What the training of a neuronal network optimizes

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In the study a model of training of neuronal networks built of integrate-and-fire neurons is investigated. Neurons are assembled into complex networks of Watts-Strogatz type. Every neuronal network contains a single receptor neuron. The receptor neuron, stimulated by an external signal, evokes spikes in equal time intervals. The spikes generated by the receptor neuron induce subsequent activity of a whole network. The depolarization signals, traveling the network, modify synaptic couplings according to a kick-and-delay rule, whose process is termed "training." It is shown that the training decreases the mean length of paths along which a depolarization signal is transmitted from the receptor neuron. Consequently, the training also decreases the reaction time and the energy expense necessary for the network to react to the external stimulus. It is shown that the initial distribution of synaptic couplings crucially determines the performance of trained networks.

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INTRODUCTION

An important class of phenomena, related to applications of practical relevance, involves the transmission of some signal through excitatory networks. In an excitatory network a node (bond) is open for transport, provided that the amplitude of a signal applied to it exceeds a threshold value and is closed otherwise. Problems of a similar kind arise in networks of diodes [1], flow of Bingham plastic [2], or foam [3] in porous media and dielectric breakdown [4]. An important area of application of excitatory networks is also modeling neuronal networks, in which case additional degrees of freedom are assigned to the network nodes [5]. The latter field of application of excitatory networks has provided especially important results concerning synchronization of neurons, the origin of memory, and pathologies such as epileptic seizures or Parkinson disease, models of learning and recognition, etc. [6].

One of the most exciting but not fully understood properties of neuronal networks is their ability to adjust their response to varied stimuli. Generally, signals traveling through a neuronal network can modify the couplings between neurons—the conductance of a synapse changes depending on the history of the depolarization of the presynaptic and postsynaptic neurons [7]. This process, termed synaptic plasticity, is believed to be responsible, for example, for the animals ability to learn and recognize spatial or temporal external signals [8]. While it has been shown [9] that under some assumptions about the network topology spatiotemporal information can indeed be coded in the activity patterns of the neurons, it is an open question what changes in the neural network properties, induced by synaptic plasticity, facilitate learning and recognition in a general case.

Depolarization of a neuron can eventually induce the subsequent depolarization of neighboring neurons. Consequently, a neuronal network acts as a depolarization transferring network. The ability to transfer the depolarization signal is the most basic property of the neuronal networks, which supports the above mentioned high-level tasks of learning and recognition.

In the present paper I study the geometry of the paths along which depolarization is transferred through neuronal networks. The geometric characteristics (e.g., numerous fractal exponents [10]) of signal transferring paths are often used to distinguish between different transport models, even if the models are defined on networks with fixed topology. I show that the primary effect of the synaptic plasticity is the change of the geometry of the depolarization paths. During continued stimulation a spatial pattern of high-conductivity connections between neurons develops, which supports the transmission of the depolarization signal. Importantly, the performance of a neuronal network after a period of stimulation strongly depends on the initial distribution of the synaptic couplings.

MODEL

In the model of an integrate-and-fire neuron [11], used in the present study, the change $dV_i(t)$ of the potential $V_i(t) < V_i^{\text{th}}$ of the ith neuron during the time dt is given by

$$dV_i(t) = -dt[V_i(t) - V_i^{\text{base}}]/\gamma_i + \sum_{j \in N_i} g_{ij}\delta(t - t_{j,S} - t_j^D), \quad (1)$$

where γ_i is a constant and V_i^{base} is the resting potential. The last term of the right-hand side of Eq. (1) represents the sum of excitatory postsynaptic potentials (EPSPs). EPSP is the change of $V_i(t)$ induced by a spike generated by the jth neighbor contained within the neighborhood N_i of the ith neuron. If the coupling g_{ij} is nonzero then the neuron i receives a spike at time t after the neuron j has fired at time $t_{j,S}=t-t_j^D$, where t_j^D is a delay time. If $V_i(t)$ crosses the threshold potential V_i^{th} (if no noise is present it is possible only provided that at least one of the neighbors of the ith neuron has fired) a spike is generated by the ith neuron and $V_i(t)$is reset to $V_i^{\text{fire}} \leq V_i^{\text{base}}$. A neuron i, which has fired is by definition active for time t_i^D (for the time necessary to transmit depolarization signal to the neighboring neurons). After generating a spike a neuron enters a refractory period for a

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time t_i^R. I have assumed that during the refractory period a neuron neither integrates EPSPs induced by spikes arriving to its synapses nor generates spikes.

The coupling constants g_{ij} alter in response to spikes arriving to the synapses, whose process is termed synaptic plasticity [7]. Synaptic plasticity occurs across many time scales, from milliseconds for short-time enhancement to days or longer for long-term potentiation, believed to be responsible for the ability to learn. In the latter case depolarization of both presynaptic and postsynaptic neurons is necessary to alter the synaptic coupling, while for the short-time enhancement to occur it is sufficient that a presynaptic neuron depolarizes [12]. In the present study I consider long-term synaptic potentiation only. A simplest model, which qualitatively reproduces long-term potentiation-related alternations of the synaptic couplings is a kick and delay model as follows:

$$dg_{ij} = -dt\alpha(g_{ij} - g_B) + \Theta(g_T - g_{ij})K\delta(t - t_S), \qquad (2)$$

where α is a decay constant and g_B is a base value of g_{ij}. If a spike evoked by a presynaptic neuron at time t_S has led to the depolarization of the postsynaptic neuron and if g_{ij} is less than the maximal allowed value g_T of g_{ij} [this condition is represented by the Heaviside function $\Theta(g_T - g_{ij})$] then g_{ij} is increased by K. For ease of notation I use the term "training" throughout the paper to denote stimulation of the network by spikes generated by the receptor neuron S, followed by appropriate modifications of the synaptic couplings g_{ij}. That process is not training in a strict sense, because the network is not taught to recognize any spatial or temporal patterns. I have tested the influence of varied initial distribution of the synaptic couplings on the training process: all $g_{ij} < V_{\text{th}} - V_{\text{base}}$; all $g_{ij} > V_{\text{th}} - V_{\text{base}}$; some fraction of $g_{ij} > V_{\text{th}} - V_{\text{base}}$ and the other $g_{ij} < V_{\text{th}} - V_{\text{base}}$.

The neurons are assembled and form a complex network of Watts-Strogatz type [13]. The Watts-Strogatz network has been selected as a model of the topology of a neuronal network, because it is an example of a complex network type, characterized by small-world property, observed also for networks of real neurons [14]. I have assumed that every neuron is connected with two of its nearest neighbors. Then I have created additional connections between every neuron and randomly sampled number of distinct neurons. In contrast to the Watts-Strogatz model, nonlocal connections are not the result of a rewiring procedure. I have further assumed that the neuronal network contains a single "receptor" neuron S, which sends spikes in equal time intervals in the reaction to some external stimulus. Because neurons act according to the rule "all-or-nothing," the strength of the external stimulus can be coded in the frequency of firing of the receptor neuron (if the number of receptor neurons is larger than 1, then the strength of the external stimulus can be also coded in the number of firing receptor neurons). Here I am not concerning the influence of the strength of the stimulus on the activity of the network and thus the firing frequency of the receptor neuron is fixed and equal to one spike per unit of time. Under some circumstances (e.g., low network connectivity or sufficiently small couplings g_{ij}) it is possible that the depolarization signal will not be received by some neurons. Here I chose the network parameters such that transmission failures do not occur. It is a problem of recent interest how a neuronal networks characteristics influence its ability to transfer a depolarization signal [15]. I have assumed that the network has reacted to the stimulus if all neurons have received the depolarization signal. Consequently, the time necessary to propagate the depolarization signal through the entire network depends on the number of neurons and other network characteristics. For a network, which has generated a reaction to the stimulus, I have measured the average time t_A necessary to generate the reaction. The initial spike, which induces the activity of the network, is sent by S at $t=0$. The initial conditions are $V_i(t=0) = V_i^{\text{base}}$ for all i. Additionally, I have also measured the mean length L of signal transferring paths, connecting S to every other neuron of the network. The length of the signal transferring paths is defined in an iterative manner as follows. A neuron, which has fired after receiving a spike from, S, is at the unit length from S. A neuron, which has not yet fired and fires after receiving a spike from a neuron being at a length n from S, is at the length $n+1$ from S.

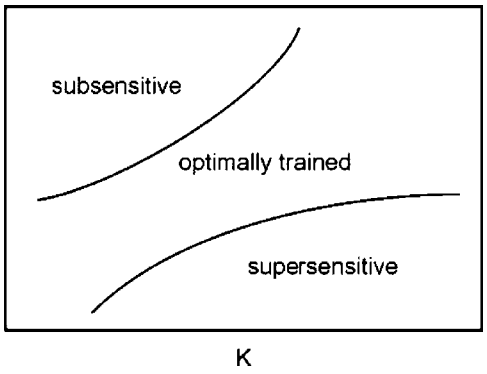

FIG. 1. Final state of a trained neural network depends on the values of the parameters α and K in Eq. (2).

The average values of L (denoted $\langle L \rangle$) and t_A (denoted $\langle t_A \rangle$) were calculated for 1000 realizations of neuronal networks and were determined as the functions of the number N of the neurons.

In the simulations I have assumed that $V_i^{\text{base}} = V_{\text{base}}$, $V_i^{\text{fire}} = V_{\text{fire}}$, $V_i^{\text{th}} = V_{\text{th}}$, $\gamma_i = \gamma$, $t_i^D = t_D$ and $t_i^R = t_R$ for all i. Typical values of these constants, used during simulations were $V_{\text{base}} = 0.8$, $V_{\text{fire}} \in \langle 0.5, 0.8 \rangle$, $V_{\text{th}} = 1.0$, $\gamma = 20.0$, $t_D = 0.05$, and $t_R \in \langle 0.1, 0.5 \rangle$. Also the basal properties of the synapses were fixed: $g_B = 0.1$ and $g_T = 0.3$. The topology of the network was fixed to be Watts-Strogatz-type with the mean number of receiving synapses in a range from 4 to 20.

RESULTS

The training process modifies the distribution of the synaptic couplings g_{ij}. The training parameters K and α, used in Eq. (2), determine the type of the distribution of g_{ij} values for the trained networks. Depending on these values (Fig. 1) g_{ij}'s are distributed either only around g_T (supersensitive networks), only around g_B (subsensitive networks), or around g_T and g_B (binomial distribution for optimally trained networks).

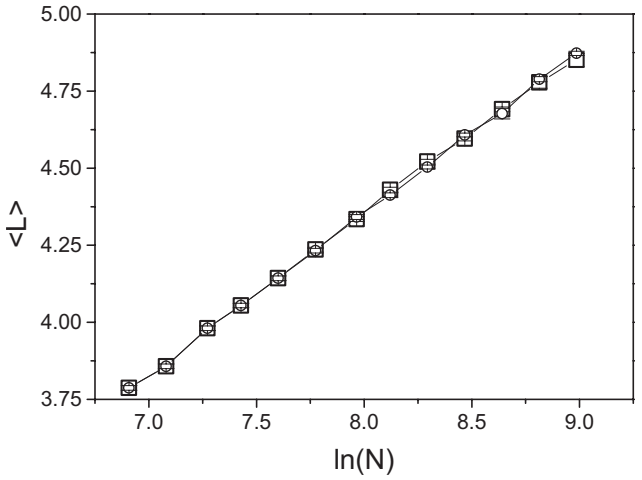

FIG. 2. The mean length $\langle L\rangle$ of depolarization paths plotted vs the logarithm of the number N of neurons for initial values of synaptic couplings $g_{ij}(t=0) > V_{\text{th}} - V_{\text{base}}$ (squares) and optimally trained networks starting from these initial values (circles). The statistical error bars are also shown in the figure. The results obtained for $t=0$ are equivalent to the results for supersensitive networks because in both cases the distribution of g_{ij}'s is the same.

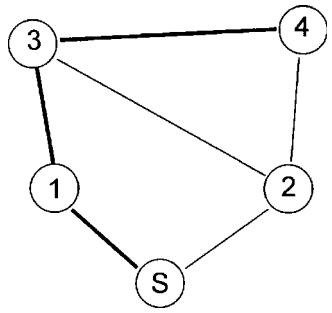

FIG. 3. In a subsensitive network depolarization paths must be different from the shortest paths. In a subsensitive network a single spike sent by a receptor neuron S is not sufficient to depolarize neurons 1 and 2. These neurons can eventually generate spikes after receiving a second spike from S. Spikes sent from neurons 1 and 2 to neuron 3 depolarize neuron 3, but a single spike sent from neurons 2 to 4 is not sufficient to depolarize neuron 4. However, depolarization of neuron 3 integrated in neuron 4 with earlier excitation from neuron 2 can depolarize neuron 4. The depolarization path from S to neuron 4 is drawn with a bold line. The length of this path is equal to 3 in contrast to the shortest path from S to neuron 4, whose length is equal only to 2.

If the value of g_T is selected such that $V_{\text{base}} + g_T > V_{\text{th}}$ (e.g., $g_T = 0.3$, used in the simulations) the geometry of the depolarization paths of supersensitive networks is the same as the geometry of the shortest paths in the small-world networks, for which $\langle L\rangle \sim \ln N$ (Fig. 2). In that case a single spike generated by any neuron is sufficient to excite all the neighbors of this neuron and thus the depolarization paths must be equivalent to the shortest paths. Mean reaction time $\langle t_A\rangle$ is a linear function of $\langle L\rangle$ $[\langle t_A\rangle = \langle L\rangle t_D]$.

In the case of subsensitive networks all the values of g_{ij} decay to g_B. If $V_{\text{base}} + g_B < V_{\text{th}}$, every neuron of a subsensitive network must necessarily receive at least two spikes to generate its own spike and because of that the depolarization paths are different from the shortest paths (Fig. 3). In fact, for the present implementation of *IF* neurons, the dependence of $\langle L\rangle$ on N is nonmonotonic and nonuniversal in this regime (Fig. 4). To explain the reasons of this phenomena I have examined the distribution of the depolarization times and the distances from the receptor neuron. Nonmonotonic behavior of $\langle L\rangle$ occurs whenever abrupt changes of the mean depolarization time are observed. For example, in my simulations most of the networks built of 2000 neurons reacted to the stimulus after time $t \approx 3.0$ (i.e., after the receptor neuron has evoked three spikes). Most of the networks built of 4000 reacted after time $t \approx 7.0$. Finally, for networks with 2880 neurons the highest dispersion of the depolarization times was observed: a similar number of networks reacted after $t \approx 7.0$ and $t \approx 3.0$, which in turn induced higher dispersion of the distances and higher $\langle L\rangle$ than for networks with $N = 2000$ and $N = 4000$. For subsensitive networks mean reaction time $\langle t_A\rangle$ is an increasing but nonlinear function of N and there is no functional dependence of $\langle t_A\rangle$ on $\langle L\rangle$.

For intermediate values of α and K bimodal distribution of g_{ij} develops during the training process. In my experiments most of the synaptic couplings decayed to g_B, while typically not more than 30% of g_{ij} were distributed around g_T. I have measured the probability $P(k)$ that a neuron has k incoming synapses for which $g_{ij} > V_{\text{th}} - V_{\text{base}}$. In all cases $P(k)$ has had a single maximum for $k=1$ and decayed for larger k. The probability $P(k=0)$ was always almost equal to 0 for trained networks (typically within 2% tolerance), even if initially $P(k=0)=1$, that is, high conductivity synaptic connections were developed for every neuron during the training process. I have also found that after the training depolarization signal propagated only through synapses for

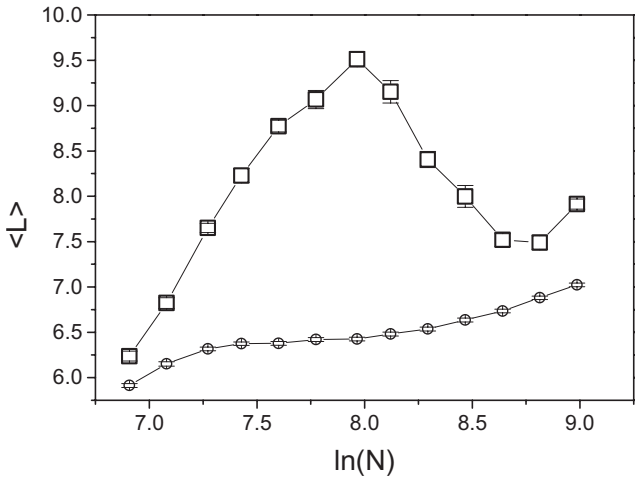

FIG. 4. The mean length $\langle L\rangle$ of depolarization paths plotted vs the logarithm of the number N of neurons for initial values of synaptic couplings $g_{ij}(t=0) < V_{\text{th}} - V_{\text{base}}$ (squares) and optimally trained networks starting from these initial values (circles). The results obtained for $t=0$ are equivalent to the results for subsensitive networks because in both cases the distribution of g_{ij}'s is the same.

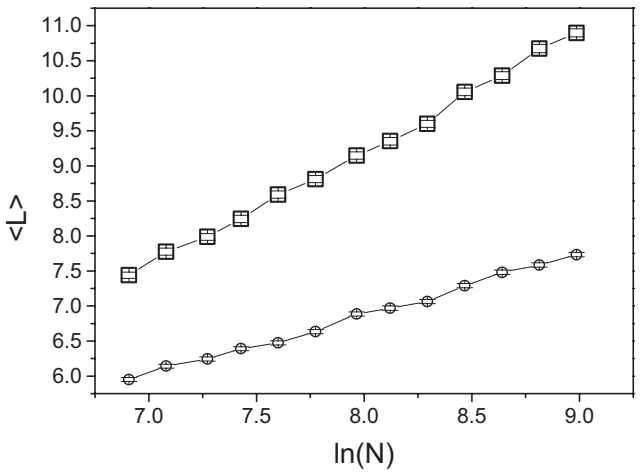

FIG. 5. The mean length $\langle L\rangle$ of depolarization paths plotted vs the logarithm of the number N of neurons for networks with 30% $g_{ij}(t=0)>V_{\mathrm{th}}-V_{\mathrm{base}}$ before (squares) and after (circles) training.

which $g_{ij}>V_{\mathrm{th}}-V_{\mathrm{base}}$. Consequently, only some fraction (for example, 30% for $K=0.01$ and $\alpha=0.01$, $V_{\mathrm{base}}=V_{\mathrm{fire}}=0.8$, $t_R=0.4$) of the initial synaptic connections participated in the propagation of the depolarization signal, while other connections were depreciated. In contrast, a depolarization signal in nontrained networks can propagate through any synapses, independently on the synaptic couplings.

I have found that the geometry of depolarization paths strongly depends on the initial distribution of g_{ij}'s. The values of $\langle L\rangle$ calculated for trained networks are plotted vs the number of neurons N in Figs. 2, 4, and 5. After the training one has always $\langle t_A\rangle=\langle L\rangle t_D$, which is another effect of the existence of high conductivity synapses ($g_{ij}>V_{\mathrm{th}}-V_{\mathrm{base}}$) for every neuron. The training process makes the depolarization paths more compact and decreases the reaction time.

Finally, I have checked how the properties of the depolarization paths change if the initial spike in a trained network is sent from a randomly selected neuron instead of the receptor neuron S. The results presented in Table I indicate that the mean length of the depolarization paths and the mean reaction time are minimal if the spikes initializing the activity of the network are sent from the receptor neuron S: the geometry of the depolarization paths is optimized for the transmission broadcast from S.

DISCUSSION

Typically the stimulation of a neuronal network causes some form of reaction. If it is assumed that the neuronal network contains "reaction" neurons (e.g., motoneurons) initiating the reaction aimed at terminating the external stimulus, then the short reaction time and short depolarization paths are advantageous. Moreover, such "reaction" neurons by possible action of inhibitory synapses may eventually suppress activity of the network after the external stimulation has been terminated. In such a case the changes induced by the training process reduce the energy expense necessary to react to the stimulus.

Under some circumstances the training process can lead to subsensitive or supersensitive networks. Neither of them is optimal, because the former requires too much time to react, while the latter, when operating, makes use of unnecessarily too great a number of synaptic connections. In an optimally trained network a spatial pattern of "strong" synaptic connections develops.

The final geometry of the depolarization paths is, however, strongly dependent on the initial conditions. In a sense, the initial values of the synaptic couplings g_{ij} are "genetically" determined, while the synaptic plasticity reflects the "lifetime experience" or learning. A bad genetic inheritance constitutes a fundamental barrier for future success. The globally optimal solution for the problem of the shortest paths within the complex network is the one with the mean length of the paths proportional to the logarithm of the number of the network nodes. In the best case the initial state of a neuronal network involves all possible synaptic couplings being strong. After training these associations are not lost and the network operates in the globally optimal manner. In the worst case the neuronal networks contain some fraction of possibly bad associations (strong couplings assigned randomly to the synapses) and such networks are overperformed even by networks which initially contain no strong associations. In both cases the networks operate, however, far from the globally optimal shortest paths regime.

One may speculate that, like in optimization problems (e.g., Monte Carlo or simulated annealing) some level of noise may eventually drive the system towards the global optimum (the noise in a neuronal network can be simulated by allowing the neurons to generate spikes randomly, without any external excitation). I have checked that in the case of trained neuronal networks even a high level of noise does

TABLE I. Mean values (standard errors) of the lengths of depolarization paths $\langle L\rangle$ and reaction times $\langle t_A\rangle$ calculated for an external signal stimulating a receptor neuron and a random neuron. The results were obtained for trained neuronal networks built of 1000 neurons.

	External signal stimulating a receptor neuron		External signal stimulating a random neuron	
Initial conditions	$\langle L\rangle$	$\langle t_A\rangle$	$\langle L\rangle$	$\langle t_A\rangle$
all $g_{ij}(t=0)<V_{\mathrm{th}}-V_{\mathrm{base}}$	5.97 (0.08)	0.30 (0.004)	7.6 (0.2)	2.0 (0.16)
30% $g_{ij}(t=0)>V_{\mathrm{th}}-V_{\mathrm{base}}$	5.90 (0.08)	0.30 (0.004)	7.8 (0.2)	2.4 (0.11)
all $g_{ij}(t=0)>V_{\mathrm{th}}-V_{\mathrm{base}}$	3.80 (0.02)	0.19 (0.001)	6.8 (0.2)	3.0 (0.18)

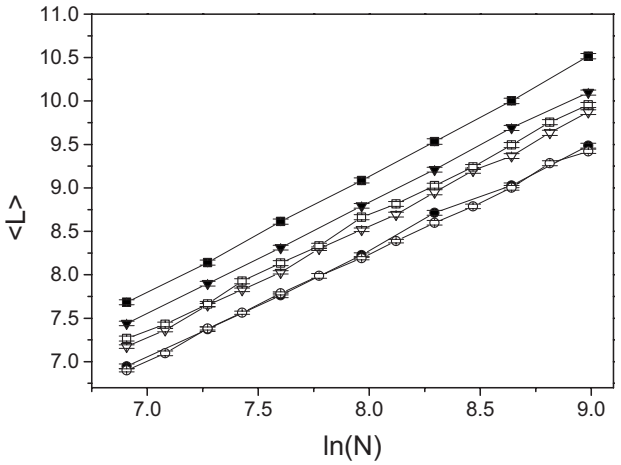

FIG. 6. The mean length $\langle L \rangle$ plotted vs the logarithm of the number of neurons N for trained (open symbols) and nontrained (solid symbols) neuronal networks in which neurons were allowed to spontaneously generate spikes with probability 0.2, without any external stimulation. The results plotted in the figure were obtained for different initial conditions $g_{ij}(t=0)$: all $g_{ij}(t=0) < V_{\text{th}} - V_{\text{base}}$ (triangles); 30% $g_{ij}(t=0) > V_{\text{th}} - V_{\text{base}}$ (squares); all $g_{ij}(t=0) > V_{\text{th}} - V_{\text{base}}$ (circles).

not fully diminish the results of unequal initial conditions. Moreover, undesirable effects of noise are decreased advantages of training (comparing nontrained and trained networks) and driving the system away from the global optimum (Fig. 6).

In the model of a neuronal network considered in the present study only one receptor neuron was present. Thus the information passed to the network can be coded only in the frequency pattern of the external stimulation. The network adjusts its answer to the stimulus in an obvious way: the bursts of activity are more frequent for more frequent stimulation. It is not, however, an advantage of training, because nontrained networks are actually also able to code external temporal information in the frequency of activity. In a more general case there can be multiple receptor neurons in a network and different depolarization signals can "interact" with one another.

In the paper the effects of training of networks built of integer-and-fire neurons were studied. The results were obtained for a simplified model of neuronal dynamics. The model of integer-and-fire neurons has only one compartment (the membrane potential), the simplest description of the synaptic dynamics (sum of delta functions) was used, and only one form of synaptic plasticity was considered. Thus I am not able to confirm that these results are general for any model of neuronal network.

However, the changes induced by the training are reasonable and can be beneficial. For the model studied I have shown that long-term synaptic plasticity optimizes the time of reaction and length of depolarization paths. First, optimizing these quantities can eventually increase the fitness of an organism to the environmental conditions. Second, it has been recently shown that the transmission of the depolarization signal in living neuronal networks can be described in terms of a branching process [16] and that the network operates optimally for a critical (equal to one) value of branching parameter, which is related to the number of synaptic connections used to transmit a depolarization signal. Because the long-term synaptic plasticity tends to decrease the number of "strong" synaptic connections it can possibly be the mechanism responsible for driving the network towards the critical state.

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