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Surgent, Daniel
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Spiking Neural Networks, Perspective

Post-Graduate Student:
Ing. Daniel Surgent

Institute of Computer Science of the ASCR, v. v. i.
Pod Vodárenskou věží 2

182 07 Prague 8, CZ

surgent@cs.cas.cz

Supervisor:
Ing. Marcel Jiřina , DrSc.

Institute of Computer Science of the ASCR, v. v. i.
Pod Vodárenskou věží 2

182 07 Prague 8, CZ

marcel@cs.cas.cz

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Abstract

Spiking neural networks are an offshoot of neural computation reasearch. In the recent years, however, we have witnessed a growing interest and shift of the emphasis in the artificial neural network community toward pulse-coupled neural networks with spike-timing as they can encode temporal information in their signals. This paper gives a brief overview of different spiking neuron models and we discuss their ability to operate in large complex networks as well as their evolvability.

1. Introduction

Artificial intelligence is a branch of computer science for which the biological inspiration seems to be crucial. Neural networks, as used in artificial intelligence, have traditionally been viewed as simplified models of neural processing in the brain, even though the first simple models of ANNs, known as the first generation neural networks [22], are considered more as mathematical or computational model for information processing based on connectionistics approach to computation and the relation between these models and the brain architecture is not in place.

From some point of view we can see the following generations of artificial neural networks as continuous acquiring of novel biological inspirations. For instance the third generation of neural networks raised the level of biological realism by employing individual pulses which allow spatial-temporal information in communication and computation, like real neurons do [5]. In this paper, we consider this third generation, i.e., we compare and contrast various models of spiking neurons with special attention focused on their ability to efficiently operate in complex networks and on "how easily" they can change their behavior through genetic mutation - their evolvability. But first of all, we briefly explain what is common for all spiking neuron models and what differs them from their non-spiking predecessors.

2. On artificial spiking neurons

Artificial spiking neurons model the relationship between the inputs and the output of a neuron in terms of single spikes (or pulses), and describe how such input leads to the generation of output spikes. Non-spiking neuron models do not employ individual pulses, but their output signals are computed in each iteration and typically lie between 0 and 1. So they do not implement the element of time in communicating. These signals can also be seen as normalized firing rates (frequencies) of the neuron within a certain period of time, and therefore non-spiking neural network is a special case of spiking neural network from some point of view.

The classical point of view that neurons transmit information exclusively via modulations of their mean firing rates [5, 21] seems to be at odds with growing empirical evidence that the patterns can be found in the firing sequences of single neuron [31] or in the relative timing of spikes of multiple neurons [13, 29] forming a functional neuronal group [4].

In spiking neuron models the transmission of a single spike from one neuron to another is mediated by synapses at the point where the two neurons interact. An input, or presynaptic spike arrives at the synapse, which in turn releases neurotransmitter which then influences the state, or a membrane potential of the target, or postsynaptic neuron. When the value of this state crosses some firing threshold (some models do not implement a fixed threshold, see next section), the target neuron generates a spike, and the state is reset by a refractory response. The size of the impact of presynaptic spike is determined by the type and efficacy (weight) of the synapse. In biology, there are known two distinct groups of neurons: excitatory neurons, which synapses release

neurotransmitter that increases the membrane potential of a target cell, and inhibitory neurons with synapses, that decrease this potential [23]. A good discussion of artificial spiking neurons can be found in [9]. Let's take a look on some of the most useful models of spiking neurons.
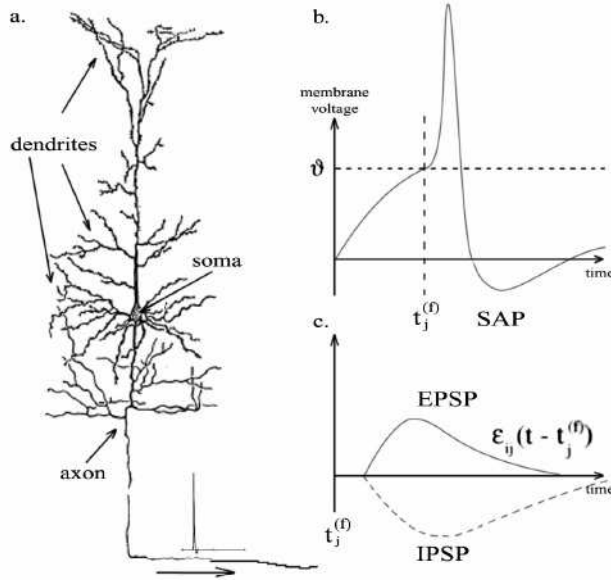
Figure 1: (a) Schematic drawing of a biological neuron.
(b) Incoming postsynaptic potentials alter the membrane voltage so it crosses threshold value θ; the neuron spike goes into a refractory state. (c) Typical forms of excitatory and inhibitory postsynaptic potentials over time. [8]

3. Spiking models

There are two main approaches in creating neuron models: computational neuroscience approach, trying to understand and model biological neuron, and connectionism on the other side of the river, trying to solve artificial intelligence related problems by creating interconnected networks of simple units (i.e. artificial neurons), which can exhibit complex global behavior, determined by the connections between the processing units and unit parameters. In this paper we try to bridge this two concepts in the sake of finding a neuron model (from the pool of the most useful models of spiking neurons) best suited for the use in large complex networks for solving some interesting problems outside biological modeling.

Below we provide a brief review of some widely used spiking neuron models as a shortened version of review [16]. But instead of Izhikevich's concern in simulating cortical spiking neurons, we ask different question: which model to use as a processing unit in some

large network architecture? Through this section, v denotes the membrane potential and v' denotes its derivative with respect to time. All the parameters in the models are chosen so that v has mV scale and the time has ms scale.

3.1. I&F

First group of spiking neuron models are known as integrate and fire neurons (I&F). The Leaky I&F neuron is one of the most widely used models in computational neuroscience

$$v' = I + a - bv, \text{if } v \geq v_{thresh}, \text{ then } v \leftarrow c,$$

where I is the input current, and a, b, c and v_{thresh} are the parameters. When the membrane potential v reaches the threshold value v_{thrash}, the neuron is said to fire a spike, and v is reset to c. From computational neuroscience point of view is the leaky I&F one of the worst models to use in cortical simulations, despite its simplicity.

The Leaky I&F has many extensions and modifications like I&F with adaptation, Integrate-and-Fire-or-Burst [34], Resonate-and-Fire and Quadratic I&F [20], but for purposes of this work we describe the Resonate-and-Fire neuron, which is two-dimensional (2-D) analogue of I&F neuron

$$z' = I + (b + i\omega)z$$
$$\text{if Im}(z) = a_{thresh}, \text{ then } z \leftarrow z_0(z)$$

where the real part of the complex variable z is the membrane potential. Here b, ω and a_{thresh} are parameters, and $z_0(z)$ is an arbitrary function describing activity-dependent after-spike reset.

By now, I&F family neurons are the only spiking neurons used outside the computational neuroscience community [1, 35] as they are easy to implement and their computational efficiency is far better than efficiency of Hodgkin-Huxley family spiking neuron models (see next section).

3.2. Hodgkin-Huxley

Second group of spiking neuron models are known as Hodgkin-Huxley-type (conductance-based) neurons as they are basicly variations and simplifications of the model developed by [12] based on data from the squid giant axon. It consists of four equations and tens of parameters, not provided here, describing membrane potential, activation of **Na** and **K** currents, and inactivation of **Na** current.

The Hodgin-Huxley model is one of the most important models in computational neuroscience not only because its parameters are biophysically meaningful and measurable, but also because they allow us to investigate questions related to synaptic integration, dendritic cable filtering, effects of dendritic morphology, the interplay between ionic currents, and other issues related to single cell dynamics. However, the end result can be at the small end tens of parameters which one must estimate or measure for an accurate model, and for complex systems of neurons not easily tractable by computer. So careful simplifications of the Hodkgin-Huxley model were therefore needed.

Sweeping simplifications to Hodgkin-Huxley model were introduced by FitzHugh-Nagumo model [6]. The parameters in this model

$$v' = a + bv + cv^2 + dv^3 - u$$
$$u' = \varepsilon(ev - u)$$

can be tuned so that model describes spiking dynamics of many resonator neurons. Although not clearly derivable from biology, the model allows for a simplified, immediately available dynamic, without being a trivial simplification [18].

From the other Hodgkin-Huxley family models we mention Morris-Lecar model [25] as a combination of Hodgkin-Huxley and FitzHugh-Nagumo into a voltage-gated **Ca** channel model with delayed-rectifier **K** channel, and the model of thalamic neuron - Hindmarsh-Rose [32], which is built upon the FitzHugh-Nagumo model and provide extra mathematical complexity that allows a great variety of dynamic behaviors for the membrane potential.

Although these models have much better dynamic properties than I&F family neuron models, and are better suited for computer simulations than Hodgkin-Huxley, they are yet still prohibitive in terms of large-scale simulations (see next section).

3.3. Izhikevich

A simple model of spiking neurons proposed recently by Izhikevich [15] combines the biological plausibility of Hodgkin-Huxley-type dynamics and the computational efficiency of I&F neurons

$$v' = 0.04v^2 + 5v + 140 - u + I$$
$$u' = a(bv - u)$$

with auxiliary after-spike resetting if $v \geq +30$ mV, then $v \leftarrow c, u \leftarrow u + d$.

Here v represents the membrane potential of the neuron and represents a membrane recovery variable, which accounts for the activation of $\mathbf{K}^+$ ionic currents and inactivation of $\mathbf{Na}^+$ ionic currents, and it provides negative feedback to v. After the spike reaches its apex ($+30$ mV), the membrane voltage and the recovery variable are reset. Synaptic currents or injected dc-currents are delivered via the variable I. If v skips over 30, then it is first reset to 30, and then to c so that all spikes have equal magnitudes.

The part $0.04v^2 + 5v + 140$ is chosen so that v has mV scale and the time has ms scale. The resting potential in the model is between -70 and -60 mV depending on the value of b. As most real neurons, the model does not have a fixed threshold (contrary to I&F neurons); Depending on the history of the membrane potential prior to the spike, the threshold potential can be as low as -55 mV or as high as -40 mV.

By now, this model was used exclusively for large-scale simulations of cortical neurons within computational neuroscience research (see [17]). In our opinion, it seems to be suitable for solving artificial intelligence related tasks as a processing unit of a complex neural network and we try to put some arguments for this statement in the next sections.
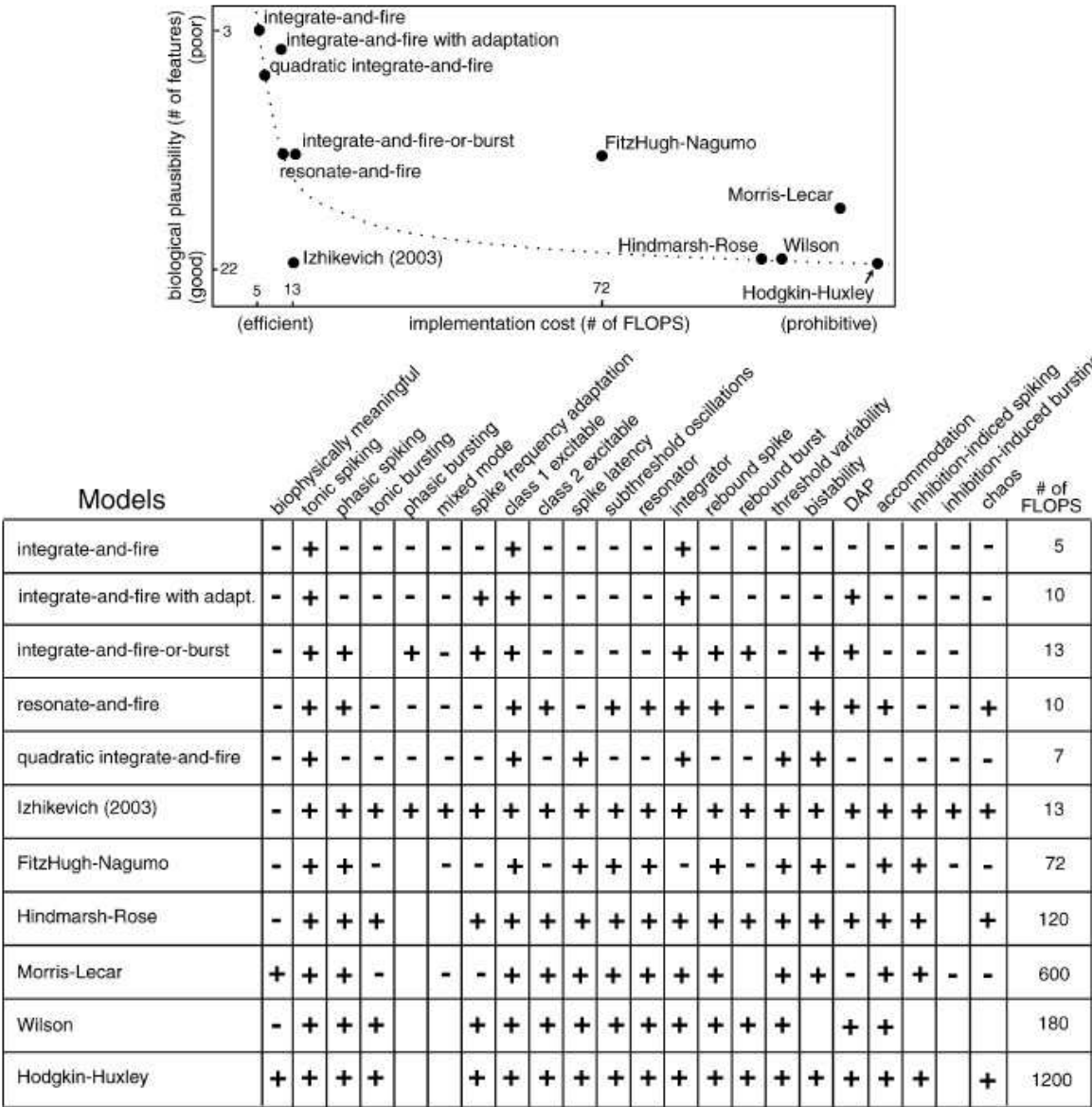
4. Computational efficiency

The notion of the efficiency of a simulation scheme is rather loosely defined in the computational neuroscience literature. [26] argue that efficiency should be defined as the simulation time required to achieve a prescribed accuracy goal. A scheme which constrains spike times to a timegrid is unsatisfactory in this respect if high accuracy is required, because the integration error drops only linearly with decreasing computation time step [11, 33].

In this section we again refer to the work by Izhikevich [16]. He compared some of the most useful models of spiking and bursting neurons from the biological plausibility and computational efficiency points of view. The summary of his comparison is in Fig. 2. To compare computational cost, he assumed that each model, written as dynamical system $x' = f(x)$, is implemented using a fixed-step first-order Euler method $x(t + \tau) = x(t) + \tau f(x(t))$ with the integration time step τ chosen to achieve reasonable numerical accuracy.

As we are interested in the idea of building a network connecting tens of thousands of neurons (maybe even more), we need to choose a neuron model that would be able to efficiently handle large numbers of neurons in

complex topologies. Considering this, it is prohibitive to use any of the Hodgkin-Huxley family neuron models (all models on the right half of the graph in Fig. 2), even the FitzHugh-Nagumo neuron model, which has "only" 72 floating-point operations is computationally too expensive. The reason why it is still not suitable, is that the efficiency of a single neuron is compared, and not the efficiency of the whole network. To estimate the computational efficacy of such network we must multiply the computational cost of its single element by the total number of its processing elements due to sequential computer processing (Fig. 3). Thus, we need to look somewhere else in a case of large-scale modeling.

Scatter plot (Figure 2, upper part):
Axes: biological plausibility (# of features) — from 3 (poor) to 22 (good) on vertical axis; implementation cost (# of FLOPS) — from (efficient) to (prohibitive) on horizontal axis. Points labeled:
- integrate-and-fire
- integrate-and-fire with adaptation
- quadratic integrate-and-fire
- integrate-and-fire-or-burst
- resonate-and-fire
- Izhikevich (2003)
- FitzHugh-Nagumo
- Morris-Lecar
- Hindmarsh-Rose
- Wilson
- Hodgkin-Huxley

(Horizontal FLOPS markers at 5, 13, 72.)

Models	biophysically meaningful	tonic spiking	phasic spiking	tonic bursting	phasic bursting	mixed mode	spike frequency adaptation	class 1 excitable	class 2 excitable	spike latency	subthreshold oscillations	resonator	integrator	rebound spike	rebound burst	threshold variability	bistability	DAP	accommodation	inhibition-indiced spiking	inhibition-induced bursting	chaos	# of FLOPS
integrate-and-fire	-	+	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	5
integrate-and-fire with adapt.	-	+	-	-	-	-	+	+	-	-	-	-	+	-	-	-	-	+	-	-	-	-	10
integrate-and-fire-or-burst	-	+	+		+	-	+	+	-	-	-	-	+	+	+	-	+	+	-	-	-		13
resonate-and-fire	-	+	+	-	-	-	-	+	+	-	+	+	+	+	-	-	+	+	+	-	-	+	10
quadratic integrate-and-fire	-	+	-	-	-	-	-	+	-	+	-	-	+	-	-	+	+	-	-	-	-	-	7
Izhikevich (2003)	-	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	13
FitzHugh-Nagumo	-	+	+	-		-	-	+	-	+	+	+	-	+	-	+	+	-	+	+	-	-	72
Hindmarsh-Rose	-	+	+	+		+	+	+	+	+	+	+	+	+	+	+	+	+	+			+	120
Morris-Lecar	+	+	+	-		-	-	+	+	+	+	+	+	+		+	+	-	+	+	-	-	600
Wilson	-	+	+	+		+	+	+	+	+	+	+	+	+	+	+		+	+				180
Hodgkin-Huxley	+	+	+	+		+	+	+	+	+	+	+	+	+	+	+	+	+	+	+		+	1200

Figure 2: Comparison of the neuro-computational properties of spiking and bursting models. "of FLOPS" is an approximate number of floating point operations (addition, multiplication, etc.) needed to simulate the model during a 1 ms time span. Each empty square indicates the property that the model should exhibit in principle (in theory) if the parameters are chosen appropriately, but the author failed to find the parameters within a reasonable period of time. [16]
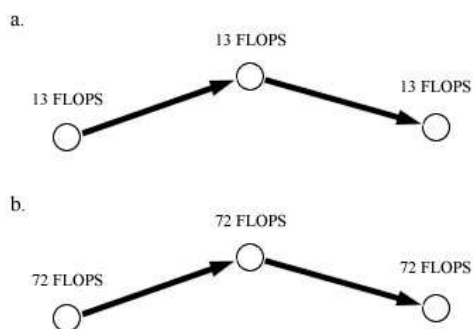
Figure 3: When the implementation is sequential (not parallel), the difference in efficiency of single neurons must be multiplied by the total number of the network's neurons to get the difference in efficiency for the networks. In this sample case the neurons efficiency difference is 59 FLOPS whereas the implementation cost difference for the networks is 177 FLOPS. (a) A sample network of Integrate-and-Fire-or-Burst neurons. (b) The same sample network with FitzHugh-Nagumo neurons.

If we want to simulate tens of thousands of spiking neurons in real time with 1 ms resolution, then there are plenty of models to choose from (all models on the left half of the graph in Fig. 2). The most efficient is the Leaky I&F neuron. It is the simplest model to implement when the integration time step τ is 1 ms. Indeed, the iteration $v(t + 1) = v(t) + I + a - bv(t)$ takes only four floating-point operations (additions, multiplications, etc.) plus one comparison with the threshold v_{thrash}.

The other I & F-type models are practically as efficient as the Leaky I & F (see table in Fig. 2) and are usable for large-scale modeling. State-of-the-art solvers for networks of I&F neurons allow for routine simulations of networks of some 10^5 neurons and 10^9 connections (synapses) on moderate computer clusters [28].

5. Evolvability

First of all it should be noted that the language of this section moves between bilological and that of artificial intelligence systems, so potentially biological terms are used in less orthodox manner than that used in biological literature.

The human brain contains over 11 billion specialized nerve cells, or neurons, capable of receiving, processing, and relaying the electrochemical pulses on which all our sensations, actions, thoughts, and emotions depend [7]. But it is not the sheer number of neurons alone that is most striking about the brain, but how they are organized and interconnected.

Despite our goal is not to create an artificial brain, it is hardly imaginable to build a large-scale network with spatio-temporal structure of activity without a proper construction algorithm. In our opinion, the only way how to solve this complex problem is to use evolutionary computation techniques (e.g. genetic algorithms, artificial embryogeny). However, this approach is considerably challenging and it has many unresolved issues. Through this section we try to shed some light on the problem of system's ability to evolve, and we try to apply this information to compare different spiking neuron models.

Evolvability is a concept in evolutionary biology that tries to measure an organism's ability to evolve (see [2, 19, 27, 37]). We see this concept as an important part of the design process of a system, which we want to evolve. As estimating a system's evolvability is not an easy task, we divide the problem in two aspects, which we think are crucial, namely phenotype-genotype compression and evolutionary potential.

5.1. Phenotype-genotype compression

From the evolutionary neuroscience point of view, there is a vaxing problem with the notion that genome provides complete information for the construction of the nervous system of humans and other mammals. It is estimated that just human neocortex alone has about 10^{15} (one thousand million million) synapses [14]. Since the human genome has only about 3.5 billion (3.5 x 10^9) bits of information (nucleotide base pairs), with 30% to 70% of these appearing silent [3], some neural and molecular scientists have concluded that our genes simply do not have enough storage capacity to specify all of these connections, in addition to including information on the location and type of each neuron plus similar information for the rest of the body. Considering this, there must be some kind of phenotype-genotype compression for every biological unit with no exception for neurons.

Most probably is such representational efficiency made possible through gene reuse mechanism. Natural organisms implement gene reuse through a process of development, or embryogeny. The same genes can be used at different points in development for different purposes, and the order in which activations of genes take place determines when and where a particular gene is expressed [30].

In our opinion there is present more general phenomenon, that is, principle of reducing the number of genes by preserving phenotype functionality, either by gene reuse or by other mechanism. Let us give an exaplme for better understandig: suppose we want to compare

evolvability of two neural cells with exactly the same current behavior but different length of their genomes (the neuron with shorter genome has better phenotype-genotype compression). Let's say there is some novel function that both neurons are able to perform if their genome is changed accordingly (by some mutations). We say that the neuron with shorter genome has better chance to gain the novel function faster, simply because its genome needs fewer mutations to "search the space of its mutants". Therefore we can conclude that the neural cell with shorter genome has better ability to evolve and would be more successful than the neuron with longer genome, in case that the novel function increases organisms's chance for reproduction.

To put it in analogy with spiking neuron models, we define genome of given neuron model (i.e. some piece of genome of the network) as a set of dimensionless parameters of the model, and we define its phenotype as a full description of the model - its differential equations with given values of its parameters, i.e, the model's dynamical behavior.

If we want to compare evolvability of different spiking models considering only their phenotype-genotype compression, than the models with fewer parametes are better, with the Resonate-and-Fire neuron as the most evolvable (it has only three parameters plus an arbitrary function) and Hodgkin-Huxley with the worst phenotype-genotype compression (Fig. 4). If this two neuron models behave as integrators and we want them to respond as resonators (see [16]), we predict that genome (parameters) of the Resonate-and-Fire neuron needs much fewer mutations to change its phenotype (dynamical behavior) than genome of the Hodgkin-Huxley neuron.
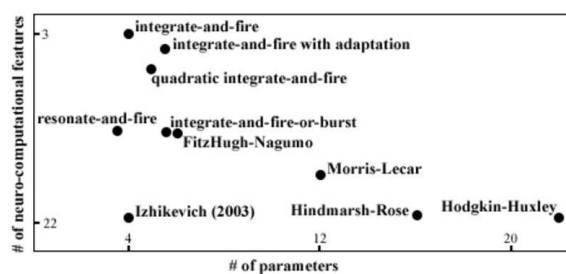
Figure 4: Comparison of the number of dimensionless parameters and the neuro-computational properties of spiking neuron models. Location of most of the dots is given approximately as the number of different parameters can vary for some of the models.

Note that we have intentionally choosed neuro-computational features that both neuron models are able to reproduce (see table in Fig. 2). What if chosen novel function would be the tonic bursting? It is clear that the Resonate-and-Fire neuron model is not able to evolve that way, thus we should not compare the neuron models only by phenotype-genotype compression, i.e, we should also compare their number of different neuro-computational properties. This conclusion leads to another important aspect of evolvability - evolutionary potential.

5.2. Evolutionary potential

The term evolutionary potential was defined from the genetics point of view as the array of successful mutants of selected gene for chosen novel function [10]. In case of neural cell we can define its evolutionary potential as the array of successful mutants of selected neuron for chosen novel behavior (i.e. response) of the neuron. Such definition does not exclude the existence of some novel behavior for which there are no mutants of given neural cell at all (its internal structure and genetic constitution simply does not allow to be changed that way). So the question is, how many different novel behaviors can a neural cell acquire through its evolution, and how many succesful mutants exist for each of these behaviors?

It seems really difficult to answer this question for biological neurons but not as difficult for artificial neurons, which we are able to investigate as dynamical systems. At least we are able to give more or less satisfying answer on the first part of the question above. If we take a look at the table in Fig. 2, we see that I&F family models can not reproduce some of the most important neuro-computational properties of real neurons, and therefore it seems that they are in general less evolvable than Hodgkin-Huxley-type neurons considering evolutionary potential. But more serious research on evolutionary potential of dynamical systems is needed and it would be interesting to investigate all of the dynamical behavior possibilities for spiking neuron models, and not "only" 20 (plus chaos) of the most prominent features of biological spiking neurons (table in Fig. 2).

6. Discussion

Neural structures are very well suited for complex information processing and it seems that the current research in computational neuroscience, evolutionary neuroscience, evolutionary computation, as well as in artificial neural networks has a promising future ahead.

Even though, almost all of the models used in computational neuroscience were created mainly to model physiologically realistic spike trains, some of these models appear also as an application in explicitly computational

contexts [36]. We see such phenomenon of overlapping of different approaches as a very important in the sake of building information systems like artificial neural networks.

In this paper some of the most useful models of spiking neurons have been proposed and compared from two perspectives - computational efficiency and evolvabilty. As our goal is to construct a large-scale complex network of spiking neurons using evolutionary computation techniques, we need a neuron model suitable for this kind of approach. From that point of view, computational efficiency and evolvability of given neuron model are its crucial properties. So which one to choose?

We argue that the neuron model proposed by Izhikevich [15] is the most suitable to fill our expectations, even though, the model was developed to understand the fine temporal structure of cortical spike trains, and to use spike-timing as an additional variable to understand how the mammalian neocortex processes information [16], i.e., it has not been used outside the biology yet. So what are the arguments for using the model as a processing element in an evolution driven complex network?

First: Izhikevich's model has the same implementation cost as the Integrate-and-Fire-or-Burst spiking neuron model (Fig. 2), and therefore it is as good as some of the I&F neurons from the computational efficiency point of view. Indeed, the model have been used to simulate a sparse network of 10.000 spiking cortical neurons with 1.000.000 synaptic connections in real time using a desktop PC and C++ programming language [15].
Second: as it has only four dimensionless parameters and it can reproduce all of the important neurocomputational features (see Fig. 4), we can conclude that the model has the best evolvabilty of all persented spiking neuron models considering both aspects - phenotype-genotype compression together with evolutionary potential.

These attributes of the model allows for the process of evolution to "experiment" with many types of model's behavior because there are plenty of them and the transition of neuron dynamics is manageable by only four parameters. In favor of this statement speaks the fact that author (in [16]) failed to find the parameters (within a reasonable period of time) for some of the neurocomputational properties for all of the Hodgkin-Huxley-type models, not surprisingly, the models with the worst phenotype-genotype compression.

In conclusion, having a "good-looking" efficient and evolvable spiking neuron model is only a beginning of the story of creating complex large-scale neural networks for information processing. There are still many problems to be solved but the main idea of this paper is that our most prominent tools for building complex systems should be the principles of emergence and self-organization.

References

[1] K.J. Cios, W. Swiercz, and W. Jackson, Networks of spiking neurons in modeling and problem solving. Neurocomputing, 61, 99-119, 2004.

[2] R. Dawkins, The evolution of evolvability. In C.G. Langton, editor, Artificial life, the proceedings of an Interdisciplinary Workshop on the Synthesis and Simulation of Living Systems. Addison-Wesley, Redwood City, CA, 1989.

[3] J.C. Eccles, Evolution of the Brain: Creation of the Self, Routledge, 1989, Reprint edition, 1991.

[4] G.M. Edelman, Neural Darwinism: Selection and reentrant signaling in higher brain function. Neuron, 10, 115-125, 1993.

[5] D. Ferstner and N. Spruston, Cracking the neuronal code. Science, 270, 756-757, 1995.

[6] R. FitzHugh, Impulses and physiological states in models of nerve membrane. Biophys. J., 1, 445-466, 1961.

[7] M.S. Gazzaniga, Nature's Mind. New York, Basic Books. 1992.

[8] W. Gerstner and C.M. Bishop, (eds.), Spiking Neurons in Maass, Pulsed Neural Networks. MIT-press, 1999.

[9] W. Gerstner and W.M. Kistler, Spiking neuronmodels: Single neurons, populations,plasticity. Cambridge, Cambridge University Press, 2002.

[10] B.G. Hall and H.S. Malik, Determining the evolutionary potential of a gene. Mol. Biol. Evol., 15(8), 1055-1061, 1998.

[11] D. Hansel, G. Mato, C. Meunier, and L. Neltner, On numerical simulations of integrate-and-fire neural networks. Neural Comput., 10, 467-483, 1998.

[12] A. Hodgkin and A. Huxley, A quantitative description of membrane current and its application to conduction and excitation in nerve. J. Physiol., 117, 500-544, 1952.

[13] E.Y. Chang, K.F. Morris, R. Shannon, and B.G. Lindsey, Repeated sequences of interspike intervals in baroresponsive respiratory related neuronal assemblies of the cat brain stem. J. Neurophysiol., 84, 1136-1148, 2000.

[14] J.P. Changeux, L'homme neuronal (Neuronal Man: The Biology of Mind, 1985), Paris, Hachette Litterature (1998).

[15] E.M. Izhikevich, Simple model of spiking neurons. IEEE Transactions on Neural Networks, 14, 1569-1572, 2003.

[16] E.M. Izhikevich, Which model to use for cortical spiking neurons? IEEE Trans. on Neural Networks, 15, 1063-1070, 2004.

[17] E.M. Izhikevich and G.M. Edelman, Large-scale model of mammalian thalamocortical systems. Proc. Natl. Acad. Sci. USA, 105, 3593-3598, 2008.

[18] E.M. Izhikevich and R. FitzHugh, FitzHugh-Nagumo Model. Scholarpedia, 1(9), 1349, 2006.

[19] M. Kirschner and J. Gerhart, Evolvability. PNAS, 95(15), 8420-8427, 1998.

[20] P.E. Latham, B.J. Richmond, P.G. Nelson, and S. Nirenberg, Intrinsic dynamics in neuronal networks. I. Theory. J. Neurophysiol., 83, 808-827, 2000.

[21] V. Litvak, H. Sompolinsky, I. Segev, and M. Abeles, On the transmission of rate code in long feedforward networks with excitatory-inhibitory balance. J. Neurosci., 23, 3006-3015, 2003.

[22] W. Maass, The Third Generation of Neural Network Models. Technische Universitat Graz, 1997.

[23] H. Markram, M. Toledo-Rodriguez, Y. Wang, A. Gupta, G. Silberberg, and C. Wu, Interneurons of the neocortical inhibitory system. Nat. Rev. Neurosci., 5, 793-807, 2004.

[24] M.E. Mazurek and M.N. Shadlen, Limits to the temporal fidelity of cortical spike rate signals. Nat. Neurosci., 5, 463-471, 2002.

[25] C. Morris and H. Lecar, Voltage oscillations in the barnacle giant muscle fiber. Biophys. J., 35, 193-213, 1981.

[26] A. Morrison, S. Straube, H.E. Plesser, and M. Diesmann, M., Exact subthreshold integration with continuous spike times in discrete time neural network simulations. Neural Comput., 19 , 47-79, 2007.

[27] C.L. Nehaniv, Evolvability. BioSystems, 69, 77-81, 2003.

[28] H.E. Plesser and M. Diesmann, Simplicity and efficiency of integrate-and-fire neuron models. Neural Comput., 21, 353-359, 2009.

[29] Y. Prut, E. Vaadia, H. Bergman, I. Haalman, H. Slovin, and M. Abeles, Spatiotemporal structure of cortical activity: Properties and behavioral relevance. J. Neurophysiol., 79, 2857-2874, 1998.

[30] R.A. Raff, The shape of life: Genes, development, and the evolution of animal form. Chicago, The University of Chicago Press, 1996.

[31] P. Reinagel and R.C. Reid, Precise firing events are conserved across neurons. J. Neurosci., 22, 6837-6841, 2002.

[32] R.M. Rose and J.L. Hindmarsh, The assembly of ionic currents in a thalamic neuron. I The three-dimensional model. Proc. R. Soc. Lond. B, 237, 267-288.

[33] M.J. Shelley and L. Tao, Efficient and accurate time-stepping schemes for integrate-and-fire neuronal networks. J Comput. Neurosci., 11, 111-119, 2001.

[34] G.D. Smith, C.L. Cox, S.M. Sherman, and J. Rinzel, Fourier analysis of sinusoidally driven thalamocortical relay neurons and a minimal integrate-and-fire-or-burst model. J. Neurophysiol., 83, 588-610, 2000.

[35] W. Swiercz, K. Cios, K. Staley, L. Kurgan, F. Accurso, and S. Sagel, A new synaptic plasticity rule for networks of spiking neurons. IEEE Trans. on Neural Networks, 17(1), 94-105, 2006.

[36] D. Tal and E.L. Schwartz, Computing with the Leaky Integrate-and-Fire Neuron: Logarithmic Computation and Multiplication. Neural Comput., 9(2), 305-318, 1997.

[37] A. Wagner, Robustness, evolvability and neutrality. FEBS, 579, 1772-1778, 2005.