An Artificial Neural Network Model Based on Neuroscience: Looking Closely at the Brain

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"Almost all aspects of life are engineered at the molecular level, and without understanding molecules we can only have a sketchy understanding of life itself."
Francis Crick, *What Mad Pursuit*, 1988

Abstract
Classical connectionist models [3, 8, 11] are based upon a simple description of the neuron taking into account the presence of pre-synaptic cells and their synaptic potentials, the activation threshold, and the propagation of an action potential. Certainly, this is an impoverished explanation of human brain characteristics [1, 9, 12]. In this paper, a mechanism to generate a biologically plausible artificial neural network model is presented [10], which is taken to be closer to some of the human brain features. In such a mechanism, the classical framework is redesigned in order to encompass not only the "traditional" features but also labels that model the binding affinities between transmitters and receptors. This is accomplished by a restricted data set, which explains the neural network behavior. In addition to feed-forward networks, the present model also contemplates recurrence in its architecture, which allows the system to have re-entrant connections [2].

1 Introduction

The presented paper [10] departs from a classical connectionist model and proposes a biologically plausible neural network model. Such model is defined by a restricted data set, which explains the neural network behavior. Unlike other models, this one introduces transmitter, receptor, and controller variables in order to account for the binding affinities between neurons. The following feature set thus defines the neurons:

$$N = \{\{w\}, \theta, g, T, R, C\}$$

where w represents the connection weights, θ is the neuron activation threshold, g stands for the activation function, T symbolizes the transmitter, R the receptor, and C the controller. θ, g, T, R, and C are part of the genetic information. T, R, and C are the labels, absent in other models.

As stated in Ramón y Cajal's *principle of connectional specificity*, "nerve cells do not communicate indiscriminately with one another" [6]. In the presented model, each neuron is connected to another neuron not only in relation to its connection weight, activation threshold, and activation function, but also in relation to its labels. Neuron i is only connected to neuron j if there is binding affinity between the transmitter of i and the receptor of j. Binding affinity means compatible types, enough amount of substrate, and compatible genes.

In addition, the coupling result of a transmitter T with a receptor R generates a controller C, which can act over other neuron connections.

2 The Biological Support

The ordinary biological neuron has many dendrites usually branched, which receive information from other neurons, and an axon which transmits the processed information, usually by propagation of an action potential [1, 5]. The axon is divided into several branches, which make synapses onto the dendrites and cell bodies of other neurons. The nervous cells influence others by (a) excitation, that is, they contribute to produce impulses on other cells, and (b) inhibition, that is, they prevent the releasing of impulses on other cells.

The predominant type of synapse in the mammalian brain is chemical, and operates through the releasing of a transmitter substance from the pre-synaptic to the post-synaptic terminal [5-7]. This release occurs in active zones, inside pre-synaptic terminals. Certain chemical synapses lack active zones, so synaptic actions between these cells are slower and more diffuse. The coupling result of a neurotransmitter with a receptor makes the post-synaptic cell releases a protein.

The synaptic contacts can be morphologically classified in two basic types: type I and type II synapses [1, 6]. Type I synapses seem to be excitatory because they have larger membrane thickness on the post-synaptic side, and the pre-synaptic process has rounded synaptic vesicles, presumably containing packets of neurotransmitter. Type II synapses seem to be inhibitory because they have smaller and flattened

synaptic vesicles and the contact zone is usually smaller than that of type I synapses.

This picture, however, can be much more complicated than implied above. In the first place, the action of a transmitter in the post-synaptic cell does not depend on the chemical nature of the neurotransmitter, but instead on the properties of the receptors with which the transmitter binds. In some cases, it is the receptor that determines whether a synapse is excitatory or inhibitory, and whether an ion channel will be activated directly by the transmitter or indirectly through a second messenger [5, 6].

Secondly, instead of propagating an action potential, an axon can produce a graded potential [1]. Because of attenuation, one should expect that this form of information signaling does not occur over long distances. These graded potentials can occur in another level. For instance, an axon terminal that makes synapse in a given cell can receive a synapse. The pre-synaptic synapse can produce only a local potential change, which is then restricted to that axon terminal.

In view of these biological facts, it was decided to model two features. On the one hand, the binding affinities between transmitters and receptors were modeled through labels T and R. On the other hand, the role of the "second messenger," the effects of graded potential, and the protein released by the coupling of transmitter and receptor were all modeled under only one label, the controller C.

3 The Roles of the Controller

Within the model, the controller can modify the binding affinities between neurons, through three main functions. Firstly, it can modify the degrees of affinity of receptors. Secondly, it can modify the amount of substrate (that is, the amount of transmitters and receptors). Finally, it can modify the gene expression, in the case of mutation. Let's consider the biological motivation for each of these functions in detail.

Degrees of affinity, at chemical synapses, are related to the way receptors gate ion channels, through which transmitter material enters the post-synaptic cell: in direct gating, receptors produce relatively fast synaptic actions, while in indirect gating, receptors produce slow synaptic actions. These slower actions often serve to *modulate* behavior [5] because they modify the degrees of affinity of receptors.

In addition, modulation can be related to the action of peptides. There are many distinct peptides, of several types and shapes, that can act as neurotransmitters [4]. There are, however, reasons to suspect that peptides are different from many conventional transmitters [1]: peptides appear to "modulate" the synaptic function instead of activating it; the action of peptides usually appears to spread slowly and persist for some time, much more than conventional transmitters; and in some cases, peptides do not act where they were released, but at some distant site.

As transmitters, peptides act at very restrict places, display a slow rate of conduction, and do not sustain the high frequencies of impulses. As neuromodulators of the synaptic function, its activity is more intense. The excitatory effects of substance P (a peptide) are very slow in the beginning but longer in duration (more than one minute) and cannot cause, per se, enough depolarization to excite the cells. The effect, however, is to make neurons more readily excited by other excitatory inputs – a clear example of "neuromodulation". Controllers, in the model presented, explain this function by modifying the degrees of affinity of receptors.

An additional function of the controller is to account for variation in the amount of substrate. In biological systems, the acetylcholine (a neurotransmitter) is spread over a short distance toward the post-synaptic membrane and acts at the specific receptor molecules in that membrane. Then, the acetylcholine is enzymatically divided and part of it is taken up again for synthesis of a new transmitter, causing an increase in the amount of substrate. In this model, the controller represents substrate increase by a variable acting over the initial substrate amount.

The final function of the controller concerns gene expression. It was shown that peptides are a second, slower, means of communication between neurons – which is more economical than using extra neurons for this purpose. This second messenger, besides altering the affinities between transmitters and receptors, can regulate gene expression thereby endowing synaptic transmission with long-lasting consequences [5]. In the model, this is achieved by the modification of the variable that represents gene expression. Consequently, mutation can be accounted for in this model.

4 The Labels and Their Dynamic Behaviors

The aim of this paper is to present a more sophisticated mathematical model of the neuron, through the definition of a restrict data set, thus explaining the behavior of a biologically plausible artificial neural network. In this sense, it is important to define the labels (T, R, and C) and their dynamic behaviors in the following way, as stated in [10]:

A. For the network genesis:

1. the specification of the number of layers;
2. the specification of the number of neurons in each layer;
3. the definition of the initial amount of substrate (transmitters and receptors) in each layer; and
4. the definition of the genetics of each layer (type of transmitter and its degree of affinity, type of receptor and its degree of affinity, and genes (name and gene expression)).

B. For the evaluation of the controllers and how they act:

1. the controllers can modify the degree of affinity of receptors;
2. the controllers can modify the initial substrate storage; and
3. the controllers can modify the gene expression value (mutation).

It is expected that these specifications lead to an artificial neural network displaying some distinctive characteristics. In the first place, each neuron has a genetic code (a set of genes plus a gene expression controller). The controller can cause *mutation*, because it can regulate gene expression.

Second, the substrate (amount of transmitter and receptor) is defined by layer. Because substrate amounts are limited, there is a chance that some post-synaptic neurons, to which a certain pre-synaptic neuron should be connected, will not be activated. Such a network, then, can be seen as favoring *clustering*.

Third, the substrate increase is related to the gene specified in the controller, because the synthesis of a new transmitter occurs in the pre-synaptic terminal (origin gene). The modification of the genetic code, that is, mutation, as well as the modification of the degree of affinity of receptors, however, is related to the target gene. The reason is that the modulation function of controller is better explained at some distance of the emission of neurotransmitter, therefore at the target.

5 A Network Simulation

In table 1, a data set for a five-layer network simulation is presented. For the specifications displayed in table 1, the network architecture and its activated connections are shown in figure 1. For the sake of simplicity, all degrees of affinity are set at 1 (the degree of affinity is represented by a real number in the range [0..1]; so that the greater the degree of affinity is the stronger the synaptic connection will be).

In figure 1, one can notice that every unit in layer 1 (the input layer) is linked to the first nine units in layer 2 (first hidden layer). The reason why not every unit in layer 2 is connected to layer 1, although the receptor of layer 2 has the same type of the transmitter of layer 1, is that the amount of substrate in layer 1 is eight units. This means that, in principle, each layer-1 unit is able to connect to at most eight units. But controller 1, from layer 1 to 2, incremented by 1 the amount of substrate of the origin layer (layer 1). The result is that each layer 1 unit can link to nine units in layer 2. Observe that from layer 2 to layer 3 (the second hidden layer) only four layer-2 units are connected to layer 3, because also of the amount of substrate of layer 3, which is 4.

Table 1. The data set for a five-layer network

layer	*1*	*2*	*3*	*4*	*5*
number of neurons	10	10	5	5	1
amount of substrate	8	10	4	5	2
type of transmitter	1	2	1	2	1
degree of affinity of transmitter	1	1	1	1	1
type of receptor	2	1	2	1	2
degree of affinity of receptor	1	1	1	1	1
genes (name/gene expression)	abc/1	abc/1	abc/1 def/2	abc/1 def/2	def/ 2

Controllers: 1/1-2: abc/s/abc/1
1/1-4: abc/e/abc/2
2/2-3: abc/a/def/0.5

(Controller syntax: *number/origin layer-target layer*: *og/t/tg/res*, where *og* = origin gene (name); *t* = type of synaptic function modulation: a = degree of affinity, s = substrate, e = gene expression; *tg* = target gene (name); *res* = control result: for t = a $\rightarrow$ *res* = new degree of affinity of receptor (target), for t = s $\rightarrow$ *res* = substrate increasing (origin), for t = e $\rightarrow$ *res* = new gene expression controller (target). The controllers from layer 2 to 5, from layer 3 to 4, and from layer 4 to 5 are absent in this simulation.)

As a result of the compatibility of layer-2 transmitter and layer-5 receptor, and the existence of remaining unused substrate of layer 2, one could expect that the first two units in layer 2 should connect to the only unit in layer 5 (the output unit). However, this does not occur because their genes are not compatible. Although gene compatibility exists, in principle, between layers 1 and 4, their units do not connect to each other because there is no remaining substrate in layer 1 and because controller 1 between layers 1 and 4 modified the gene expression of layer 4, making them incompatible. The remaining controller has the effect of modifying the degrees of affinity of receptors in layer 3 (target). Consequently, the connections between layers

2 and 3 became weakened (represented by dotted lines). Notice that, in order to allow connections, in addition to the existence of enough amount of substrate, the genes and the types of transmitters and receptors of each layer must be compatible.

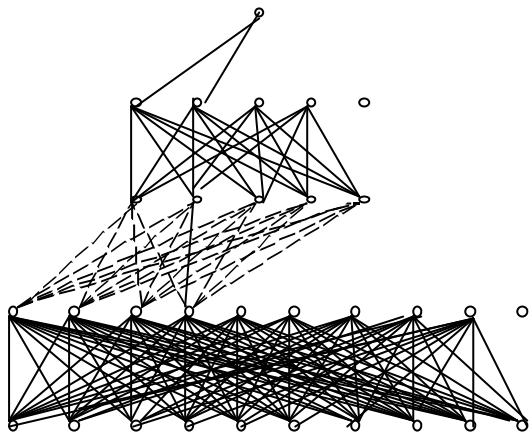

Fig. 1. A five-layer neural network for the data set in table 1. In the bottom of the figure is the layer 1 (input layer) and in the top is the layer 5 (output layer). Between them, there are three hidden layers (layers 2 to 4)

Although the architecture shown in figure 1 is feed-forward, recurrence, or re-entrance, is permitted in this model. This kind of feedback goes along with Edelman and Tononi's "dynamic core" notion [2]. This up-to-date hypothesis suggests that there are neuronal groups underlying conscious experience, the dynamic core, which is highly distributed and integrated through a network of reentrant connections.

6 Conclusion

Nowadays, models of artificial neural networks are in debt with human brain physiology. That is, for mathematical simplicity reasons mainly, conventional neural network models are too simple and thus lack several biological features of the cerebral cortex. The aim here is to present a biologically plausible artificial neural network model [10], which seeks to be closer to the human brain capacity, although only a few brain features are considered. In this model, the possibility of connections between neurons is related not only to synaptic weights, activation threshold, and activation function, but also to labels that embody the binding affinities between transmitters and receptors. This type of neural network would be closer to human evolutionary capacity, since it purports to be a genetically well-suited model of the brain. The recent hypothesis of the "dynamic core" [2] is also contemplated because this model allows reentrancy in its architecture connections.

Acknowledgements: I would like to thank Armando Freitas da Rocha, Márcio Luiz de Andrade Netto, and Edson Françozo (of Unicamp) for their extensive comments on previous drafts of this paper. In addition, I am in debt with ICANNGA 2001 anonymous reviewers for their valuable suggestions.

References

[1] Crick, F. and Asanuma, C.: Certain Aspects of the Anatomy and Physiology of the Cerebral Cortex, in J. L. McClelland and D. E. Rumelhart (eds.), Parallel Distributed Processing, Vol. 2, Cambridge, Massachusetts - London, England, The MIT Press, 1986.

[2] Edelman, G. M. and Tononi, G.: A Universe of Consciousness – How Matter Becomes Imagination, Basic Books, 2000.

[3] Haykin, S.: Neural Networks - A Comprehensive Foundation, 2nd ed. Prentice Hall, Upper Saddle River, New Jersey, 1999.

[4] Iversen, L. L.: Amino Acids and Peptides: Fast and Slow Chemical Signals in the Nervous System?, Proceedings of the Royal Society of London, B, 221 (1224), 22 may, pp. 245-260, (1984).

[5] Kandel, E. R., Schwartz, J. H., and Jessell, T. M. (eds.): Principles of Neural Science. 4th ed., McGraw-Hill, 2000.

[6] Kandel, E. R., Schwartz, J. H., and Jessell, T. M. (eds.): Essentials of Neural Science and Behavior. Appleton & Lange, Stamford, Connecticut, 1995.

[7] Kuffler, S. W., Nicholls, J. G., and Martin, A. R.: From Neuron to Brain - A Cellular Approach to the Function of the Nervous System, 2nd ed., Sinauer Associates Inc. Publ. Sunderland, Mass., 1984.

[8] McCulloch, W. S. and Pitts, W.: A Logical Calculus of the Ideas Immanent in Nervous Activity, Bulletin of Mathematical Biophysics, 5, pp. 115-137, (1943).

[9] Rocha, A. F.: Neural Nets - A Theory for Brains and Machines, Berlin, Heidelberg: Springer-Verlag, 1992.

[10] Rosa, J. L. G. and Françozo, E.: A Biologically Fine-grained Artificial Neural Network: Towards a Hybrid Model, in Proceedings of the IASTED Intl. Conference on Artificial Intelligence and Soft Computing. Cancun, Mexico. May 27-30, pp. 302-305, (1998).

[11] Rosenblatt, F.: The Perceptron: A Perceiving and Recognizing Automaton, Rep. 85-460-1, Proj. PARA, Comell Aeronautical Lab., Ithaca, New York, 1957.

[12] Rumelhart, D. E. and McClelland, J. L.: PDP Models and General Issues in Cognitive Science, in D. E. Rumelhart and J. L. McClelland (eds.), Parallel Distributed Processing, Vol. 1, Cambridge, Massachusetts - London, England, The MIT Press, 1986.