Integrate-and-fire neuron model with STDP plasticity bounded by neurotransmitter receptor pool

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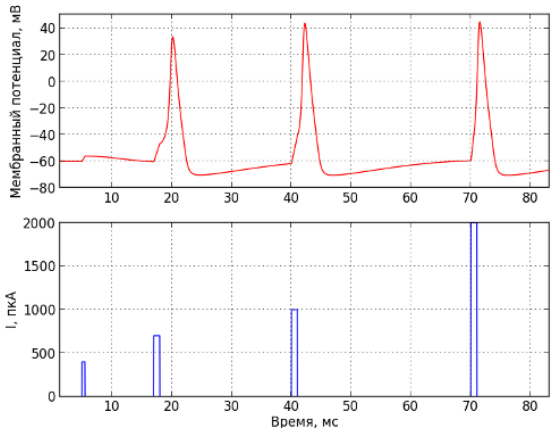
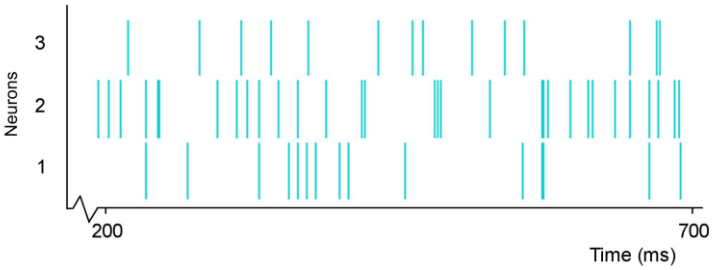
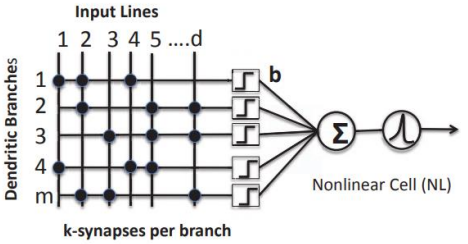
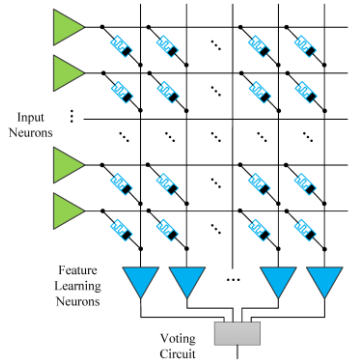
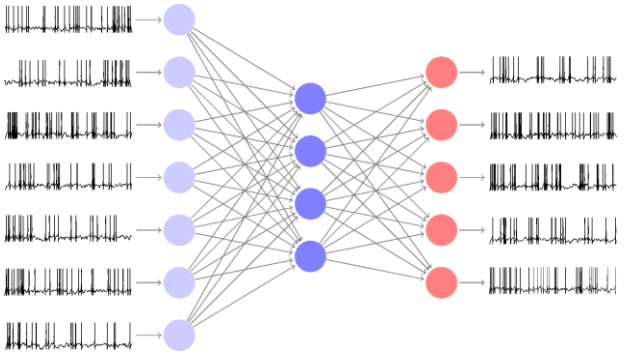
Spiking Neural Networks

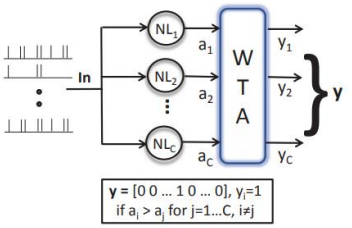


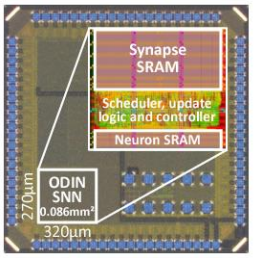






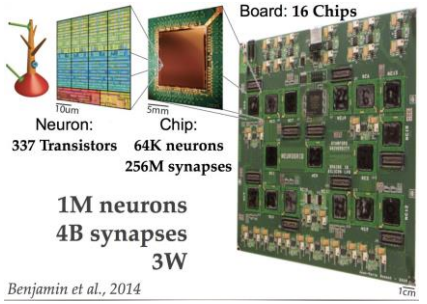


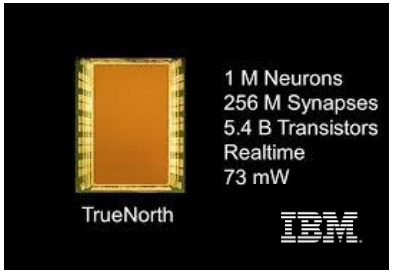




b)

Technology	28nm FDSOI
Implementation	Digital
Area	0.086mm²
# neurons	256
# synapses	64k
# Izhikevich behav.	20
Online learning	SDSP, (3+1)-bit weight
Time constant	Biological to accelerated
Supply voltage	0.55V – 1.0V
Leakage power (P_{leak})	27.3µW @0.55V
Idle power (P_{idle})	1.78µW/MHz @0.55V
Incr. energy/SOP (E_{SOP})	8.43pJ @0.55V
Global energy/SOP ($E_{tot,SOP}$)	>12.7pJ @0.55V

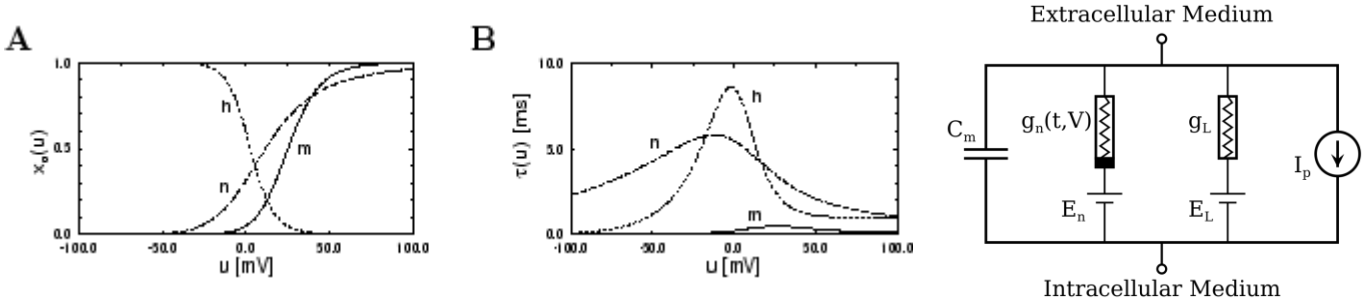




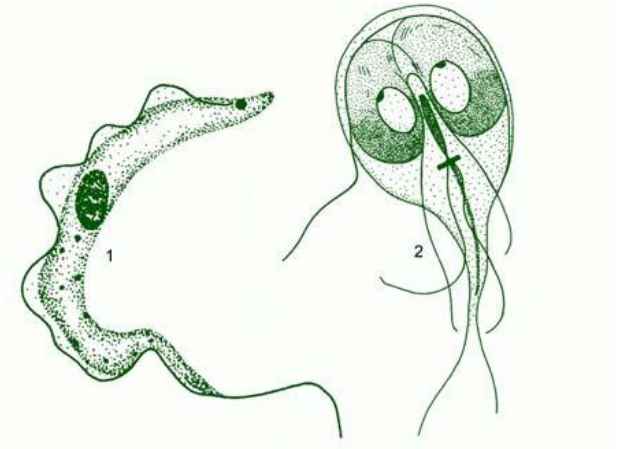
Neurons generate binary signals and are trained according to biomimetic rules (Hebbian learning). Networks have a sparse structure.

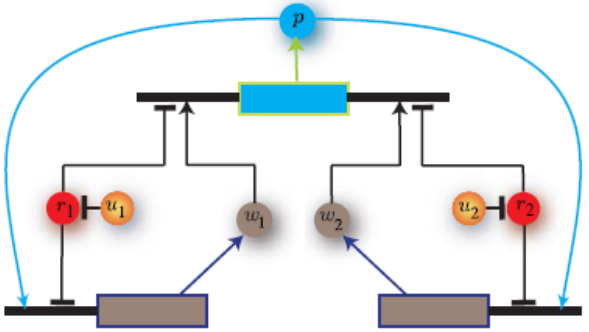
Motivation

Currently simulation of neurons is descriptive and assumes neurons as passive transmitters



Even the unicellulars act and learn in the complex world using limited sensing and reasoning

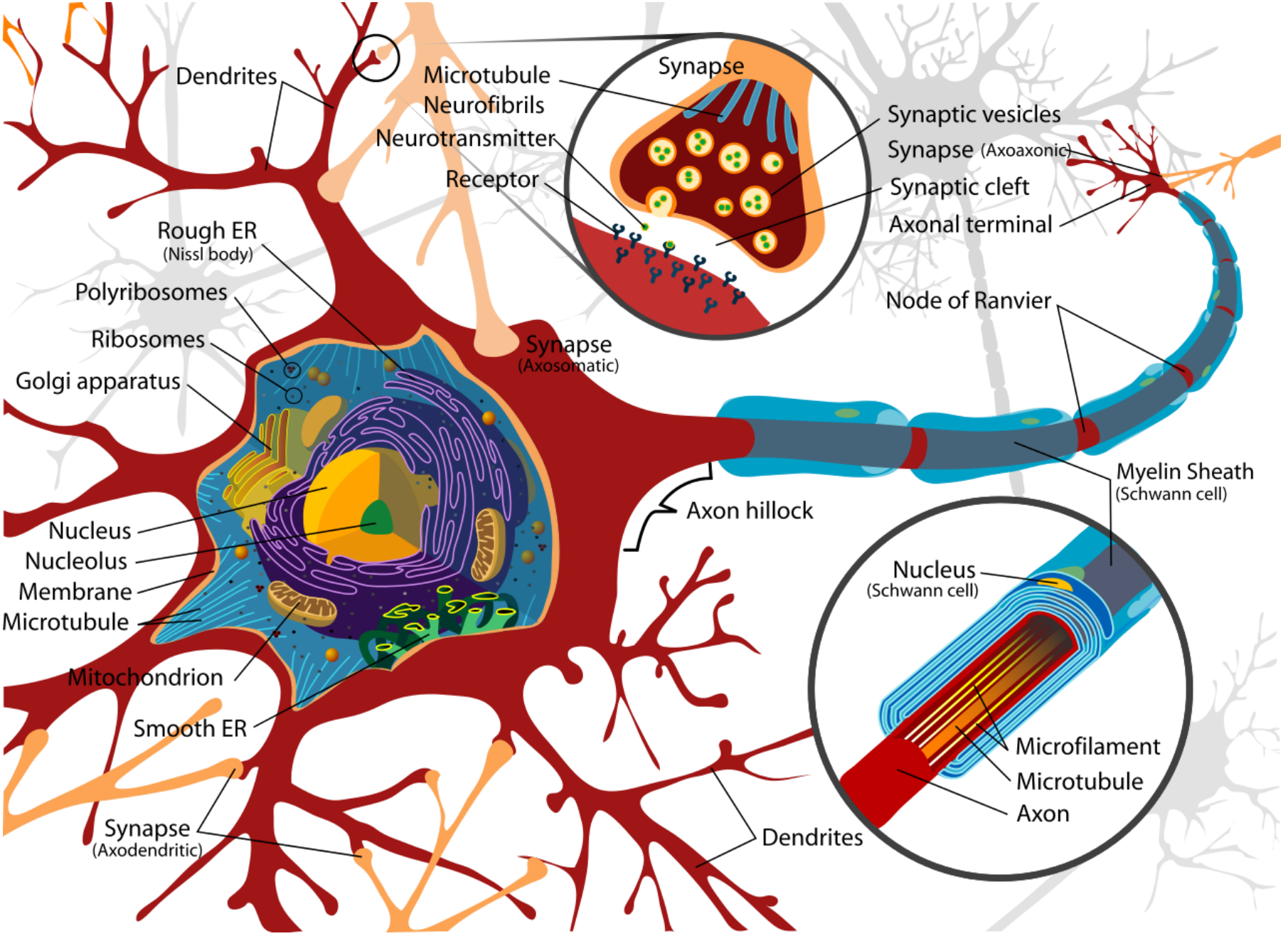




Simulation of conditional learning in unicellulars based on phosphorylation cycle (Fernando et al, 2008)

Motivation

Neurons are very complex cells and might be very big and compartmentalized

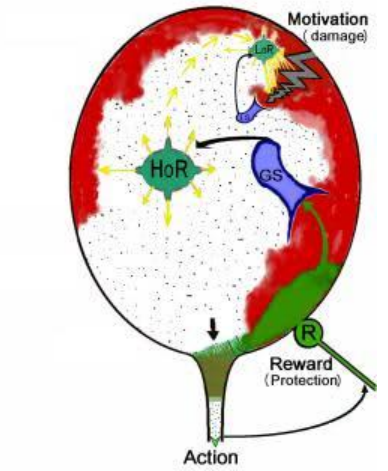


The complexity of even a single neuron. Credit: Mariana Ruiz Villarreal, Wikipedia Commons

Motivation

- Simulation of neurons often does not take into account an internal cellular adaptability (limited self-organizing and biological plausibility)
- Intrinsically motivated models might be the solution to create real world applications
- Inclusion of reward into the plasticity rule leads to emergence of self-organized adaptive behavior [Kappel et al, 2017; Zenke, F. and Ganguli, S., 2018]

We may link the intrinsic motivation of neural cells with the synaptic plasticity rule and have intrinsically motivated distributed RL approach



Neuronal intracellular homeostasis model
[Tsitolovsky, 2005]

Adaptive correction of neuronal activity

Some important processes:

- synaptic plasticity, including homeostatic, [*Turrigiano, 2008*]
- regulation of intracellular dynamics of transport and secretion of neurotransmitter receptors, [*Antunes and Simoes-de-Souza, 2018*], [*Hanus et al, 2014*], [*Kneussel and Hausrat, 2016*]
- correction of the cell membrane excitability threshold, [*Franklin et al, 1992*]
- adaptive functioning intracellular regulatory networks. [*Szita et al, 2006*]

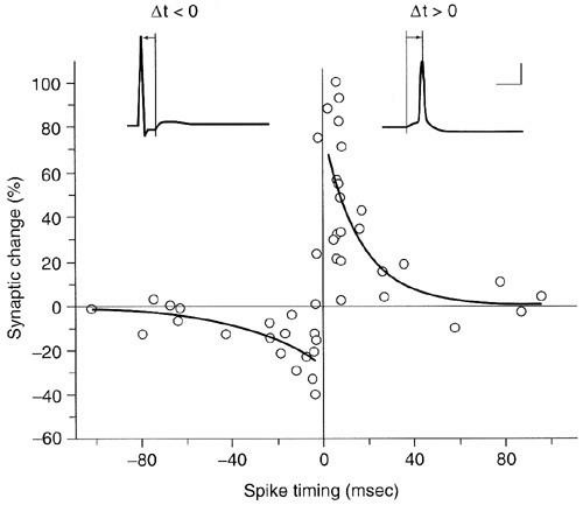
Synaptic plasticity

We consider three processes of synaptic plasticity, depending on neurotransmitter receptors:

- **spike-time dependent plasticity (STDP)** (positive feedback loop),
- **activity-dependent receptor degradation (ADRD)** (negative feedback loop)
- **synaptic scaling (SS)** (heterosynaptic homeostatic stabilization).

$$\Delta W_{nm}^{STDP} = \begin{cases} A_+(W_{nm}) \cdot e^{\frac{-\Delta t}{\tau_+}}, & \Delta t \geq 0 \\ -A_-(W_{nm}) \cdot e^{\frac{\Delta t}{\tau_-}}, & \Delta t < 0 \end{cases}$$

where: $\Delta W_{nm_{STDP}}$ – update of the synaptic weight, where nm – is a synapse m on the dendrite n, $\Delta t = t_{sp}^{(pre)} - t_{sp}^{(post)}$ – interval between the occurrence of subsequent spikes at presynaptic and postsynaptic neurons, τ_+ and τ_- – a time interval, which may cause successful correlation effects, A_+ and A_- – coefficients that reflect the maximum possible rate of change of synaptic weights W_{nm} and may depend upon its current value.



$$A_+(W_{nm}) = (W_{max} - W_{nm}) \cdot A_+$$
$$A_-(W_{nm}) = (W_{nm} - W_{min}) \cdot A_-$$

where A_+ and A_- – positive constants.

$$\Delta W_{nm}^{ADRD} = k_{Pw} \cdot x_{nm}$$
$$x_{nm} = I_{nm} \cdot W_{nm}$$

$$\Delta W_{nm}^{STDP} = \begin{cases} \Delta W_{nm}^{STDP}, & \sum^{m} \Delta W_{m}^{STDP} \leq Pw_n \\ \Delta W_{nm}^{STDP} \cdot \frac{Pw_n}{\Delta Pw_n(\Delta W)}, & \sum^{m} \Delta W_{m}^{STDP} > Pw_n \end{cases}$$

$$\Delta W_n^{SS} = \alpha \cdot W_{nm} \cdot \left(1 - \frac{\sum W_m^{goal}}{\sum W_m}\right)$$

Neurotransmitter receptors: synthesis, transport and storage

Neurotransmitter receptors (here and below ***Pw***) are the special proteins, responsible for signal transmission in chemical synapses.

$$\Delta Pw_n = \begin{cases} \Delta Pw_{from\ soma}, & demand(Pw_n) > 0 \\ -\Delta Pw_n(\Delta W) - degr(Pw_n), & demand(Pw_n) \le 0 \end{cases}$$

$$\Delta Pw_{from\ soma} = \begin{cases} Pw_{soma}, & demand(Pw_n) \ge Pw_{soma} \\ demand(Pw_n), & demand(Pw_n) < Pw_{soma} \end{cases}$$

$$demand(Pw_n) = \begin{cases} want(Pw_n), & want(Pw_n) \ge 0 \\ 0, & want(Pw_n) < 0 \end{cases}$$

$$want(Pw_n) = -S \cdot \left(Pw_n - \Delta Pw_n(\Delta W) - degr(Pw_n)\right)$$

$$S = \alpha \cdot \left(\frac{1}{1 + e^{-C_n}} - \beta\right)$$

$$\Delta Pw_n(\Delta W) = \begin{cases} \Delta W_{STDP}, & \Delta W_{STDP} \ge 0 \\ \alpha \Delta W_{STDP}, & \Delta W_{STDP} < 0 \end{cases}$$

$$degr(Pw_n) = k_{Pw} \cdot \left(\sum^{m} x_m\right)$$

$$secr(Pw_{soma}) = \alpha \cdot \left(1 - \frac{1}{1 + e^{-C_{soma}}}\right) \cdot \left(\frac{1}{1 + e^{-k_{secr}}} - 0{,}5\right)$$

The learning processes depend on the dynamics of the storage of neurotransmitter receptors. Together with synaptic plasticity, they cause a change in the strength of the synaptic interconnections between nerve cells. These processes can be defined as ”dendritic” part of the learning in the neuron.

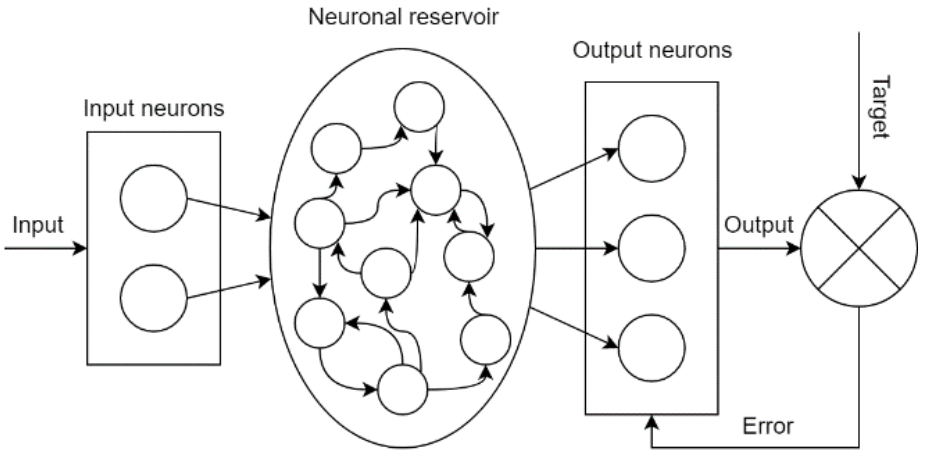
Complex cellular networks as reservoir-computing

Genetic regulation is a non-linear dynamic information processing that occurs in the network structure and has a complex interaction graph consisting of the work of genetic regulatory networks (GRN) and protein interactions. In general, all this can be called **complex cellular networks (CCN)**. CCN was modeled using **echo state networks (ESN)**.

For a predictive correction of this level of excitability, we can assume the action of **CCN**, as **ESN** in the task of **reinforcement learning**.

Modeling of genetic regulatory networks of cells using ESN has been proposed in [*Szita et al, 2006*].

We propose to use the ESN for the creation of an adapted Q-learning system for dynamic optimization of neuron parameters.



Level of the secretion of neurotransmitter receptors (ΔPw_{global}) were controlled by ESN giving **three options of actions: to decrease, increase and leave unchanged ΔPw_{global} .**

Model of a homeostatic neuron with predictive activity correction

We combine:

- the model of synaptic plasticity, including STDP, synaptic scaling and degradation components,
- activity-dependent regulation of intracellular dynamics of transport and secretion of neurotransmitter receptors,
- the reservoir-computing paradigm for predictive regulation of these parameters.

Model of a homeostatic neuron with predictive activity correction

Let there be a neuron consisting of soma, ***n*** dendritic compartments, each of which collects the signal from the ***m*** synapses. The excitatory postsynaptic potential (EPSP), received by the dendrite ***n*** through the synapse ***m***, is expressed in terms of the equation:

$$x_{nm} = I_{nm} \cdot W_{nm}$$

I_{nm} – the value of the input signal through the synapse m on the dendrite n. It can be 1 or 0, and it is the output value of the presynaptic neuron,
W_{nm_t} – the intensity of the synaptic connection (synaptic weight), which is the number of neurotransmitter receptors on the surface of the synaptic membrane,

$$\Delta W_{nm} = \Delta W_{nm}^{STDP} - \Delta W_{nm}^{ADRD} - \Delta W_{n}^{SS}$$

$$\Delta C_n = \left(\sum^{m} x_m\right) - decay(C_n)$$

$$decay(C_n) = \frac{\alpha}{e^{-\beta \cdot C_n}} - \gamma$$

$$\Delta C_{soma} = k_{Cg} \cdot O$$

$$O = \begin{cases} 1, & \sum x_{nm} \geq Tr \\ 0, & \sum x_{nm} < Tr \end{cases}$$

Tr – membrane threshold,
O – the output of a neuron

Model of a homeostatic neuron with predictive activity correction

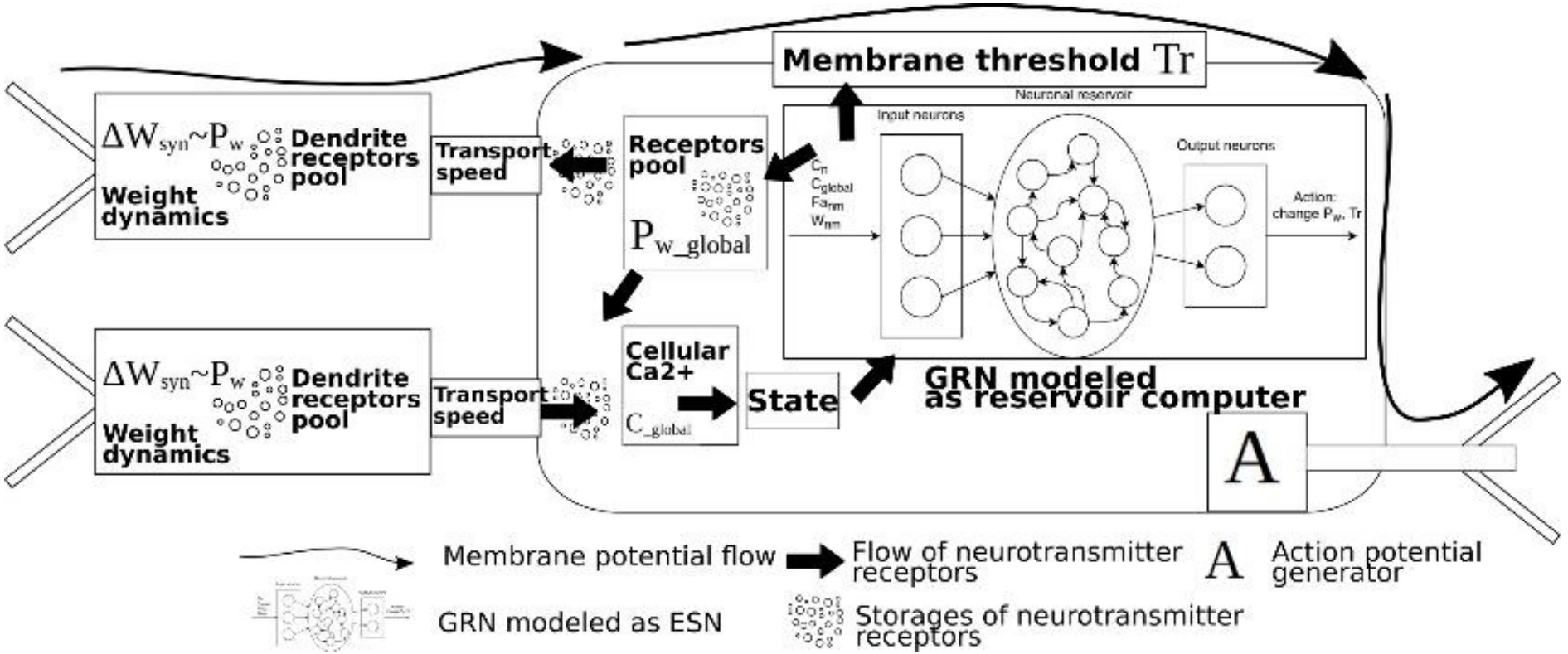
Internal values controlled by neuronal ESN reinforcement learning

State	$Pw_{dendrite}$, Pw_{soma} , C_{soma} , $C_{dendrite}$
Actions	Up/Down of Pw_{soma} secretion
Q-values	Divergence from $C_{optimal}$ level

Intraneural ESN-network controls $\boldsymbol{Pw_{soma}}$ during the Q-learning, thus constraining the growth of synaptic weights.

Neuron is trying to optimize an internal calcium level $\boldsymbol{C_{soma}}$

Model of a homeostatic neuron with predictive activity correction



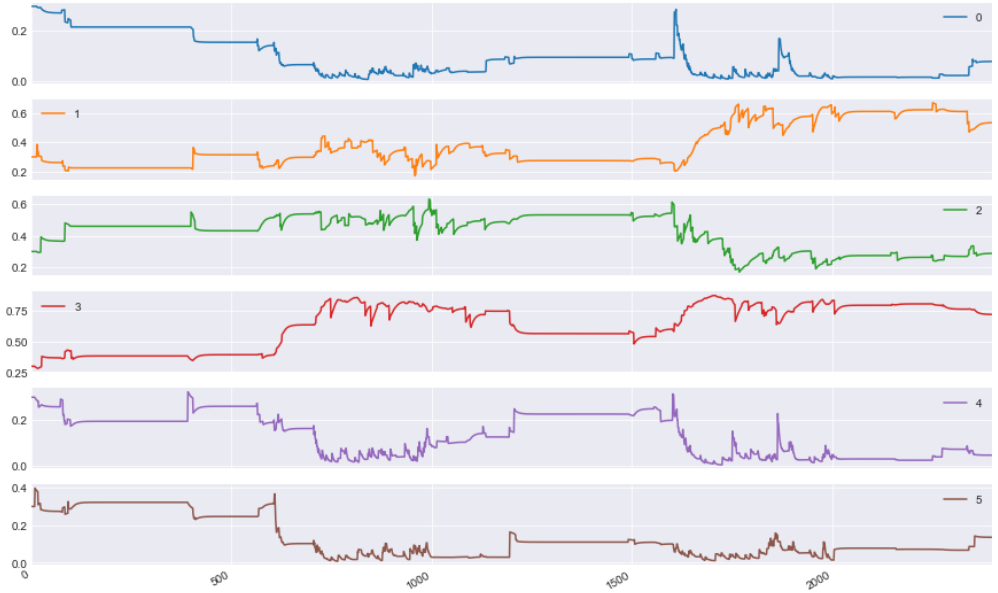
Weights can only be increased if the storage of neurotransmitters receptors Pw_{soma} has enough free molecules

Three processes of synaptic plasticity:

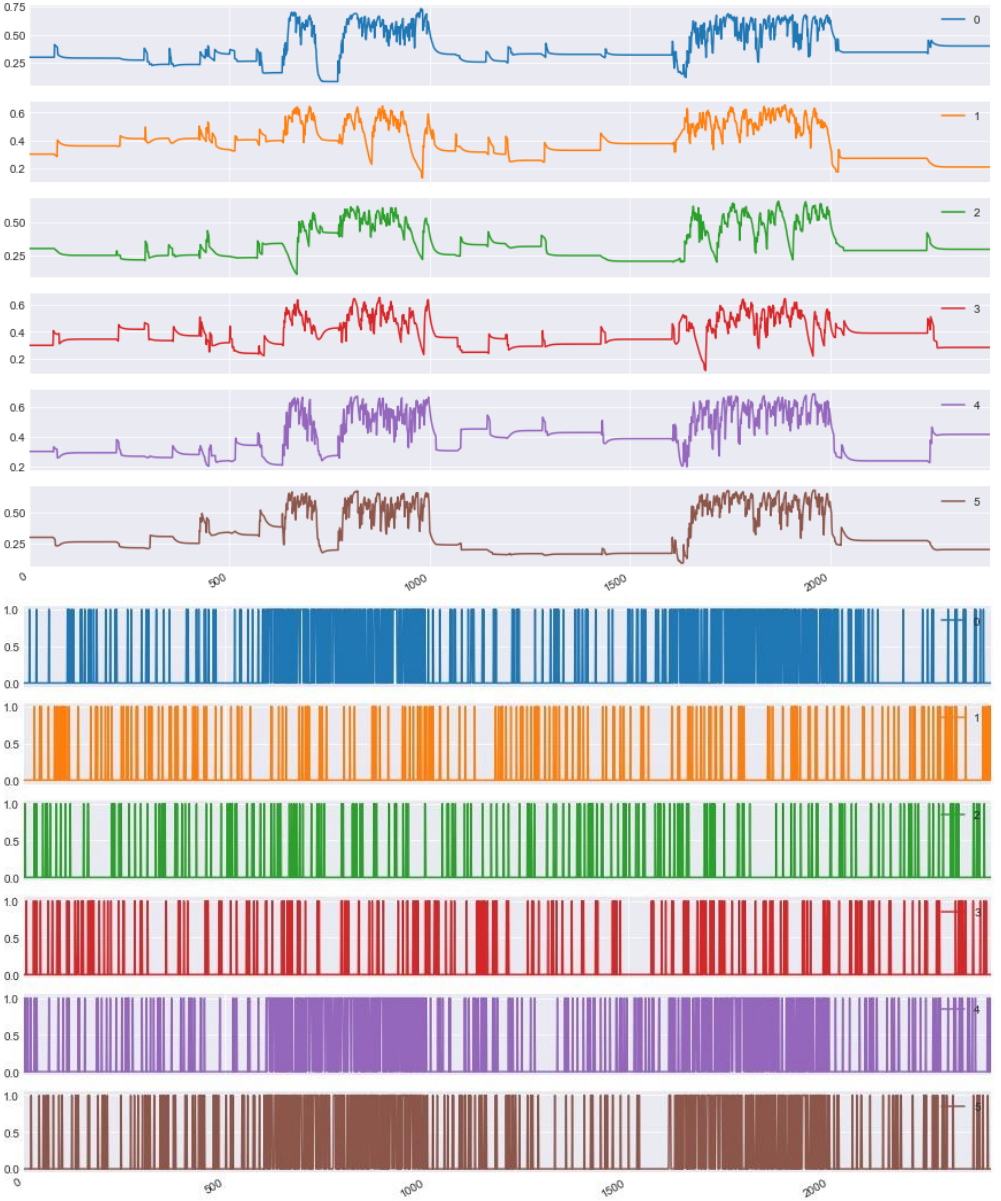
- **spike-time dependent plasticity (STDP)** (positive feedback loop),
- **activity-dependent receptor degradation (ADRD)** (negative feedback loop),
- **synaptic scaling (SS)** (heterosynaptic homeostatic stabilization).

Functionally defined Pw dynamics

Low frequency target



High frequency target

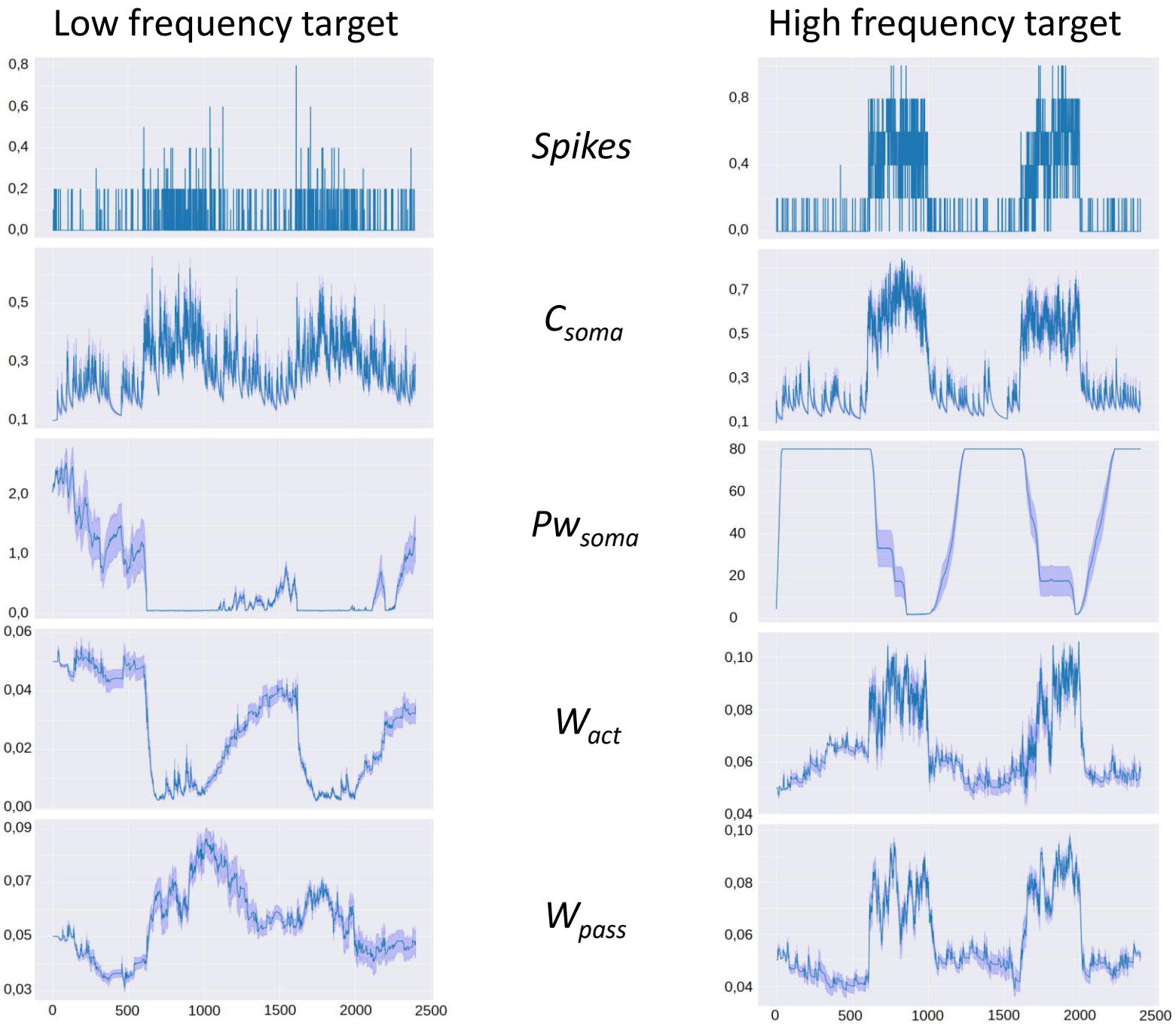


In all experiments, the operation of a neuron was compared under conditions of uniform input (probability 0.1) and the presence of sections with high-frequency random signals (probability 0.5) on steps 600-1000 and 1200-2000

In case of functionally defined *Pw*, its dynamics was opposite to Calcium level

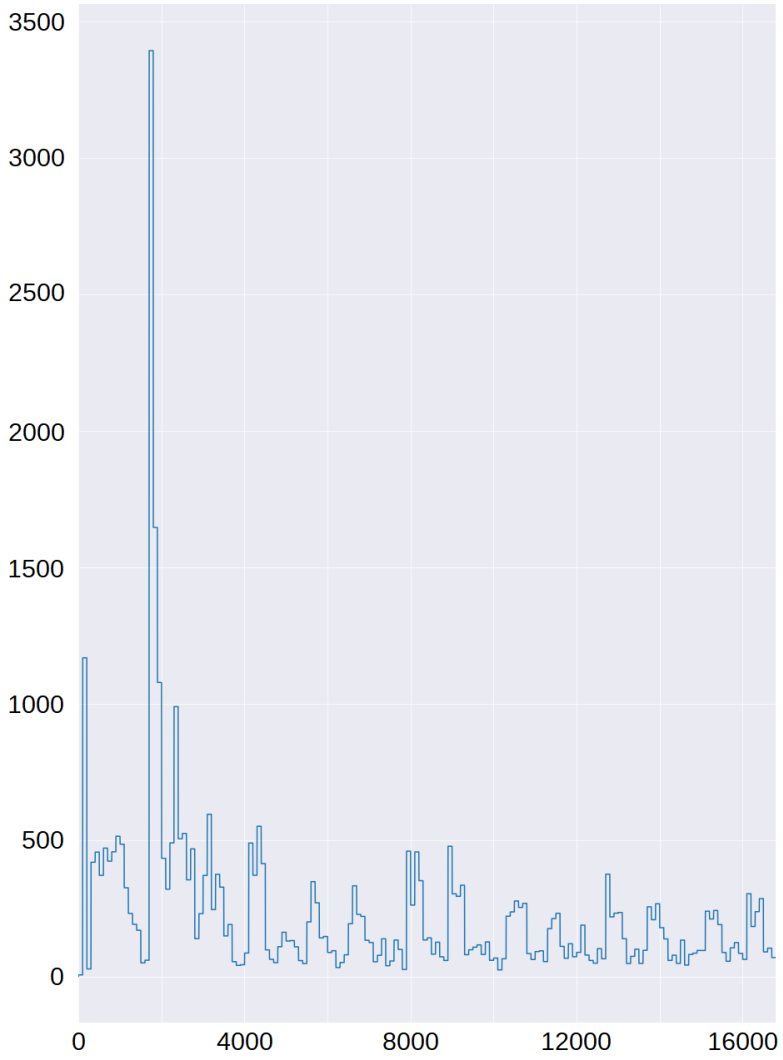
$$Pw_{soma}(t+1) = Pw_{soma}(t) + (1 - \tanh(C_{soma}(t))$$

Functionally defined Pw dynamics



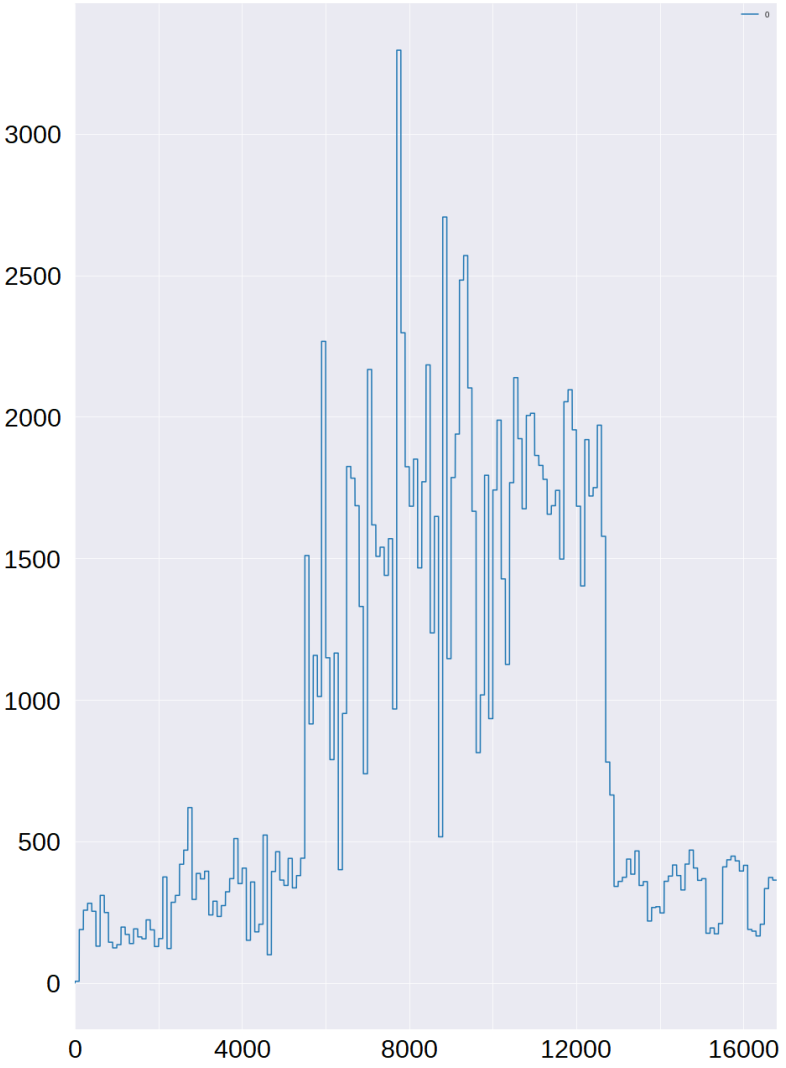
ESN controlled Pw dynamics

Low frequency target



ESN train error

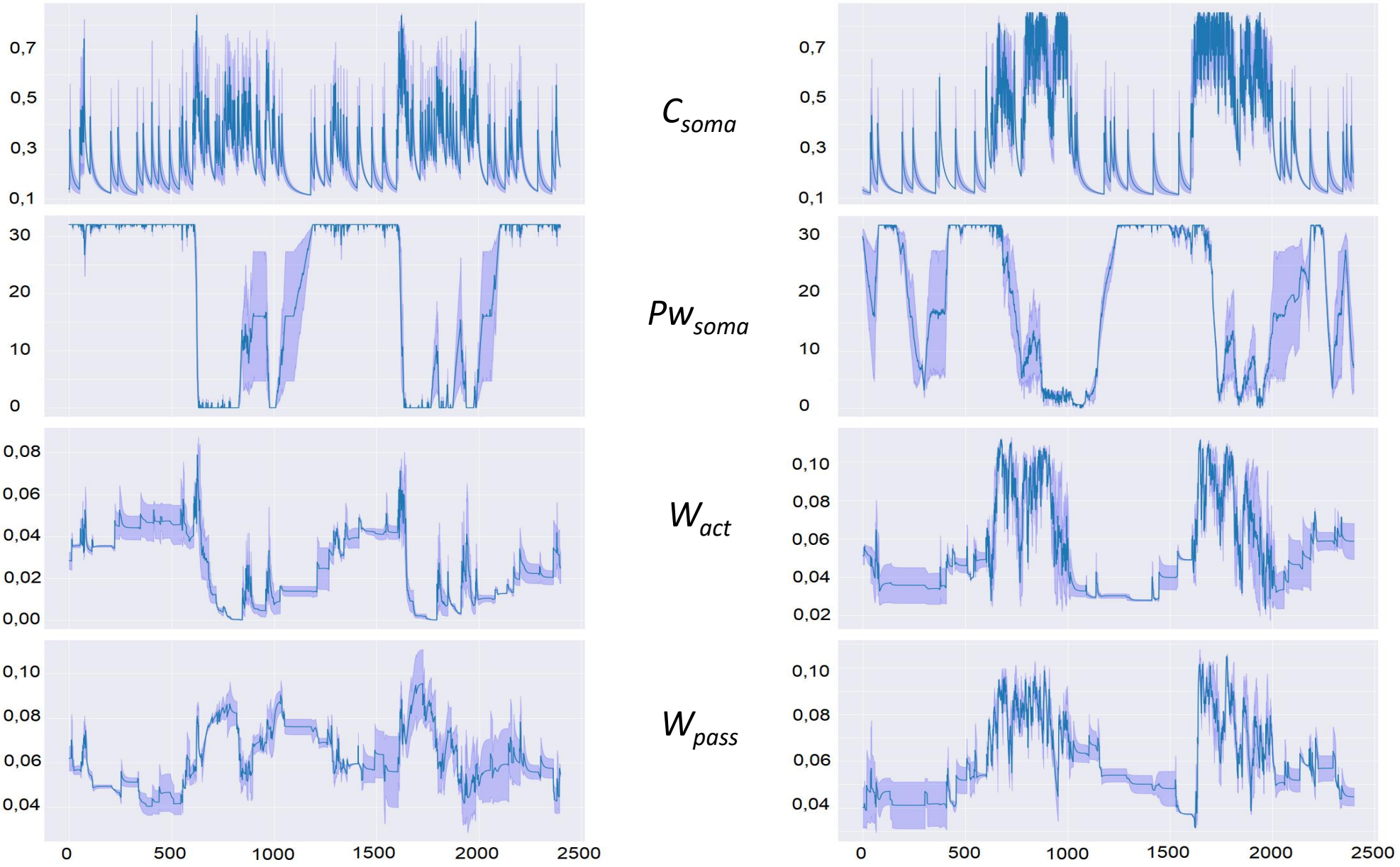
High frequency target



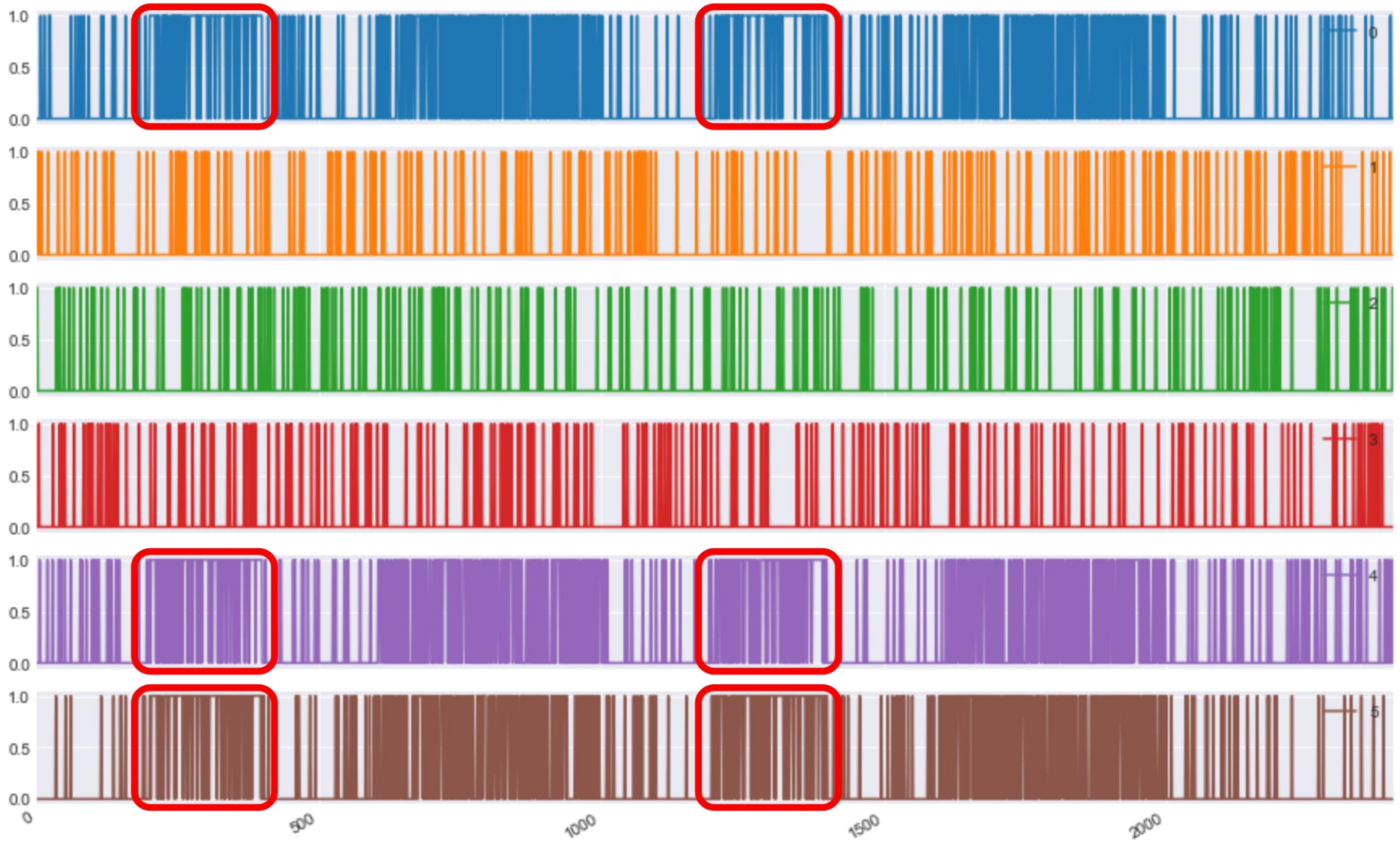
ESN controlled Pw dynamics

Low frequency target

High frequency target

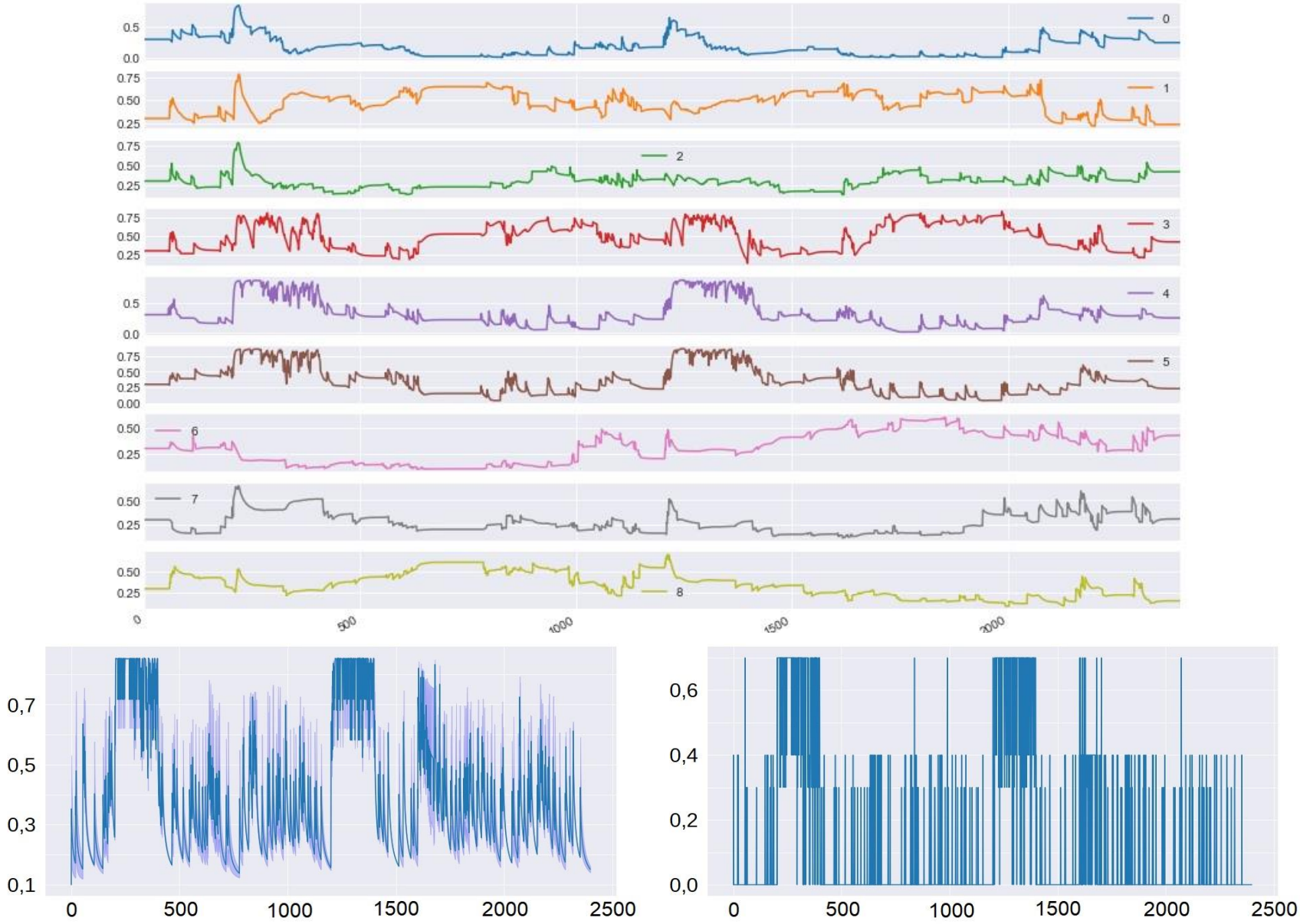


Correlated signals



On steps 200-400 and 1200-1400 neuron has correlated inputs (generated by stochastic process of common nature). Coincident signals were also delivered as before.

Correlated signals



Correlated signals led to the reliable spiking output. While coincident frequent signals still were blocked (note steps 600-1000, 1200-2000).

Conclusions

- A neuron model, including synaptic plasticity limited by the level of supply of neurotransmitter receptors was constructed.
- It was shown that, a neuron can selectively respond to certain frequencies of input signals depending on the mode of receptor production.
- Especially important feature of model that neuron with low-frequency target filters frequent coincident input, while reacting to correlated signals.
- Neurons of this model are suitable for creating spiking neural networks and can be used for unsupervised training tasks even on modern neuromorphic hardware (IBM TrueNorth).
- Python module based on common PyTorch framework was developed. It allows GPU accelerated network simulations and may be efficiently scaled to multiple nodes.

HPC GPU resources at CC FEB RAS

Presented work was performed using the resources of shared resource facility Data Center of FEB RAS

We also offer free resources to any Russian government funded institutions!

IBM Power nodes with GPU acceleration:

2 Power8 CPU (160 threads), 2 Tesla P100 GPU, 256 GB RAM

each node has ≈ 8 Tflops speed (Linpack)

Software: **DL:** TensorFlow, PyTorch, Cafee etc.; **Scientific packages:** ESSL, GAMESS-US, ABINIT, Quantum Espresso; CUDA OpenMP, OpenCL, OpenACC

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Thank you!

Oleg Nikitin

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