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Control of an agent in the multi-goal environment with homeostasis-based neural network

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Abstract

Here we present the model of bio-inspired neuron, and synaptic plasticity, incorporating cellular homeostasis. Network of such neurons is used for multi-goal oriented control task. It was showed that such a model provides adaptive and robust behavior for the controlled agent.

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1 Introduction

Simplified models of neural networks have important value for practical and theoretical tasks. Nevertheless, it is hard to achieve the level of adaptivity of natural neural systems by modeling them.

Presented paper is aimed to investigate the role of different cellular and synaptic homeostasis mechanisms in adaptive properties of neurocontrollers. Model of bio-inspired neuron, and synaptic plasticity, incorporating cellular homeostasis is presented in the paper. Functioning of a network of such neurons is analyzed. The model was for the first time presented in (Nikitin & Lukyanova, 2016). Here we investigate it to understand the dynamics and interconnections of the system in different setups.

The goal of the work is also to find out how homeostatically-dependant weight correction influences the training and efficiency of neurocontroller.

2 Neural model

Below we present the model of the neural cell, incorporating keeping of cellular homeostasis and homeostatically determined synaptic plasticity. In the model formal neuron performs a choice and with simple memory trace subsystem. Neuron performs summation of input signals and generates action potential based on its internal state. Efficiency of a neuron is measured by the grade of

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deviation of its homeostatic state from the optimal point. We propose the simplified approach to maintaining of homeostasis in neural sells and networks.

Network has neurons n_i, where $i = 1 \ldots m$ and m is a total number of neurons. Action potentials (AP) x_i is output values for the neurons and input values for the following neurons $\left(x_j\right)$. Total input signal to the neuron i - $g_i^{in}\left(t\right)$ at the time t is equal to:

$$g_i^{in}\left(t\right) = \sum_1^m \left(x_j\left(t\right) w_{i,j}\left(t\right)\right),\ x_j(t) \geq 1, \tag{1}$$

where:

$j = 1 \ldots m$ - numbers of neurons, connected with neuron i;

$x_j\left(t\right)$ - signal of the neuron j at the time t;

$w_{i,j}\left(t\right)$ - weight of the connection between the neurons i and j at the time t.

Input signals disturb homeostatic state q_i of the neuron:

$$q_i(t) = q_i(t-1) - k_{dam} g_i^{in}(t-1), \tag{2}$$

where:

k_{dam} - coefficient of damage of the homeostatic state

Neuron may generate AP x_i if it receives input signals $g_i^{in}\left(t\right)$, exceed the threshold T_{spike} and has sufficient energy storage for the AP generation. If input signals $g_i^{in}\left(t\right)$ are not strong enough and neuron has redundant energy storage it may generate AP stochastically.

Alternatives for the functioning of the model are presented in equations (3–5):

$$x_i(t) = \begin{cases} 1, if\ A \wedge B \\ 0, if\ A \wedge !B \\ 0, if\ !A \wedge !C \\ 1, if\ !A \wedge C \wedge D \end{cases}; \tag{3}$$

$$q_i\left(t\right) = \begin{cases} q_i\left(t-1\right), if\ A \wedge B \\ q_i\left(t-1\right) - k_{dam} g_i^{in}\left(t\right), if\ A \wedge !B \\ q_i\left(t-1\right) + \Delta_{q_i}\left(t\right), if\ !A \wedge !C \\ q_i\left(t-1\right), if\ !A \wedge C \wedge D \end{cases}; \tag{4}$$

$$e_i\left(t\right) = \begin{cases} e_i\left(t-1\right) + \Delta_{e_i}\left(t\right) - e_{spike}, if\ A \wedge B \\ e_i\left(t-1\right) + \Delta_{e_i}\left(t\right), if\ A \wedge !B \\ e_i\left(t-1\right) + \Delta_{e_i}\left(t\right) - \left|k_{eloss}\ \Delta_{q_i}\left(t\right)\right|, if\ !A \wedge !C \\ e_i\left(t-1\right) + \Delta_{e_i}\left(t\right) - e_{spike}, if\ !A \wedge C \wedge D \end{cases}; \tag{5}$$

where:

$\Delta_{e_i}\left(t\right)$ – value of neuronal energy refill;

$\Delta_{q_i}\left(t\right)$ – change of homeostatic state;

k_{eloss} – coefficient of energy loss for recovery of homeostasis;