The first version of openLSNS package is developed.

The core of openLSNS is developed and includes:

General information,

Architecture,

Kernels,

Interactions.

I. Simulation engine (the core of openLSNS package provides the basic abilities to perform computational simulations of neural networks of Hodgkin-Huxley type neurons).

1. Cells model

a) synaptic model;

b) ions dynamics;

c) ions current (including synapses);

d) membrane potential.

2. Network units

a) drives;

b) outputs;

c) feedbacks.

3. Biomechanics

a) muscles;

b) arm model.

II. Translator.

**Cells model.**

**Synaptic model**

***General information***

Synaptic current for postsynaptic neuron that generated by j-th synapse is calculated according to [Ermentrout&Terman, 2010, Destexhe et al., 1994, Destexhe&Mainen, 1994, Destexhe et al., 1998]:

(1)

Where is maximal conductance; is gate variable that characterizes the transmitter release; is the factor that defines how effectively the post-synaptic cell responds to neurotransmitters (=1 for the most synapses, except those the mechanism of synaptic plasticity is implemented); is reversal potential for *j*-th synapse.

Let suppose (for simplicity) that , are equal for N synapses (*j* = 1…N) and =1, then

(2)

According to (2) the synaptic current for postsynaptic neuron from all similar synapses (*j* = 1..N) is calculating as:

(3)

***Weighted sum***

(4)

Total synaptic current for postsynaptic neuron is:

(5)

where:

(6)

***Instant synapse***

The simplest model of transmitter release at *j*-th synapse between post- and pre- synaptic neurons is modeling by sigmoid function and is described as follow:

(7)

where: – rate of transmitter release; - weight of connection for *j*-th synapse between post- and pre- synaptic neurons; – the membrane potential of presynaptic neuron; - half-voltage; k-slope.

Total synaptic current for postsynaptic neuron is:

(8)

where:

(9)

***Pulse model of synapse (fast synapse)***

The model of transmitter release for fast synapse at *i*-th integration step [##ref] is described as:

(10)

where: - integration step; T - time constant; – rate of transmitter release; - weight of connection for *j*-th synapse between post- and pre- synaptic neurons; – Dirac function (1 then spike generated by presynaptic neuron; 0 otherwise); – the membrane potential of presynaptic neuron; =0; *i*=1…L.

Total synaptic current for postsynaptic neuron is:

(11)

where:

(12)

The proposed model can be used as rough approximation for the model of AMPA/GABA(a/b) synapses. The advantage of the proposed model of a synapse is that it is not necessary to store intermediate results of synaptic summation () into the local memory which improve the performance of synaptic computing.

**Implementation**

The equations (6, 9, 12) could be rewritten as linear recurrence equation:

(13)

where:

; *k* = 1..L

=0

*Blah-blah-blah*

***Model of NMDA synapse.***

***General information.***

The synaptic current for postsynaptic neuron that generated by j-th NMDA synapse is calculated similar to equation (1) [Destexhe&Mainen, 1994, Ermentrout&Terman, 2010]

(11)

where is maximal conductance; is gate variable that characterizes the transmitter release, is reversal potential for *j*-th synapse; z(V) represents the magnesium block and is calculating as:

(25)

The model of transmitter release [Destexhe et al., 1994, Destexhe&Mainen, 1994, Destexhe et al., 1998] described similar to the model of transmitter release for AMPA/GABA(a) synapses (see eq. 10-12).

**Implementation**

The implementation of simplified model of synaptic current of NMDA synapse is similar to the model of AMPA/GABA(a) synapses (see eq. 13, 14). The magnesium block is calculating according to (eq. 25).

The more complicated model of the synapse (see 26, 27) might be implemented similar to implementation of GABA(b) synapses.

1. **Presynaptic inhibition**

***General information***

The presynaptic inhibition affects to the rate of transmitter release in synaptic vesicles (parameter in all equation for calculating of the dynamics of transmitter release, see eq(s) 4, 9, 17 etc). Then the simplest model of presynaptic inhibition can be written as follow:

(28)

where: is maximal rate of transmitter release; is presynaptic inhibition.

**Implementation**

The model of modulation will be implemented if necessary

1. **Synaptic plasticity**

***General information***

**Implementation**

n/a

Ions current (including synapses).

where and are gate variables (activation and inactivation), is maximal conductance; is membrane potential and is reversal potential.

In general, the gate variables () are described as follow:

()

where is time constant, is steady-state value of correspondent gate variable (activation or inactivation, correspondingly).

Voltage dependent currents:

Implemented several types of description of gate variables:

**Generic description**

The steady state value is describes as follow:

The time constants of different subtypes of gate variables are describes as follow:

1) instant

2) generic description

3) modified generic description

4) 'ggate4' modified generic for A-current:

**Alpha-Beta model of ion current**

The steady state value is describes as follow:

, where:

1) instant

2) generic description

**Z-channels (DeShutter&Bauer)**

// activated ion channel. T\*d[M/H]/dt = [M/H]inf-[M/H];

//-----------------------------------------------------------------------------

// 3) 'zgate3' instant alpha/beta description (time constant is 0):

//=========================== zgate1 ==========================================

**Leakage**

<..... non specific model, specific model>

Ions dynamics.

Reversal potential (E = RT/Fz\*ln[Out]/[In])

Dynamics:

Ca-ions

Na-ions

Cells description

differential equation for membrane potential; definition of spike onsets.

()

Network units

a) drives;

b) outputs;

c) feedbacks.

Biomechanics

a) muscles;

b) arm model.

**References**

1. G.B. Ermentrout and D.H. Terman, Mathematical Foundations of Neuroscience, Interdisciplinary Applied Mathematics 35, DOI 10.1007/978-0-387-87708-27, Springer Science+Business Media, LLC 2010
2. Destexhe, A., and Mainen, Z.F. Synthesis of Models for Excitable Membranes, Synaptic Transmission and Neuromodulation Using a Common Kinetic Formalism. Journal Of Computational Neuroscience, 1, 195-230, 1994
3. Destexhe, A., Mainen, Z.F. and Sejnowski, T.J. An efficient method for computing synaptic conductances based on a kinetic model of receptor binding Neural Computation 6: 10-14, 1994.
4. Destexhe, A., Mainen, Z.F. and Sejnowski, T.J. Kinetic models of synaptic transmission. In: Methods in Neuronal Modeling (2nd edition; edited by Koch, C. and Segev, I.), MIT press, Cambridge, 1998, pp. 1-25
5. Destexhe, A. and Sejnowski, T.J. G-protein activation kinetics and spill-over of GABA may account for differences between inhibitory responses in the hippocampus and thalamus. Proc. Natl. Acad. Sci. USA 92: 9515-9519, 1995.
6. Destexhe, A., Bal, T., McCormick, D.A. and Sejnowski, T.J. Ionic mechanisms underlying synchronized oscillations and propagating waves in a model of ferret thalamic slices. Journal of Neurophysiology 76: 2049-2070, 1996.
7. X.-J. Wang, J. Tegne, C. Constantinidis, and P. S. Goldman-Rakic Division of labor among distinct subtypes of inhibitory neurons in a cortical microcircuit of working memory. PNAS, V101, No 5, 1368 –1373, 2004