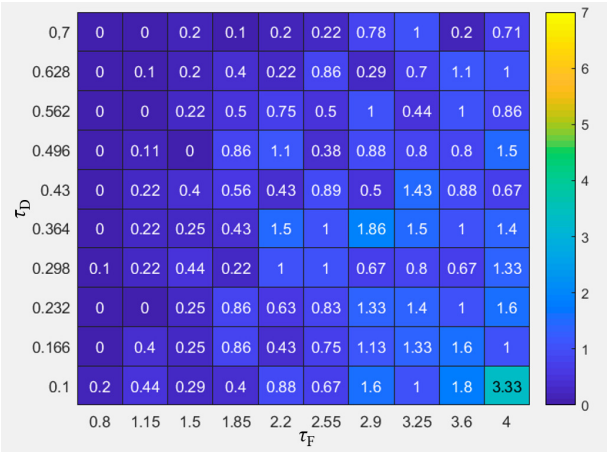

Рис. 6. Working memory capacity in a network with overlapping clusters as a function of τ_F and τ_D obtained with numerical simulations of equations (1)–(6) for $J_p = 2.3$ and $\mu_{ext} = 10$ and $\sigma^2_{ext} = 0.12$.

facilitation and depression and background excitation of the network. These parameters are the basic parameters of cortical networks.

We found, that working memory capacity increases as synaptic facilitation timescale τ_F grow up. Synaptic depression timescale τ_D shows opposite role in our simulations: for a fixed τ_F the capacity increases with τ_D becomes lower. This result partly contradicts conclusions of Mi et. al. [42] as "WM capacity is chiefly increasing with the time constant of synaptic depression" but "increasing the time constant of synaptic depression above a certain value brings the network to the regime where no PSs are possible and, hence, WM breaks down". Our results show that there is no such a controversy in the role of synaptic depression time scale.

We have also showed that WM capacity decreases to zero with decrease of global network activity as a result of lower values of both the mean μ_{ext} and the varianceσ^2_{ext} of the background noisy excitation. The variance seems to play more significant role for the capacity. This is interesting, because demonstrates possible significant role of nonidentities and non-idealities in neural network dynamics and should be taken into account in mathematical modelling of neural networks. The dependence of WM capacity on the background excitation enables the system to be efficiently «tuned» to the desired capacity.

Estimated WM capacity is shown to be possibly larger than classical experimental estimations of four items [39,47]. But capacity strongly depends on parameters of neural networks that can't be significantly improved by simple training. Such strong dependence of the capacity on intrinsic parameters of neural network could be the reason of individual differences in experimental studies. These parameters could also be one of the reasons of WM dysfunction in schizophrenia [48,49]. All the results on WM capacity dependence on neural network parameters could be useful in clinical research of memory impairments associated with neurological disorders.

Conclusions

This paper presents a detailed study of the role of time scales of synaptic facilitation and depression and background excitation of the neural network on working memory capacity. Working memory capacity strongly depends on many parameters and is shown to be possibly larger than classical experimental estimations. Our results demonstrate possibility of synaptic theory of working memory to imitate various experimental estimations of WM capacity by individual differences in synaptic plasticity parameters. The model could be improved to imitate different experimental protocols of memory load.

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Мищенко Михаил Андреевич – родился в городе Горьком (1987). Окончил радиофизический факультет Нижегородского государственного университета им. Н.И. Лобачевского (2010). Кандидат физико-математических наук (2013). Старший научный сотрудник и доцент кафедры теории колебаний и автоматического регулирования радиофизического факультета Национального исследовательского Нижегородского государственного университета им. Н.И. Лобачевского. Область научных интересов -– нейродинамика, нелинейная динамика и синхронизация. Опубликовал свыше 20 научных статей по указанным направлениям.

Россия, 603950 Нижний Новгород, пр. Гагарина, 23
Нижегородский государственный университет им. Н.И. Лобачевского
E-mail: mischenko@neuro.nnov.ru
ORCID: 0000-0001-8801-7664