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A Connectionist Model based on Physiological Properties of the Neuron

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Abstract

Recent research on artificial neural network models regards as relevant a new characteristic called biological plausibility or realism. Nevertheless, there is no agreement about this new feature, so some researchers develop their own visions. Two of these are highlighted here: the first is related directly to the cerebral cortex biological structure, and the second focuses the neural features and the signaling between neurons. The proposed model departs from the previous existing system models, adopting the standpoint that a biologically plausible artificial neural network aims to create a more faithful model concerning the biological structure, properties, and functionalities of the cerebral cortex, not disregarding its computational efficiency.

1 Introduction

Classical connectionist models are based on the pioneering McCulloch-Pitts neuron proposal [9] although their operations are far from the way human brain works. Artificial Neural Networks (ANN) nowadays are designed to mimic, with limited realism, some brain functionalities including pattern recognition, new information acquisition, and motor performance [7, 3].

More recently, some researchers of this area have approached the proposition of new models based on cerebral structure, that is, on cerebral cortex biological properties. This new direction on research makes noticeable a new characteristic to be incorporated to ANNs: the biological plausibility.

However there is no agreement about the definition of this new concept. For this reason, biological plausibility is still being analyzed under several aspects. One of these main visions concerns the cerebral cortex structure.

Taken the ideas presented previously into consideration, this paper proposes a new artificial neuron model based on biological properties of the physiological neuron.

2 Intraneuron Signaling

2.1 The biological neuron and the intraneuron signaling

The intraneuron signaling is based on the principle of dynamic polarization, proposed by Ramón y Cajal, which establishes that "electric signals inside a nervous cell flow only in a direction: from neuron reception (often the dendrites and cell body) to the axon trigger zone" [5].

Based on physiological evidences of principle of dynamic polarization the signaling inside the neuron is performed by four basic elements: *Receptive*, responsible for input signals; *Trigger*, responsible for neuron activation threshold; *Signaling*, responsible for conducting and keeping the signal; and *Secretor*, responsible for signal releasing to another related neuron.

These elements are related to the dendritic region, cell body, axon, and presynaptic terminals of the biological neuron, respectively [5].

The cell body is the neuron portion where cell genes are and is the place where protein syntheses occur. Considered the neural cell metabolic center, the cell body is the main control place of ordinary neural electric signals. From the cell body - or soma - originate two signal conducting branches, the axon and the dendrites.

The main function of a dendrite is the reception of signals originated from other cells. The axon, in general, comes from a cell body area called axon hillock [5], located on the opposite side of the tree trunk formed by dendrites, and ended in a micro-ramification form called axonal tree or axonal branch. These branches have in their terminals synaptic buttons, responsible for connectivity between neurons.

The axon is considered the main neuron conductivity unit [5], responsible for the transmission of electric signals (action potentials or spikes) from the axonal cone - area situated at the end of cell body - to the presynaptic terminals, keeping the same amplitude and duration, varying only in intensity or frequency.

The transmission rules of neuron output signaling are limited, but very efficient. This efficiency is verified through fast propagation and signal properties maintenance between initial and final points of involved axons [5, 6].

There are other elements, co-responsible for propagation speed and amplitude maintenance of action potentials, for example, the glial cell and the Ranvier nodules. The glial cell involves the neural cell bodies and their axons in a process called myelination, which provides as an insulating final product, the myelin sheaths, white fatty extensions of neuroglial cells (or Schwann cells), involving the axon in several layers. These sheaths are interrupted at regular intervals by the nodes of Ranvier, which are electrically non-insulated and are responsible for the action potential regeneration [5, 6].

There is also the ionic mechanism, which can act as a signaling structure in an excitable cell, independently of synapse localization. This mechanism refers to the current flow generated by the ionic movement through protein membranes - channels - generating the signals (membrane potentials) in neural cells. These alterations in resting potentials of the cell membrane provoked by the ionic current flowing through open channels produce input signals that develop the intraneuron signaling process.

The intraneuron signaling process begins after neuron binding, either directly or by means of modulation (see item about interneuron signaling), altering the membrane potential and transforming chemical potential energy in an electric signal called synaptic potential. The synaptic potential is graded and its amplitude is directly related to volume and releasing time of chemical neurotransmitters in interneuron signaling process. Once generated, the input signal - synaptic potential - often flows through dendrites and cell body where it is accumulated until reaching a certain threshold, responsible for pulse or action potential generation. This input signal can be excitatory or inhibitory, so the latter tries to prevent action potential from releasing.

The over stimulus of the target cell makes the inputs compete. In the "struggle," the weaker synapses are eliminated and the stronger synapses keep the inputs to the postsynaptic cell [5]. These competitive inputs are combined into the postsynaptic neuron through a process called neuronal integration, which sum up all the signals received by a neuron and takes the decision of firing or not an action potential, which reflects the brain fundamental ability of decision-making in the neuron [5, 11]. In neuronal integration process, the brain ability in opting for one of the competitive alternatives (fire or not fire), selecting one and suppressing the other, is known as the integrative action of the nervous system [5].

Action potentials are electric signals used for manipulating brain information - reception, analysis, and transmission. They are very fast and transient, of the kind all-or-none [3, 5], originating in a specific trigger zone - the beginning of the axon. The fact that the action potential, the neuron conducting signal, is an "all-or-none" signal or binary is motivated by the fact that a stimulus smaller than threshold does not produce signals and a stimulus greater than threshold produces the same signal independently of amplitude variation and stimulus duration. Consequently, there are only two pulse characteristics for the information transmission process to be taken into consideration: the amount of action potentials and the time intervals between them [5, 12, 4].

The action potential is generated only if the input signal, or the weighted sum of the input signals by their synaptic weights, is greater than a certain activation threshold, that is, once the threshold is reached, the additional increase in amplitude of the input signal implies in an increase of the action potential frequency and not in an increase of the potential amplitude [5]. Since action potentials are of the kind "all-or-none" and conducted without alterations through the whole axon, the information contained in the signal is represented only by the frequency and pulse amount, without taking into account their amplitude. So, the larger the stimulus duration, the longer the action potential sequence lasts, and consequently, the larger the amount of pulses [5].

Once the synaptic terminals are reached, action potentials stimulate chemical neurotransmitter releasing by the cell, considered as analogical output signals. In this graded transmitter releasing, there is, repeatedly, the transformation of digital signal, represented by action potential, in analogical signal. Also in this process, the exact amount of neurotransmitters to be released by the cell is defined by the total number of action potentials in a given time interval [5]. After an action potential releasing, a very short refractoriness time period happens, that is, a period of lesser excitability. This time period - called refractory period - can be divided in: (1) *absolute*, when happens immediately after the action potential; and (2) *relative*, which is subsequent to the absolute refractory period.

In the absolute refractory period, it is impossible to excite a cell; the same does not happen in the relative period, when a pulse can be released [5, 11].

2.2 The artificial neuron and the intraneuron signaling

The neuron is considered the fundamental operational unit of a neural network - natural or artificial -, due to its capacity of processing and transmitting information [5, 12, 4].

In artificial neural network primordial research, McCulloch and Pitts [9] define a neuron model that served as basis for building several artificial neurons (for instance, Rosenblatt's perceptron [3]).

McCulloch-Pitts model [9] is based on an "all-or-none"

property of a pulse releasing for a specific neuron. The basic idea to represent the "all-or-none" property proposed by the two researchers consists in dividing the refractory period in time units so that, at most, one pulse could be generated by the neuron in each interval. Besides, the model output is binary, that is, it is 0 (zero) or 1 (one) according to the value of the induced local field or neuron activation potential. McCulloch and Pitts [9] modeled, indeed, an artificial neuron as a time-independent binary element.

Maass [7] proposes a classification based on model generations to situate his proposition in a biological realism evolutionary scale. Maass [7] suggests three generations:

1. The *first generation* artificial model is based on McCulloch-Pitts neuron, and its main feature is the ability to produce only digital outputs - zero or one;

2. The *second generation* model is based on the application of the activation function as a computational unit, which produces a continuous set of output values for a weighted (or polynomial) sum of inputs; and

3. The *third generation* model uses the duration time of an action potential to encode information, that is, uses time as a computational and interneuron communication resource.

Maass's integrate and fire neuron (spiking neuron) model is argued to be a third generation model [7]. That is, Maass [7, 8] employs the spike transmission time, the refractory period, and the membrane electric potential in the pulse trigger zone as model's main elements.

The model proposed by Rosa [13] considers the classic neuron model basic elements like connection weights, activation threshold, and activation function. In addition, Rosa [13] introduces three more elements that act directly in intraneuron and interneuron signaling. These elements are the transmitters T, the receptors R, and the controllers C, representing the amount of substrate, the binding affinity between transmitters and receptors, and the genes - name and expression. However, Rosa [13] does not consider some intraneuron signaling features, as for instance, the association of the pulse frequency with the amount of substrate released by transmitter T.

3 Interneuron signaling

3.1 The biological neuron and the interneuron signaling

The interneuron signaling occurs by means of electrical or chemical synapse, which have completely different morphologies. At electrical synapses, the transmission occurs through special ionic channels called gap junction channels, which are located in the pre and postsynaptic cell membranes and serve as a cytoplasmatic connection between the two cells. At chemical synapses, there is a small cellular separation between the cells called synaptic cleft, instead of cytoplasmatic continuity.

At electrical synapses, part of electric current injected in presynaptic cell escapes through the resting channels and the remaining current is driven to the inside of the postsynaptic cell through the gap junction channels, which connect cytoplasms of involved cells.

At chemical synapses, in the presynaptic terminal specialized zones, there are vesicles containing neurotransmitter molecules. When the action potential reaches these synaptic vesicles, the neurotransmitters are released to the synaptic cleft, and the interneuron signaling chemical process, or chemical synapse, takes place.

These vesicles, when stimulated, flow towards the plasmatic membrane, with which are melted, releasing neurotransmitters to the synaptic cleft. The amount of neurotransmitters released is directly related to the depolarization amplitude inside presynaptic terminals and to the frequency of the action potentials that cross the axon [5, 6].

The releasing of chemical neurotransmitters towards the synaptic cleft operates as an output signal. After releasing, the neurotransmitters spread along the synaptic cleft in order to reach the receptors of the postsynaptic cell membrane of another neuron. The neurotransmitter binding at the postsynaptic cell generates a synaptic potential which produces excitatory or inhibitory synapse, depending on the receptor type. In other words, receptors are the elements which define the type of synapse and the occurrence of modulation [5, 13]. The fact that the receptors define the type of synapse agrees with Ramón y Cajal's principle of connectional specificity [5].

In some cases, neurotransmitters are enzymatically divided, synthesizing new transmitters, therefore increasing the amount of substrate in the synaptic transmission process [13].

The chemical neurotransmitters are classified based on their control over the ionic channels of the postsynaptic cell, acting directly or indirectly through direct or indirect chemical synaptic actions, respectively. In direct way, the receptors responsible for ionic channels control belonging to its structure, recognize the neurotransmitter and through its binding, open these channels. In indirect way, the receptor does not take part effectively of the ionic channel structure, but there is an activation through a fixing protein - called G protein - which is consequence of the binding between transmitter and receptor. This protein activates a set of second messengers which modulates transmission at ionic channel.

The direct interference of a receptor over ionic channels triggers chemical synaptic actions, very fast in general,

which often takes part in behavior, in direct way. In indirect interference, the synaptic actions which serve also to modulate behavior are slower, but alter neuron excitability and synaptic connection strengths of neural circuits.

The modulation process in chemical synaptic actions occurs also through peptides that can act as neurotransmitters, but in a broader way - in relation to actuation area - and longer-lasting - in relation to action time. These peptides are characterized by acting in restricted areas, showing low conductance, without sustaining high frequency impulses, showing persistency and small excitatory effects, and not producing enough depolarization to excite a cell by itself [13]. In the case of depolarization, although insufficient, the peptide transmission can provoke a fast excitatory effect by means of a second input, also excitatory.

Mutation happens through modification of gene expression, and it is another factor associated with synaptic modulation process. Mutation is often due to the second messenger intense action in changes of binding affinities between transmitters and receptors.

3.2 The artificial neuron and the interneuron signaling

Maass [7] defines his model taking into account the biological neuron output and the frequency of individual action potentials as the mechanism for information representation.

Rosa [13], otherwise, takes into consideration Ramón y Cajal's connective specificity principle, shown previously. That is, Rosa [13] proposes the inclusion of three new variables - Transmitter (T), Receptor (R), and Controller (C) - in McCulloch-Pitts classical model [9], responsible for the affinity control between neurons and the way signal transmission occurs at chemical synapse. The new elements, according to Rosa [13], are absent in conventional models.

4 The biologically plausible artificial neuron proposal

There is no agreement about the characteristics that the proposal of a biologically more realistic artificial neuron model can be based on, but there are some fundamental principles, which provide a basis for such a proposal.

Focusing on neural signaling, there are two large groups of characteristics that must be taken into consideration: the intraneuron signaling and the interneuron signaling. This proposal considers both and is based mainly on Maass' [7] neuron formalism and on Rosa's [13] model functionalities, because they present most of the basic principles for biological plausibility characteristics. Merging Maass [7] and Rosa [13] models and eliminating the redundancies, the proposed model can be defined as:

$$N_{vr} = \{\ P, W, \Theta, \tau, \eta, \xi, C\ \}$$

where each basic element can be described as:

- P represents the membrane potential in neuron trigger zone - the place in neuron soma where the pulses are initiated,
- W represents the connection weight set,
- Θ represents the activation threshold,
- τ represents the pulse transmission time between involved neuron somas,
- η represents the time the neuron is kept inert between pulses,
- ξ represents the response function that defines the type of synapse (excitatory or inhibitory), and
- C represents the controller set.

In this proposal, the controller is considered an external element for the artificial neuron composition, because it acts at the interface of chemical synaptic transmission process. The controller is an element set that represents the binding affinity degree, the amount of substrate, and the modification of gene expression in modulation. Here, the elements P, W, Θ, τ, η, and ξ act on information transmission and on binding affinity control due to direct synaptic action. The element set C acts on binding affinity control as a result of the modulation effect, on the amount of substrate control due the action of acetylcholine or other transmitters that act in a similar way, and on the modification of gene expression as a consequence of the second messenger action.

Here, the artificial neural network is a finite set of spiking-response control neurons N_{vr}, defined as:

- A set of synapses $S \subseteq N_{vr} \times N_{vr}$,
- A synaptic weight $w \geq 0$ for each pre(*nu*) and post(*nv*) synaptic neuron binding,
- A response-function $\xi_{nu,nv} : R^+ \rightarrow R$ for each synapse between the pre(*nu*) and post(*nv*) synaptic neuron <*nu*, *nv*> $\in S$, where $R^+ = \{x \in R : x \geq 0\}$,
- A threshold function $\Theta_{nv} : R^+ \rightarrow R^+$ for each presynaptic neuron $nv \in N_{vr}$ [7],
- A set of controllers $C \subseteq N_{vr} \times N_{vr}$,
- A label or number for the origin and target genes (name),
- A modulatory synaptic function $m_{nu,nv} \in \{\gamma, \varphi, \psi\}$, where γ, φ, and ψ represent a gene expression, a binding affinity degree, and a substrate variation, respectively, and

- A control function $\chi_{nu,nv} : R^+ \rightarrow R^+, \forall m_{nu,nv}$ [13].

So, if a firing time set *s* of presynaptic neuron *nu* is defined by $F_{nu} \subseteq R^+$, then the trigger zone potential of postsynaptic neuron *nv* in time *t* is defined by [7]:

$$P_{nv}(t) = \sum_{nu:<nu,nv>\in S} \sum_{s\in F_{nu}:s<t} w_{nu,nv} \cdot \xi_{nu,nv}(t-s)$$

so the neuron *nv* fires in time *t* when $P_{nv}(t)$ reaches $\Theta(t-t')$, where *t'* is the time of the more recent *nv* firing.

In addition to the modulatory synaptic function $m_{nu,nv}$, there is the control function $\chi_{nu,nv}$ as responsible for the representation of the type of synaptic function modulation. This way, it is possible to define the formalism of controller C, by means of control function $\chi_{nu,nv} : R^+ \rightarrow R^+$, $\forall m_{nu,nv}$, in the following way:

$$\chi_{nu,nv} = \gamma_{nv} \cdot \varphi_{nv} \cdot \psi_{nu} \cdot \rho_{nu,nv}$$

where

- γ_{nv} represents the new controller of gene expression (at target cell),
- φ_{nv} represents the new binding affinity degree of receptor (at target cell),
- ψ_{nu} represents the increasing of the amount of substrate (at origin cell), and
- $\rho_{nu,nv}$ represents the type of postsynaptic potential (excitatory or inhibitory) in relation to the type of transmitter and receptor, by means of direct action.

The negative signal of the inhibitory synapse is defined by the response function $\xi_{nu,nv}(t-s)$ of the postsynaptic neuron, through control element $\rho_{nu,nv}$, since the biological synapse does not change signals during learning process. For this reason, the synaptic weights have only positive or zero values.

The response function or postsynaptic potential is given by [2]:

$$\xi_{nu,nv}(t) = \frac{1}{1-(\tau_s/\tau_m)}(exp(-\frac{t-\delta}{\tau_m}) - exp(-\frac{t-\delta}{\tau_s})) \cdot \theta(t) \cdot \chi_{nu,nv}$$

where

- τ_s represents the synapse time constant,
- τ_m represents the membrane time constant,
- t represents the current time,
- δ represents the delay time constant in axonal transmission,
- $\theta(t)$ represents the heaviside function [2], which is the transformation of the differential equation $\xi_{nu,nv}$ with initial conditions in an algebraic equation, in order to obtain a solution of these conditions in an indirect way, without calculating the general solution of $\xi_{nu,nv}$ by means of integrals and derivatives, and
- $\chi_{nu,nv}$ represents the control function of postsynaptic potential.

The function for the refractory period (calculation of negative contribuition) is given by [2]:

$$\eta(t) = -\Theta \cdot exp(\frac{t}{\tau}) \cdot \theta(t) \cdot \chi_{nu,nv}$$

where

- Θ represents pulse (spike) firing threshold,
- t represents the current time,
- τ represents the time constant used to calculate the neural refractoriness,
- $\theta(t)$ represents the heaviside function [2] too, but now applied to the function $\eta(t)$, and
- $\chi_{nu,nv}$ represents the control function.

The control function $\chi_{nu,nv}$ is given only by gene expression of the target cell, because there is no pulse and no refractory period without gene expression affinities between the two involved signaling neurons.

Finally, these formalisms aim to guarantee the new proposed model adherence to the biological neural signaling basic principles. This way, it adds new biological plausibility characteristics to Maass' [7] and Rosa's [13] models.

5 Simulation

The behavioral comparative analysis of the proposed model is based on three variations of learning models and algorithms: Multilayer Perceptron with Back-propagation (MLP+BackProp); Multilayer Perceptron with GeneRec (MLP+GeneRec); and Spiking Response Model with GeneRec (SRM+GeneRec).

For the proposed model, the learning algorithm GeneRec, proposed by O'Reilly [10, 11], was employed. The reason for this choice relies on its presumable biological plausibility. Instead Back-propagation, considered biologically implausible [1], GeneRec is argued to be more biologically plausible: learning happens through synaptic weight modifications using only the local information available in synapses.

In simulation, the features of employed networks for comparison and of the proposed model are: the input, hidden, and output layers for the three models - MLP+BackProp, MLP+GeneRec, and SRM+GeneRec - are built by eighty, ten, and ten neurons respectively, and the weights had their values assigned randomly. Specifically, the values of the structural elements of the SRM base model are assigned randomly in defined range for τ_m between 40 and 2,000 ms, τ_s between 20 and 1,000 ms, θ between 0.1 and 0.8 mV, τ between 2,000 and 8,000, δ between 20 and 200 ms. The same happens with the structural elements of the proposed model, where randomly values were assigned to γ_{nv} between 0 and 1, ϕ_{nv} between 0.6 and 1.0, ψ_{nu} between 1 and the amount of neurons of the post-synaptic layer, and $\rho_{nu,nv}$ receives value 3 or 5.

In learning process, 100 patterns were presented, divided in 10 variations for each value. These patterns were presented to the network in an alternate way to avoid recognizing problems, through 101 iterations and learning rate of 0.2.

In recognizing process, 10 patterns with varied complexity were presented, and 90% of the patterns was recognized effectively. The unrecognized pattern is the same for every network, and this result was expected because this pattern resembles a pattern between digits 3 and 9 (see figure 1).

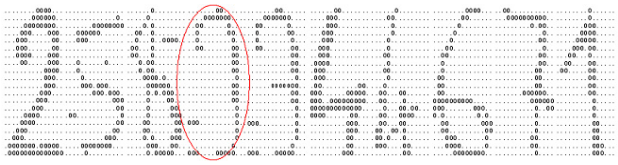

Figure 1. Detail of the unrecognized pattern presented to the involved networks.

About the unrecognized pattern, a 10-fold cross validation technique was applied, employing the training patterns that represent the digit nine. The test considered a total of ten correct patterns, a total of a hundred selected patterns, and six correctly selected patterns for the MLP+GeneRec and MLP+BackProp models and eight correctly selected patterns for the SRM+GeneRec model. From the results shown on table 1 it is possible to notice that learning was more effective in the SRM + GeneRec model.

Table 1. Performance of the models (in %).

Model	*Recall*	*Precision*	*F-Measure*
MLP+GeneRec	43.75	41.18	42.42
SRM+GeneRec	50.00	47.37	48.65
MLP+BackProp	40.00	37.50	38.71

6 Conclusion

The proposed model considers all the functionalities discussed earlier, and, specially, tries to merge Maass' [7] and Rosa's [13] neuron model elements, adopting a new format for the element controller. This model aims to consider a better modulation process, regarding the amount of substrate, the binding affinity degree, and the modification of gene expression. This way, the proposed model can add variety to the models considered biologically plausible.

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