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Volumen 9. Número 2. Agosto 2015. DOI: 10.7714/cnps/9.2.204

Bio-connectionist model based in the thalamo-cortical circuit.

Modelo Bioconexionista basado en el circuito tálamo-cortical.

Modelo Bio-conexionista baseado no circuito tálamo-cortical.

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Resumen

Estudios recientes sobre la interacción del tálamo con el neocórtex alimentan positivamente la sospecha de que no sólo el neocórtex es el principal responsable de los procesos de aprendizaje, sino su intrínseca relación con el tálamo. La simulación computacional ofrece un soporte conveniente para complementar las hipótesis derivadas de estos nuevos resultados experimentales. En el presente trabajo argumentamos a favor de la necesidad de una disciplina intermedia entre la neurociencia cognitiva computacional y el conexionismo, a la cual denominamos bioconexionismo. Presentamos un modelo bioconexionista que simula las proyecciones entre el tálamo y el neocórtex, en particular en el área sensorial primaria. Una nueva acepción del término memoria resulta del diseño bioconexionista. Finalmente, en busca de una interpretación de los resultados, lo comparamos con el trabajo de un conexionista de la primera ola, W. K. Taylor, quien, como resultado de una profusa contribución a la tradición, publicó en 1964 los lineamientos para un modelo cuyos supuestos son semejantes al modelo aquí expuesto. En el plano neurocientífico, llamamos la atención sobre la relevancia del tálamo en su relación con el neocórtex y en su responsabilidad en los procesos cognitivos. En relación al conexionismo, presentamos un modelo bioconexionista del que se deriva una posible explicación a los datos experimentales recientes. Desde el punto de vista de la historia del conexionismo, intentamos mostrar por qué conviene que los trabajos de Wilfred Kenelm Taylor sean reconsiderados.

Palabras clave: Conexionismo; Neurociencia cognitiva computacional; Redes neurales; Tálamo-cortical; Estimulación sensorial; Percepción; Capa neocortical 6

Abstract

Recent studies on the interaction of the thalamus with the neocortex positively feed the intuition that not only the neocortex is primarily responsible for the learning process, but also its intrinsic relationship to the thalamus is. Computer simulation conveniently support the hypotheses derived from these new experimental results. In this paper we argue for the need for an intermediate discipline between the computational cognitive neuroscience and connectionism, which we call bio-connectionism. We present a bio-connectionist model that simulates projections between the thalamus and the neocortex - particularly in the primary sensory area. A new sense of the word memory results from this bio-connectionist design. Finally, through the search of an interpretation of the model results, we contrast it with the work of a first-wave connectionist: W. K. Taylor, who, as a result of profuse contribution to the tradition, published in 1964 guidelines for a model whose assumptions are similar to the model discussed herein. On the neuroscientific level, highlights the importance of the thalamus in its relation to the neocortex and their responsibility in cognitive processes. In relation to connectionism, we present a bio-connectionist model from which a possible explanation for the recent experimental data is derived. From the standpoint of the history of connectionism, we try to show why the work of Wilfred Kenelm Taylor should be reconsidered.

Key words: Connectionism; Cognitive computational neuroscience; Neural network; Thalamocortical; Sensory stimulation; Perception; Neocortical layer 6

Resumo

Estudos recentes sobre a interação do tálamo com o neocórtex alimentam positivamente a suspeita de que não só o neocórtex é o principal responsável pelo processo de aprendizagem, mas sua relação intrínseca com o tálamo. A simulação computacional oferece um suporte conveniente para complementar as hipóteses derivadas destes novos resultados experimentais. Neste artigo, defendemos a necessidade de uma disciplina intermediária entre a neurociência cognitiva computacional e o conexionismo, que chamam bioconexionismo. Nós apresentamos um modelo que simula projeções bioconexionista entre o tálamo e o neocórtex, particularmente na área sensorial primária. Um novo sentido da palavra memória resulta do projeto bioconexionista. Finalmente, em busca de uma interpretação dos resultados, em comparação com o trabalho de um conexionista da primeira gama, WK Taylor, que, como resultado da contribuição profusa com a tradição, em 1964 diretrizes publicadas para um modelo cujas premissas são semelhante ao modelo aqui exposto. No plano neurocientífico, chamamos a atenção para a relevância do tálamo em relação com o neocórtex e sua responsabilidade nos processos cognitivos. Em relação ao conexionismo, apresentamos um modelo bioconexionista que deriva uma possível explicação para os dados experimentais recentes . Do ponto de vista da história da conexionismo, tentamos mostrar que a obra de Wilfred Taylor Kenelm seja reconsiderada.

Palavras chaves: Conexionismo; Neurociência cognitiva computacional; Redes neurais; Tálamo-cortical; Estimulação sensorial; Percepção; Capa neocortical 6

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Acknowledgements:

We specially thank Pablo Verdenelli for providing the space where most of the programming was carried on. We also thank Ricardo Pautassi, María Eugenia Sabatino, Irene Audisio, Laura Alonso Alemany, Hernán Acosta and Nicolás Venturelli for their help and orientation on publication standards.

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Computational modeling is a tool that offers alternative support to speculations and hypotheses from experimental data previously collected in the laboratory (Sommer & Wennekers, 2000). The cortico-thalamic projections which complete a closed circuit between the thalamus and the neocortex present an opportunity for the application of computational modeling and thus it may contribute to the effort to explain the in vivo function of these projections (Hillenbrand & Hemmen, 2002; Alitto & Usrey, 2003). In this paper, we present a computational model to confirm a particular view of the interaction between the thalamus and the neocortex which could open new possible interpretations of phenomena such as perception or memory.

Recent studies have shown that the cortico-thalamic projection in the primary sensory neocortical area to the corresponding sub-nuclei of the thalamus directly results in the coordination and precision in the firing of thalamic neurons from these sub-nuclei to the neocortex (Andolina, Jones, Wang & Sillito, 2007; Wörgötter, Eyding, Macklis & Funke, 2002; Sillito, Jones, Gerstein & Wet, 1994). This cortico-thalamic projection has the function of not only refining the receptive fields and / or modulating the firing of thalamic neurons, but also increasing the transmission of sensory signals from the periphery to the neocortex (Briggs & Usrey, 2008). The computational model presented here aims to provide explanatory support to this experimental evidence.

We propose the rearrangement on existing data on connections between the thalamus and the neocortex in computational design, suggesting there is a positive advance towards understanding the cognitive phenomena from the biological substrate of thalamo-cortical circuit if we modify the methodological approach in order to bring the spotlight into thalamic connections. A first argument for this *gestalt* turn - that a mammal can survive the ablation of large cortical areas, but completely lose the sense of reality to the lower thalamic dysfunction at some subnucleus - can be considered. We propose an excitatory recurrent architecture (Douglas, Koch, Mahowald, Martin & Suarez, 1995) to support this idea.

As for radial projections, we simply consider the thalamus and the neocortex; tangential dimensions, on the other hand, were framed based on studies of cortical cells (Yates, 2013; Gray & Singer, 1989). Since the proposed mechanism is limited to the primary sensory areas of thalamo-cortical complex, we suggest the term *perception* - understood as conscious perception - when we discuss the effect that emerges as a result of this mechanism, rather than the terms cognition and learning, whose overuse and misuse have been recently criticized (Cromwell & Panksepp, 2011; beim Graben, 2011; Globus, 1992). At the end of this paper, we present a discussion and took position on considering the possibility of speaking of *perceptual experience* based on the mechanism shown.

Connectionism, Computational Cognitive Neuroscience and Bio-connectionism

According to a particular point of view (Westen & Gabbard, 2002a, 2002b), connectionism can be considered a historical derivative of cognitivism because it attempts to decipher facts concerning knowledge of cognition from the simulation of neural networks, and not from computer algorithms, as classic cognitivism

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does. However, since the 1950s up to the present the cognitive premise has been gradually set aside from the connectionist task, usually to follow two distinct lines (Arbib, 2000). In the first group, there is a list of studies in which there are few attempts at reorganization of neurons according to a biological map although in most cases they are based on the modeling of neurons, with a degree of simplification which varies (with respect to the function of neuronal activation, for example, see summary of Kouh & Poggio, 2008). This group, which can be set as current connectionism, copies from biology the physiology of a neuron or part of it, but not the anatomy that directs the connection between certain neurons or neural groups in the brain. The second line of investigation comprises studies belonging to an even newer discipline known as Computational Cognitive Neuroscience (CCN), which adhere to biology seeking such a degree of accuracy that hardly manage to extend their speculations to cognitivism (Ashby & Hélie, 2011).

In order to distinguish between the modeling which tries to stay as close as possible to biology - always in view of technology limitations when programming - from the modeling which allows a rearrangement of *neurons* regardless of a biological order, we propose the term bio-connectionism to designate the former. An important difference between connectionism and bio-connectionism is that the former is inspired by the biology of the nervous system but its main focus is pragmatic, so that a classical connectionist system does not seem to be significant unless it demonstrates its usefulness (Minsky & Papert, 1972). The bio-connectionist system, instead, aims at the simulation of biological complexity just to understand such complexity; moreover, in the case any subsequent practical application is found, it will be welcome. Many recent publications on connectionism share the characteristic of not only representing a simplified version of the neural physiology, which is inevitable in any instance, but also adding to the creation of artificial networks features that are not derived from experimental results, but drawn from mathematical games, highlighting the difference with the bio-connectionism. Focusing expectation on a numerical result, classical connectionist networks are generally designed to solve problems; however, a system that simply is as close as possible to neural biology will not seek another result apart from the one which is generally in living things: to learn how to get away from noxious stimuli and how to search pleasurable stimuli.

Likewise, bio-connectionism differs from CCN in the sense that it does not look for accurate details in modeling, but merely deviates as little as possible from biological data, even with the simplifications needed to perform the system programming with the available resources (Piccinini & Scarantino, 2011). Thus bio-connectionism has the advantage, typical of CCN, to allow a greater restriction on the model so that two or more researchers can model the same neural structure and, if they share the same conditions for simplifying the modeled structure, could match results to a greater degree than classical connectionism (Ashby & Hélie, 2011). At the same time, bio-connectionism, the same as classical connectionism, has the advantage of achieving results more easily than CCN, which, in turn, bet on perfection in modeling and minimization of the simplifications, thematically resigning the possibility of seeing the forest for the trees. See Table 1 For a resume of the differences between classic cognitivism, cognitive computational neuroscience and bio-connectionism.

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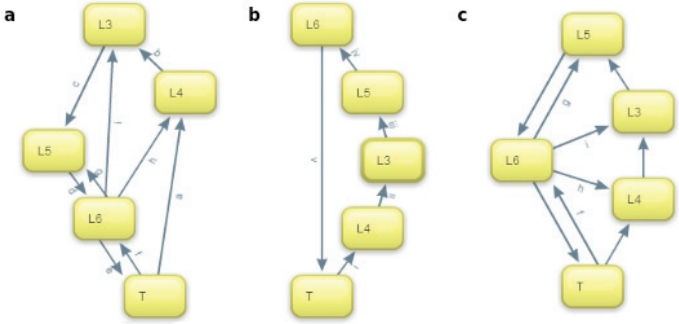
Biological Background of the Model

Now we will consider some of the most important neural pathways in the primary sensory area and, being the most studied, the one linked with vision in particular: projections associated with the lateral geniculate nucleus of the thalamus in its connection to the primary visual cortex. Projections reach the lateral geniculate nucleus through the optic tract. In turn, this thalamic nucleus projects to the primary visual neocortex. Finally, it receives projections from cortical area, but not necessarily from the same cortical layer. The main objective of thalamic projections in the neocortex is layer 4 and, in the second place, layer 6, due to the massiveness of the projection (Thomson & Bannister, 2003). Despite these are not the only projections that reach layer 4, the thalamus-cortical neurons are primarily responsible for the activity in this layer (Thomson, Bannister, Mercer & Morris, 2002). It is very well known that layer 4 projects to layer 3, and this one, in turn, projects to layer 5 with impulses that are not reciprocal (Thomson, 2010). Layer 6 receives pulses from layer 5 and in turn massively projects to the thalamic nucleus from which receives the stimulation (Sherman & Guillery, 1996).

Based on this evidence, the following successive projections have been considered to perform the bio-connectionist design: i) from the thalamus to layer 4 of the neocortex; ii) from layer 4 to layer 3 of neocortex; iii) from layer 3 to layer 5; iv) from layer 5 to layer 6; and finally v) from layer 6 to the same thalamic sub-core that started this chain. As a result, a closed five-membered or five-stations circuit is formed (Figure 1.b). To diagram the circuit, not only the most voluminous but also direct (not fuzzy) projections have been considered. Also note that cortico-cortical connections, -even those from the primary sensory area, - which is the most studied to date - have not been completely elucidated and many of them, including some of those taken here to model, especially those involving layer 6, are not fully confirmed and significant differences between experimental animals are found (Thomson, 2010).

Taking into account the great simplification that has been necessary to carry out for the design of the model, it is preferable to provide some explanations. For acceptance of this bio-connectionist model, it has been enough to find out that the modeled neural distribution is possible. The proposed demarcation or the underlining of certain projections on a multitude of others does not necessarily suggest that the distribution shown is the most probably responsible for the phenomenon that we try to explain; however, it highlights that is just not implausible. While the thalamo-cortical, cortico-cortical and cortico-thalamic pathways that have been extracted for modeling are some of the most important in the general biological circuit, they are not the only important ones, so the possibility of extending the circuit in subsequent studies to do it polycyclic or cross-linked (see Figure 1.c) should be emphasized. A modeling like this, under the relevant considerations, would certainly be a step closer to the *mimesis* of biology as the genetic evolution has "designed" over millennia.

Figure 1.



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Now it is regarded, as an example of the above, the thalamo-cortical projection to layer 6 focusing on neurons that fire into the same thalamic nucleus. Don't we have to imagine a narrower closed circuit, with two stations or layers, which only involves the thalamus and neocortical layer 6? No experimental data invites to rule out this possibility. Layer 6 is the first neocortical layer evolving in ontogeny (Rakic, 2009), which suggests that the thalamo-cortical connections to layer 6 are remnants of a precarious structure necessary when a more complex structure - including layers 3, 4 and 5 - had not reached its maturation time. In this report, it is not possible to expand on the consideration of the circuit from its ontogenetic development. Literature data concerning the connections in the mature system have certainly been taken into account for this model layout.

Bio-connectionist model

For modeling, another element apart from the functional unit called neuron and the connection between neurons called synapses has not been required (Hines & Carnevale, 1997). All neurons in the system are identical in structure. According to their structure, each neuron can be divided into four parts: a specific initial amount *d* of dendrites, a cell body which statistically processes the incoming stimuli to the dendrites, an axon which triggers as a function of the statistical result of the cell body process, and a specific initial amount of axon-terminals reached by the axonal impulse after each shot. For the sake of simplicity, the design has omitted both the axon and axon-terminals as subelements, as the design allows the cell body of the presynaptic neuron directly assigns a stimulus to the dendrites of postsynaptic neurons. Thus, upon activation of a presynaptic neuron, the program has, as a function that depends on the strength of the synapse, a voltage (weight) of each dendrite of postsynaptic neurons (Brette et al., 2007). The function of the synapse as a structural element is to reduce the voltage that reaches each dendrite, depending on a factor that varies according to the history of the neurons that participate in that synapse (Pappas, Asada & Bennet, 1971). Thus, the program distinguishes three subelements of a neuron: i) dendrites, ii) cell body (counter), iii) synapses.

The neurons are distributed into groups forming links or stations that connect successively. Each station has a specific and constant number *N* of neurons. The neurons in each station only fires to the next station, and the distribution of their synapses with neurons between stations is initially random, which ensures unsupervised dynamic and stochastic results that are more consistent with the biological architecture (Svozil, Kvasnicka & Pospichal, 1997). According to the biological evidence proposed, the closed circuit has five stations (E_0 - E_4) connected successively, while, at the same time, the last station is connected to the first one to form such circuit (Figure 1.b). For example, the fifth axon-terminal of neuron 3 of station 2, $a_{5.3.2}$, is connected with the seventh dendrite of the first neuron of station 3, $d_{7.1.3}$.

The electrical impulse (weight), measured in *V* (volts), reaches dendrites of a neuron as a result of their synapses with axon-terminals of different neurons from the previous station. After that, it is processed by the cell body in order to be distributed in multiple dendrites of different neurons in the next station. Each dendrite has assigned a voltage V_d whose absolute value decreases linearly with time (depending on the factor $r = DV_d / I$, where *I* represents the elapsed time),

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but grows strongly when it receives an impulse. At every moment, the cell body (counter) takes the sum of the voltage of each of their dendrites $V_c = S\ V_d$. Only when the sum exceeds a constant threshold value, U_A, such that $V_c > U_A$, the trigger occurs, with an intensity V_A that is always the same for the same neuron (not proportional to V_c), following a stepped nonlinear shooting or all-or-nothing logic (Adrian & Forbes, 1922). The value of V_A can be positive (corresponding to an excitatory neuron) or negative (corresponding to an inhibitory neuron). Note that both positive and negative firing occur when the sum exceeds the threshold value $V_c > U_A$ which is always positive. The axon shooting is manifested in the dendrites of the next station to which that neuron is connected, with an intensity V_a depending on the amount of these dendrites, such that $V_a = V_A / a$, and with an unchanged delay t. The delay in the program follows the premise that the only significant delay in neural time is that of the sinapse (McCulloch & Pitts, 1943). The firing produces the axon inactivity for a period T, called idle period, during which the axon is not able to shoot again (Brette et al., 2007). Synapse resistance translates into a drop in voltage that occurs in the dendrite, and is given by a permeability factor which goes from 0 to 1, allowing the relationship $p = DV_d / V_a$.

On Memory in Biochemical Terms

The "learning" of the system is related to the permeability p of the synapse, which should vary depending on the time and the shared activity of neurons that form synapse (Sutton & Shuman, 2006). The permeability at a given time at a specific synapse depends on the initial permeability p and on the previous history of this synapse (Stent, 1973). We will speak of synaptic facilitation when the permeability increases (such that $p' > p$) and of synaptic depression when the permeability decreases (such that $p' < p$). Also, we will say there is long-term facilitation when new synaptic connections are formed and there is long-term depression when a synaptic connection disappears (Brown, Chapman, Kairiss & Keenan, 1988; Ito, 1989).

Synaptic Facilitation

When the dendrite's voltage exceeds a specific threshold value $V_d > U_d$, and at the same time the axon of the same neuron is in a period of inactivity (for just shot) governed by T, the permeability factor of the synapse involved increases an amount x, so that $p = p\ ' + x$ (the resistance decreases). This coordination between the voltage of the dendrite and axon shot makes the synaptic resistance dependent on the full activity of the neuron, as the sum of the voltages of all dendrites leads to the axonal shot (Hebb, 1949). Thus, synaptic facilitation occurs only when the dendrite is stimulated in synchrony with the other dendrites in the same neuron.

If the presynaptic neuron is inhibitory (with a negative tripping current of its axon V_A), facilitating conditions are exactly reversed. It is necessary that voltage variation in the dendrite decreases below a threshold value $V_d < U_{-d}$ (such that $U_{-d} = -U_d$), and also that the postsynaptic neuron is not in an idle period, i.e. that it has not shot lately. In this case, as with excitatory synapses, permeability factor increases, $p' = p + x$. The condition that the presynaptic neuron fires in asynchrony with other presynaptic neurons that reach the dendrites of the same postsynaptic neuron reflects the effect studied for inhibitory (presynaptic) neurons to enhance their synapses with (postsynaptic) neurons that do not participate of the path that is being empowered (Stent, 1973).

The system also includes the possibility of long-term facilitation. When the increase in the p factor is such that its value reaches the unity, a new synapse between stations belonging to the connected neurons is formed so that the presynaptic neuron

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acquires a new axon-terminal and the postsynaptic neuron acquires a new dendrite (Brown et. al, 1988). In forming the new axon-terminal and the new dendrite, permeability *p* of the synapse that gave rise to them goes back to its original value. The new axon-terminal is not connected with the new dendrite, but awaits off until the next long term facilitation event, in which both a new axon-terminal and a new dendrite appear in other connections between the same two stations. The dendrite is also awaiting until the next axon-terminal is formed. Thus, long term facilitation lets the increase in branching among neurons firing in synchrony over time (Bliss & Collingridge, 1993).

Synaptic Depression

When the voltage of a dendrite exceeds a given threshold value $V_d > U_d$, and also the axon is not in an idle period, the permeability factor of the synapse of that dendrite reduces such that $p' = p - x$. In this case, the strength of the synapse increases because its activity occurs in asynchrony with the other synapses of the same neuron, since if synchronously it would produce the axonal firing (see above). Note how the system necessarily modifies the permeability of the synapses when the dendrite exceeds the threshold value, enhancing it if the neuron is in a period of inactivity, or reducing it if it is not. When the presynaptic neuron is inhibitory, the requirement to decrease the permeability $p' = p - x$ will be that the dendrite voltage falls below a given threshold value $V_d > U_{-d}$ (such that $U_{-d} = -U_d$) and also that the axon is in a period of inactivity; i.e., that it has fired recently. Finally, if the factor *p* value decreases to zero, the axon-terminal and dendrite synapse involved disappear (long-term depression) (Ito, 1989). When synapses disappear, the presynaptic neuron loses an axon-terminal and the postsynaptic neuron loses a dendrite.

On Memory in Physiological Terms

A direct consequence of the Hebb's rule (1949) - immediately understood by his contemporaries (cf. Taylor, 1958) - is that, due to synaptic potentiation, an external impulse (input) eventually forms neural paths with neurons whose connection is enhanced in relation to other neurons in the same area that have not been reached by the neural impulse. For the thalamo-cortical circuit here studied, the external impulse is sensorial and the pathways are formed through the stations in the closed circuit. Each sensory stimulated neuron receives a voltage V_{ext} in its dendrites. Synaptic modifications have the gradual effect of forming cell assemblies (CA) through the successive stations, which are sets of neurons of each station connected to each other with less resistance (or higher permeability) than other neurons (Lansner, 2009). According to the number of neurons per station, a closed circuit is capable of forming a specific amount of CA before becoming saturated. Once the circuit is saturated, the formation of a new CA involves the dissolution of one or more of CA previously formed.

CA formation is led by the first station, which corresponds to the thalamus. This first station E_o receives external activation corresponding to the sensory pathway, and also receives neural activation by the closed circuit, that is, projections from the last station (Figure 2). Accordingly, each dendrite of neurons of the first station is indiscriminately and randomly connected with both sensory neurons and neurons from the last station (Destexhe, 2000). The thermodynamic effect expected for this feature of the circuit is that, in

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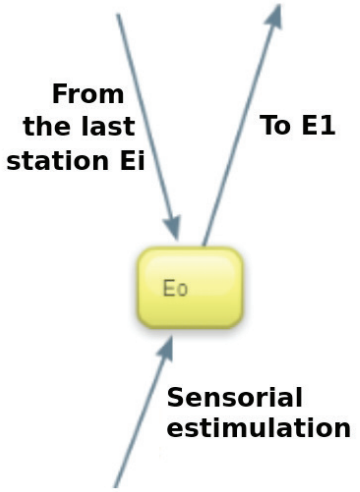
the first station, neurons that are being stimulated by both sides simultaneously will most likely trigger and, therefore, these will be more likely to participate in CA, which marks a *neural groove* along the circuit that ends in the same station. Two extreme cases can be plotted: when stimulation chain along the closed circuit derives in neurons in the last station which, in general, are not connected with neurons of the first station, the enhanced route is not reinforced. In contrast, when the chain stimulation results in neurons of the last station activating generally the same neurons in the first station that are activated by outside sensory via, the pathway stimulated along the whole closed circuit reinforces and a CA is formed and/or reinforced. In this sense, it is fair to say that the circuit functions as a servo loop (Ahissar & Kleinfeld, 2003; Wiener, 1949). This result is also consistent with experimental evidence according to which the cortico-thalamic projections from layer 6 produce alignment and empowerment of thalamic neurons that are firing (those within CA) as an effect (Andolina et. al, 2007; Sillito et. al, 1994).

In the formation of a CA, sensory neurons must remain activated enough time for the corresponding synapse along the circuit can be enhanced. Biochemistry memory stored in each synapse of the circuit now comprises a physiological sense when considered statistically, since it corresponds to the memory of the circuit saved as a set of neurons (a CA). This is because the activity in the thalamo-cortical loop immediately modifies synaptic properties of neurons involved in this closed circuit (Steriade, 2001). This proposal is outlined to solve the problem of translating the neural dynamics to a macrostrucutre (beim Graben, Barrett & Atmanspacher, 2009). The term *memory* has been used in its broadest sense as the ability to storage *something* and then to bring it back. This *something* in terms of the thalamo-cortical dynamic proposed is CA (in physiological terms), or the set of synaptic permeabilities of neurons participating in the code (in biochemical terms) (confront Adams & Cox, 2002). CA can be stored for retrieval in the future. If the same distribution of active sensory neurons is repeated, the code potentiated in the past directly activates without the need to recreate synaptic facilitation in each of the synapses throughout the stations (Azouz & Gray, 1999; Civillico & Contreras, 2012). The system thus tends to increase synchrony (Brette, 2012) between the neurons forming CA in the closed circuit and sensory neurons afferent to the thalamus whenever activated.

Figure 2.



Computational bio-connectionist modeling

The structure we call “brain” consists of a set of neurons arranged in accordance to the biological evidence which was analyzed previously here (see above). Time is measured in iterations, and an iteration corresponds to one pass through the entire algorithm of the source code. The set of iterations within that the

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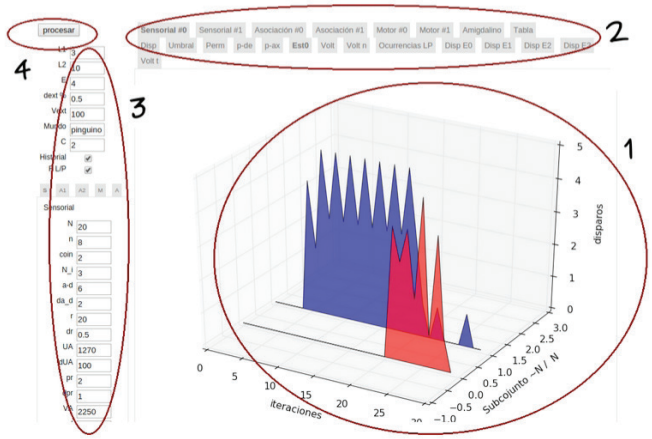
external stimulation is kept constant is called pulse, so that each pulse consists of a certain number *L* of iterations. In turn, the set of pulses constitute a run. The run is the lifetime of the brain, from birth to death, and includes a certain number of pulses. The brain has an initial state, before the run, and a final state, after the run. The run has the only effect on the structure of modifying synaptic permeability of neurons. The initial state is determined by parameters entered by the operator. After the process the operator can use the final state of the brain to evaluate the performance of the program. After observing the results (final state), the operator can modify the system parameters (initial state) and start a new run, by iterating until achieve optimal values. Figure 3 shows the platform designed for operator use.

E_o station is divided into three equal groups of neurons: *A*, *B* and *C*. For each pulse, the external stimulation is kept constant by activating one and only one set of the E_o station. In the following pulse, external stimulation can change or remain constant. It is desired to evaluate the degree of formation achieved for CA, which is referred as Encoding Index, and the trend of neurons in E_o station to facilitate dendritic permeability as a coordination between the external stimulation and stimulation from the last station E_4; this tendency is measured by an index called Configuration Index.

Optimal parameters were obtained by running a single pulse with *L = 100* iterations. By the end of the run, those neurons that had been activated the most, were expected to have their dendritic permeability

Figure 3.



above the value of initial permeability *p*; neurons with an average activation should have had the permeability of their dendrites below the initial permeability *p*; and finally neurons that had been hardly activated should have had permeability close to *p* (Figure 4). The first of these three neuronal groups corresponds to the neurons firing in synchrony and, therefore, their dendritic permeability is enhanced; the second one corresponds to neurons firing in asynchrony and, therefore, their dendritic permeability decreases; and the third one corresponds to neurons that did not fired or that hardly fired and, therefore, their permeability is maintained.

To specify the width comprised of the cerebral cortex, we turn to the study of cortical columns (Yates, 2013; Gray & Singer, 1989). In general, each column implies around 80 neurons per cortical layer (Yates, 2013), so we set *N = 81*. The number is divisible by three to facilitate sensory stimulation symmetry, divided in three groups of neurons in the first station: A, B and C. For each pulse, sensory stimulation activates 27 neurons. With respect to the dendrites, we define *d = 6*. While it is known that the number of dendrites per neuron is much higher, we argue that

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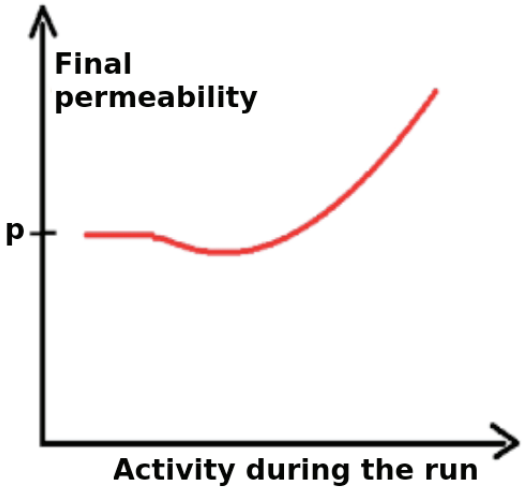
Figure 4.



i) in the model, dendrites that receive impulses from subcortical areas were not considered, with the only exception of sensory stimulated neurons; ii) dendrites that receive stimulation from other areas of neocortex or other thalamic subnuclei were no considered; and iii) a neuron is connected to other adjacent station neuron via a set of synapses and not just one, and the modeling was able to simplify this by just connecting neurons with one synapse. The values for each parameter, including those just discussed, are shown in Table 2.

Also a run of three pulses was performed as a second part of the experiment: a first pulse *L1* consisting of 100 iterations with sensory stimulation A, a second pulse *L2* consisting of 50 iterations with stimulation B, and a third pulse *L3* consisting of 50 iterations with stimulation A again. The objective of the experiment is that CA achieved during *L1* is maintained to some extent when repeated sensory stimulation A in L3. We have used two indexes to measure results.

Encoding Index.

Define P as a set of neurons such that | *P* | = *N / 3 = 27*. *P* is the set of neurons of each station that have fired more during the pulse. For each pulse, the system returns the values *Pp* and *nPp*, being *Pp* the average of *P* neurons firing during the pulse, and *nPp* the average of shots from other neurons (neurons *not-P* or *nP*) during the pulse in the entire circuit. The encoding index is defined as *ICd: Pp / nPp* and should be as large as possible for L3. *ICd ≤ 1* equals a failed case where no CA is formed.

Table 2.

Parameter	Value
L	100
E	5
Vext	100
N	81
d	6
r	20
UA	1270
VA	2250
Ud	700
p	0.5
x	0.05

Configuration Index.

In this case *P*, the set of neurons that have fired more during pulse, is a variable value ranging from 1 to *N*. This time, the system evaluates dendritic permeability of *P* neurons. Dendrites connected to *P* neurons in the previous station are called *dP*; dendrites connected with *nP* neurons in the previous station are

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called *dnP*. For each pulse, the system returns the values *dPp* and *dnPp* for each *P*, being *dPp* the average permeability in dendrites connected with *P* neurons, and *dnPp* the average permeability in dendrites connected with *nP* neurons during the pulse across the circuit. The configuration index is defined as *ICf = dPp / dnPp* and should be as large as possible for *L3*. This confirms that *P* neurons develop a distinguished permeability with respect to *nP* neurons. *ICf ≤ 1* equals a failed case where no CA is formed.

Technical Characteristics of the Software we Have Used

It is considered essential to have a programming language that allows the rapid development of the program and that adapts to frequent changes that are usual in the development phase. With no efficiency requirements, such as memory management, pointers and other aspects of low-level programming, languages like Python or Java are an advantage to languages like C++ in which it is necessary to handle these issues. Python has been chosen as the programming language that best suits the needs of the project. As for the GUI, we have decided to use Flask, since it is a minimalist and simplistic framework whose learning curve to begin developing applications is very short. Furthermore, it has no restrictions and allows implementing each part as the programmer decides at any time. On the other hand, there is no database related to the project, so this feature is not significant in terms of our development.

Results of the computational simulation

In the first part of the experiment, after getting the optimal value for each parameter, with the help of graphics, a set of more active neurons have been observed and also the increased dendritic permeability in synapses which connect these neurons along stations. The first graph (Figure 5.a) shows how the most active neurons along the stations are those that increased their dendritic permeability above the initial permeability. In this graph it cannot be observed, however, how dendrites of neurons with an average activity ended with permeability below the initial permeability *p*, with the exception of the first station. The reason for this is that the bars show the average of each neuron dendritic permeability, so the graph is not sensitive to the discrimination between the same neuron dendrites. For this reason, a graph showing the distribution of the permeability of each station by dendrite (Figure 6) has been designed. In this graph, it can be seen how, in each station, there are dendrites with decreased permeability compared to the original, others that have increased it, and a third group that has maintained its initial permeability *p*. The first group belongs to active neurons, the second one to neurons with intermediate level, and the third one to neurons with a low level of activation or that have not been activated.

The next graph (Figure 5.b) shows axon permeability by neuron, that is, the permeability of the postsynaptic dendrites to which each neuron is connected. This chart shows more clearly that the most active neurons are those with the highest axonal permeability once the run is finished. In the last station, a different behavior is observed. The most active neurons are the ones that have modified more their permeability, but not all of them have increased it. This is because the axon permeability of neurons in the last station depends on its connection with neurons of the first station, some of

Check Figure 5a in page 89
Check Figure 5b in page 89
Check Figure 6 in page 90
Check Figure 7 in page 90

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which have external stimulation. When an active neuron in the last station strikes a neuron of the first station which is being sensory stimulated, its axon permeability increases, because this neuron of the first station is active. When an active neuron in the last station strikes a neuron of the first station that is not being sensory stimulated, its axon permeability decreases because it is less likely that this neuron of the first station is active. This graph is, therefore, an evidence of the prominent role of the thalamus (corresponding to the first station) in the formation of CA.

The fourth graph (Figure 7) shows the number of shots per station over time (over iterations). It shows how, from the beginning, the activation is succeeding throughout the stations, reaching the last station at iteration 22 when the entire circuit is disabled and the dendritic voltage *Vd* is equal to 0. This finding is relevant to read the fifth graph (figure 8) which shows the activation of neurons in the first station E_o not being sensory stimulated. Since the beginning of the run, none of these neurons triggers, as they receive neither external stimulation nor anything from the same closed loop until iteration 22. These neurons of the first station which do not receive external stimulation activate in iteration 26, but immediately after that the system tends to remove this "parasitic" and not sensory reinforced activation. In iteration 54, the level of activation of these neurons is already negligible.

For the design of the sixth graph a neuron from the second station was taken at random and the voltage V_d of each of its dendrites along the run was measured. In the first sample (Figure 9.a) there are only three dendrites that receive stimulation from the previous station, and two of them in a very weak way. The third of these dendrites receives energetic activation, but it cannot produce the firing of the neuron as it is not in synchrony with the others. For this reason, its permeability decreases gradually. Each stepped segment corresponds to a period in which the voltage rises to exceed the threshold $V_d > U_d$, and, as permeability changes negatively, the next period shows lower intensity steps. It is confirmed that the first step of the first cycle reaches 167.5 V, to gradually decrease until the first step comes only up to 103.5 V in the last cycle before finishing the run. The second sample (Figure 9.b) is an example of the opposite. Most of dendrites of the neuron in the second station being monitored are activated by neurons from the previous station. This causes the neuron to have a high activation and, therefore, the permeability of dendrites increases successively along the run. These last two graphs are an example of application of the Hebb's rule for memory in biochemical terms.

In the second part of the experiment, with a run of three pulses, optimal parameters (shown in Table 3) where found. *ICd* and *ICf* for each station as well as the media for *ICd* and *ICf* for the circuit are indicated. Both were positive and also showed an increase between *L1* and *L3*. *ICd* rose from 161.2 to 286.8. *ICf* rose from 1.1961 to 1.2617. To confirm the trend, the run continued until *L15*, and *ICd* remained with values close to *L3* (Cf. Table 4), whereas *ICf* rose to values around 1.4. The last column of Table 3 shows the values of the last pulse, while Table 4 shows the values of the last eight pulses. These values clearly show the system tends to form a CA and settle it with the passing of the iterations.

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Table 3.

	L1 = 100 Est Ext = A		L2 = 50 Est Ext = B		L3 = 50 Est Ext = A		L15	
Estation	Icd	Icf	Icd	Icf	Icd	Icf	Icd	Icf
0	123	1.4240	347	1.3325	393	1.3799	217	1.2546
1	335	1.2242	140	1.1836	193	1.2629	205	1.4904
2	159	1.1523	174	1.1667	201	1.2342	654	1.4616
3	124	1.1492	186	1.2274	235	1.3073	155	1.4821
4	65	1.0309	243	1.0463	412	1.1243	57	1.4293
Prom	161	1.1961	218	1.1913	287	1.2617	258	1.4236

Discussion

It has been demonstrated that it is possible to obtain configuration via a suitable neuronal distribution and without recourse to further elements than neurons. The configuration is defined as the tendency in the thalamo-cortical circuit to produce CA, in other words, it is the tendency to strengthen the connection between some neurons along the circuit, according to external stimulation entering through the first station. This first E_o station, in biological terms, corresponds to the thalamus and the configuration is, as a tendency, the condition of perception. This last statement is discussed below by comparing the results obtained with the work of W. K. Taylor.

Limitations of the Model

Now the experiments have been presented and before beginning the main discussion of the results, the possibilities of the future program development should be highlighted. First, even if the showed features are attributed doubtlessly to the connections between the neocortex and thalamus, we should clarify that influences of connections with other subcortical areas were not taken into account. Although the omission is justified, we suggest the possibility of extending the application of the model to other neural nuclei.

Neurons communicate not only according to the spatial region (the core of gray matter) in which they are and the spatial region where they project their axons, but also depending on the shooting mode (Brette, 2012). This has not been considered in the modeling. With the nonlinear stepped shot, the intensity of stimulation results in: a maximum frequency (equivalent to a shot every two iterations) if the dendrites of the neuron keep the sum over the activation level U_a; zero frequency (without firing) if U_a activation threshold is not reached; an irregular state alternating between the previous two, which cannot be considered a low frequency by its irregularity. There is, therefore, an entire neural *language* that has been left out, and it is more than necessary to propose the study and reflection on the design of programming networks that, apart from representing topical distributions - which is inevitable and essential - , take into account the firing pattern of neural populations involved in the circuit.

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It should be also clarified that the rearrangement of the closed circuit has not been deducted from an analytical view and understanding of the pathways between the neocortex and thalamus; on the contrary, once the abstract model has been apprehended, we sought and found in the thalamo-cortical system an opportunity to apply the model. That is why the level of detail and consideration for biology by model has been scarce. There is a long and continuous way to the subsequent realization of increasingly finer details, incorporating them to the model, for it to be progressively more comprehensive and reflects its object with increasing appropriateness. The following are suggestions on how to advance the development of a model to increase the credibility of the program results and the possibility to directly translate them into biology.

i) What is the effect on the synchrony achieved if it is added to the circuit the thalamo-cortical projection, less bulky but not negligible, into the layer 6 innervating neurons that directly shoot into the primary somatosensory thalamus?

ii) What happens if every variety of inhibitory interneuron, both in the neocortex and in thalamus, is included?

iii) What are the results of adding each of the secondary projections that achieve the same objective but mediately, for example cortico-thalamic projections in layer 6 projecting to the reticular nucleus of the thalamus, which in turn triggers inhibitory projections to the thalamic corresponding subnuclei considered?

iv) In the model presented, sensory stimulation involves the activation of one of three neural groups in the first station. How to achieve a system that recognizes external stimulation increasingly bounded?

These all are considerations that aim at refining the model, always in view of the marked ability to synchronization between sensory activation and activation of the first station from the last one, and that does not account for more than the primary sensory area. All this great horizon ahead is understood under the programmatic nature of this report and a short gestation period. However, if we think of it, there is not a period somewhat shorter than connectionism as discipline itself.

Taylor's intuition

The bio-connectionist model has allowed the distinction in the neuronal network of a cellular assembly from another, or from the rest of the circuit. It is necessary to clarify in what sense this is desirable and what feature of the nervous system has been intended to be modeled. In relation to this, we would like to mention that the British W. K. Taylor drafted a similar idea half a century ago. Nevertheless, he could not carry it out with the model proposed.

Although the roots of connectionism can be traced far back, it was in the fifties when neural networks began to be used to account for two abilities attributed as characteristic of human mind: the associative memory and recognition of patterns. It is noteworthy that the first work in this field belongs to Taylor himself who, on that occasion, proposed a three-layers network: one layer with sensory units, one with associative units and the latter with motor units, with a training procedure under Hebb's rule: activated weights increase if desired motor units are activated (Taylor, 1956). In its processing, the network gets to associate different sensory patterns, and displays a behavior similar to Pavlovian conditioning. In later works (1958, 1960, 1965), Taylor built a more elaborate network, with synapses returning from motor to sensory units and synapses between units of the same layer. Near the end of his career, he even tried to build electronic devices for storing memory (Taylor, 1975) and pattern recognition (Taylor, 1979). In 1964,

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he ventured a biological interpretation of his network, suggesting that the association areas of the cerebral cortex and thalamus contained those networks. That work is analyzed further below.

Taylor says that perception is produced from a fixed system which forms certain functions of the incoming signals and selects the maximum. This "fixed system" should be in the thalamus, and should be understood as fixed in the sense of *permanent* despite variations in perception. The fact that the system selects the maximum can be interpreted from recent evidence (Andolina et. al, 2007), whereby the cortico-thalamic feedback increases the accuracy of the firing of thalamic neurons, as explained above. There is a memory system -Taylor continues- that initially shows no pattern of recognition (perception), but automatically forms the functions required for such recognition. The neocortex is interpreted as a memory system, but Taylor fails in this case to speculate on the cortical "function" that makes this "memory" possible. He postulates a mathematical operation performed by the cortical neurons which results in the selection of the maximum. The existence of this mathematical operation has never been demonstrated. In this paper we have shown how this "function" can be fully understood from the activity of the thalamus which orders his shot according to the sensory stimulation and, at the same time, the cortico-thalamic stimulation (measured as Configuration Index). So the main difference in our proposal is that we don´t postulate the perception as the result of a mathematical operation that selects a maximum, but as a result of a tendency (called configuration) guided by a biological mechanism.

There is a learning process -Taylor moves on explaining- in which each stimulus produces a peak of activity in cortical efferents (to the thalamus) that have produced the most favorable response to the stimulus in the past. This is equivalent to saying that cortico-thalamic stimulation enhances thalamic activity towards the neocortex in a dynamic that has to do with its past activation. If the distribution of cortico-thalamic neurons at a time produced no enhancement in the thalamic projections; that distribution will be removed in the future. If, instead, the distribution in the cortico-thalamic projection strengthens the thalamus-cortical shots, this distribution is enhanced. This mechanism does not differ substantially from the bio-connectionist mechanism that has just been shown.

These lines have briefly summarized the Taylor´s proposal but not the intuition resulting in the application of the mechanism. Taylor was interested in the learning process explained from a (bio-) connectionist model. When we interpret his writing, it becomes clear that learning has to do with the maximum being selected from successive interactions between neurons, but *we consider a mistake to interpret that learning corresponds to the mathematical function postulated by Taylor to select the maximum*. We think that, for Taylor, learning has to do with the tendency to maximum, not the function that allows this tendency. Mathematical function is behind this tendency, but it is not possible to access the principal idea of Taylor by identifying the mathematical result of the equation with the decision that corresponds to this tendency.

According to the previous clarification, the closeness of Taylor's study to our proposal may be understood. Although we start from the same background idea, we avoid postulating a mathematical function as a condition of the perception. If the thalamus selects and powers those neural pathways that make the cortico-thalamic projections match the sensory projections, this does not mean the thalamo-cortical

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system has a Boolean behavior. Instead, the particular role of the thalamus in the thalamo-cortical circuit can be understood from a trend that gradually causes synaptic permeabilities to go changing: they increase when the stimulation that converges with the thalamus is coincident (activating the same thalamic CA) and they fall when it is not. This trend is the one we try to compare with the "decision" for which the mechanism selects a maximum in the Taylor system.

Conclusions

While it is true that, in the beginning, the original purpose of modeling neural networks was to mimic brain function or parts or aspects of it (Taylor, 1956), it is noteworthy that this activity made it possible to understand the brain itself from a "connectionist" point of view (Lashley, 1950; Luria, 1962), and this trend or theoretical preference is still present today (beim Graben, 2011). Nowadays, the theory of cell assembly (Lansner, 2009; Hebb, 1949), besides being a powerful tool for network design, can be considered part of the background of most of the speculation about brain function in biological terms, regardless of the possibility that this function is simulated by a computer model. The generalization of this idea leads to the conclusion that not only does the biological map serve as a substrate for the computer simulation, but also the simulation results and the conclusions which are drawn from these results -and even the language used in modeling- may influence how we understand the biological brain.

The thalamo-cortical mechanism centered in the thalamus we presented here attempts at explaining, first and foremost, the function of the projections from the thalamus to layer 6 of the neocortex (Wörgötter et. al, 2002; Andolina et. al, 2007; Sillito et. al, 1994). Secondly, it also helps to explain phenomena that are still not fully understood as synchronized dynamic activity patterns the so called brain oscillations (Briggs & Usrey, 2010; Gray & Singer, 1989; Eckhorn et al., 1988), reinforcing the view that the cortical control on the thalamus is essential in coordinating the widespread and coherent oscillations (Destexhe, 2000).

An important step has been taken in admitting that the thalamus is not merely a relay center to the neocortex (Gerstein, Kirkland, Musial & Talwar, 2002). This paper suggests a need to take another step towards consideration of the thalamus as a center of activity in its communication with the neocortex. When it is decided to make the thalamus, and no longer the neocortex, the center of the mechanism responsible for perception, the above conclusion can be reformulated by saying that conscious perception occurs when, in neural terms, sensory stimulation to the thalamus overlaps with the cortical stimulation to the thalamus, but not identified with the function that allows the overlay, but with overlapping itself. The new view allows understanding the feedback between the thalamus and the neocortex as a closed loop that begins and ends in the thalamus.

Along these lines, it has been reached a system in which the memory is formed, stored, retrieved and reformulated in terms of synaptic forces without apply more than permeability (or resistance) of the synapse to recognize a memory system. The *choice* of the maximum path corresponding to the conscious perception in terms of Taylor, is not the result of a mathematical formula, but a temporal Hebbian process (Fiori, 2005).

Received: 16/01/2015
Accepted: 07/06/2015

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Table 1.

	CCN	Connectionism	Bio-connectionism
Limitations of the modeling	It adheres to biology seeking such a degree of accuracy that hardly manage to extend its speculations to cognitivism	It copies from biology the physiology of a neuron but not the connection between neurons or neural groups in the brain	It makes simplifications of the biological map that requires further justifications
Consideration of biology	It maintains as close as possible to biology	It allows a rearrangement of neurons without considering a biological order	It tries to stay as close as possible to biology
Main focus	It seeks pragmatical results, but its main focus is the simulation of biology	Its main focus is pragmatic; it needs to demonstrate its usefulness	It wants to understand biological complexity; practical application is negligible
Empirical bases	It reproduces neurons features and neural networks from experimental results	It creates artificial networks that are drawn from mathematical games	It creates artificial networks that are derived from experimental results
Simplification criteria	It seeks accurate details in programming	There is not a criteria of simplification	Substantial simplifications are needed
Reproduction of results	Two or more researchers can model the same structure and reach matching results	It is not possible to have a map of simplification conditions from biology according to which to be able to compare results	Two or more researchers who share simplification conditions can model the same structure and reach matching results
Cost of results	Perfection in modeling seeking makes harder to reach significant results	It achieves results more easily and with lower cost	It achieves results more easily and with lower cost

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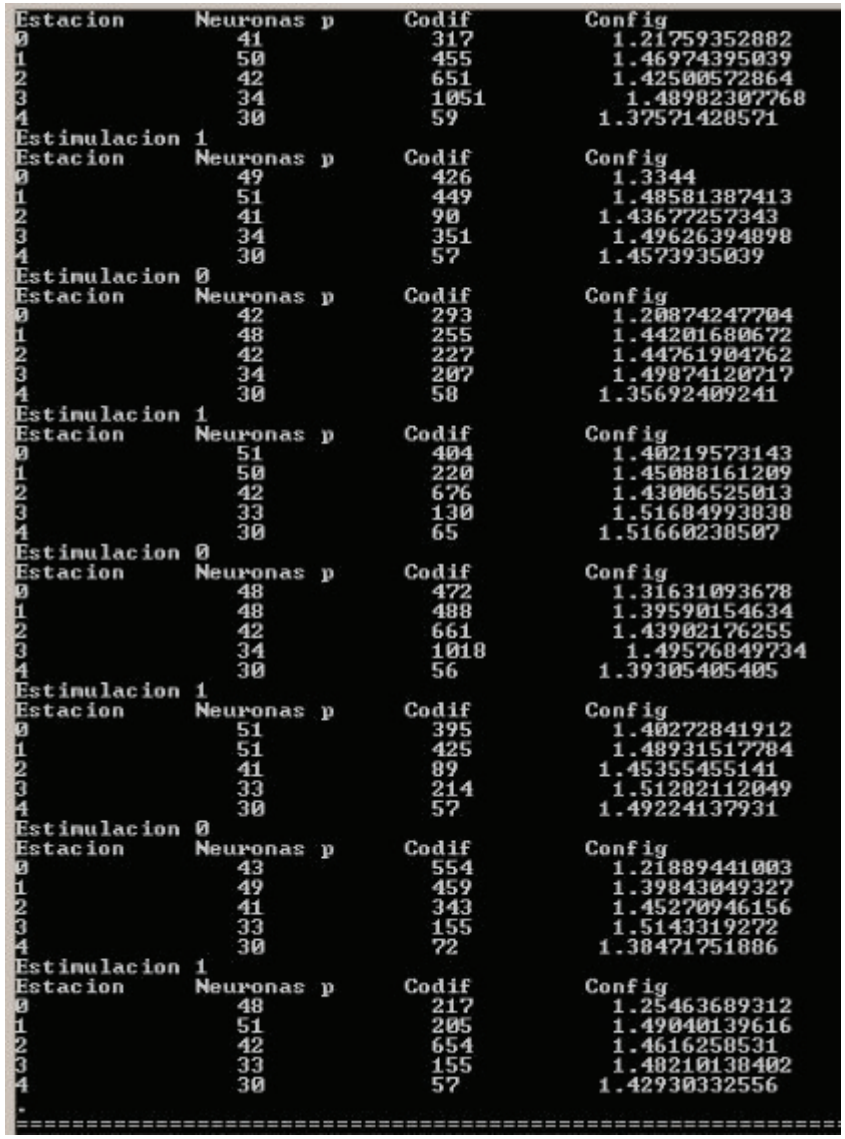
Table 4.



Estacion	Neuronas p	Codif	Config
0	41	317	1.21759352882
1	50	455	1.46974395039
2	42	651	1.42500572864
3	34	1051	1.48982307768
4	30	59	1.37571428571
Estimulacion 1			
Estacion	Neuronas p	Codif	Config
0	49	426	1.3344
1	51	449	1.48581387413
2	41	90	1.43677257343
3	34	351	1.49626394898
4	30	57	1.4573935039
Estimulacion 0			
Estacion	Neuronas p	Codif	Config
0	42	293	1.20874247704
1	48	255	1.44201680672
2	42	227	1.44761904762
3	34	207	1.49874120717
4	30	58	1.35692409241
Estimulacion 1			
Estacion	Neuronas p	Codif	Config
0	51	404	1.40219573143
1	50	220	1.45088161209
2	42	676	1.43006525013
3	33	130	1.51684993038
4	30	65	1.51660238507
Estimulacion 0			
Estacion	Neuronas p	Codif	Config
0	48	472	1.31631093678
1	48	488	1.39590154634
2	42	661	1.43902176255
3	34	1018	1.49576849734
4	30	56	1.39305405405
Estimulacion 1			
Estacion	Neuronas p	Codif	Config
0	51	395	1.40272841912
1	51	425	1.48931517784
2	41	89	1.45355455141
3	33	214	1.51202112049
4	30	57	1.49224137931
Estimulacion 0			
Estacion	Neuronas p	Codif	Config
0	43	554	1.21889441003
1	49	459	1.39843049327
2	41	343	1.45270946156
3	33	155	1.5143319272
4	30	72	1.38471751886
Estimulacion 1			
Estacion	Neuronas p	Codif	Config
0	48	217	1.25463689312
1	51	205	1.49040139616
2	42	654	1.4616258531
3	33	155	1.48210138402
4	30	57	1.42930332556

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Figure 5a.

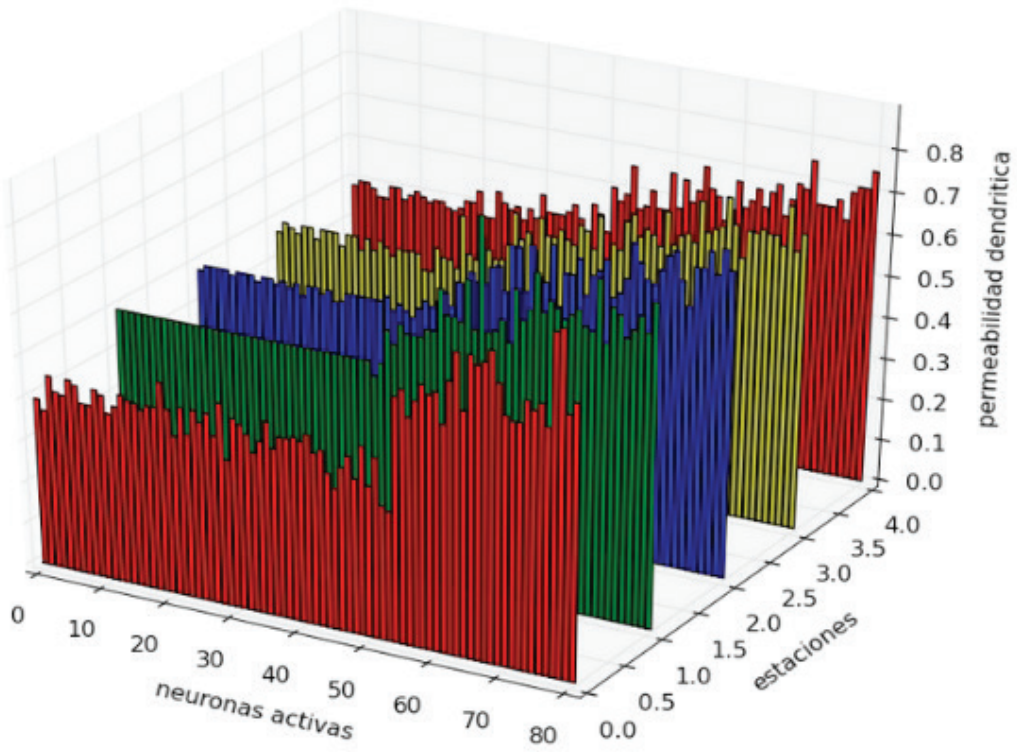

Figure 5b.

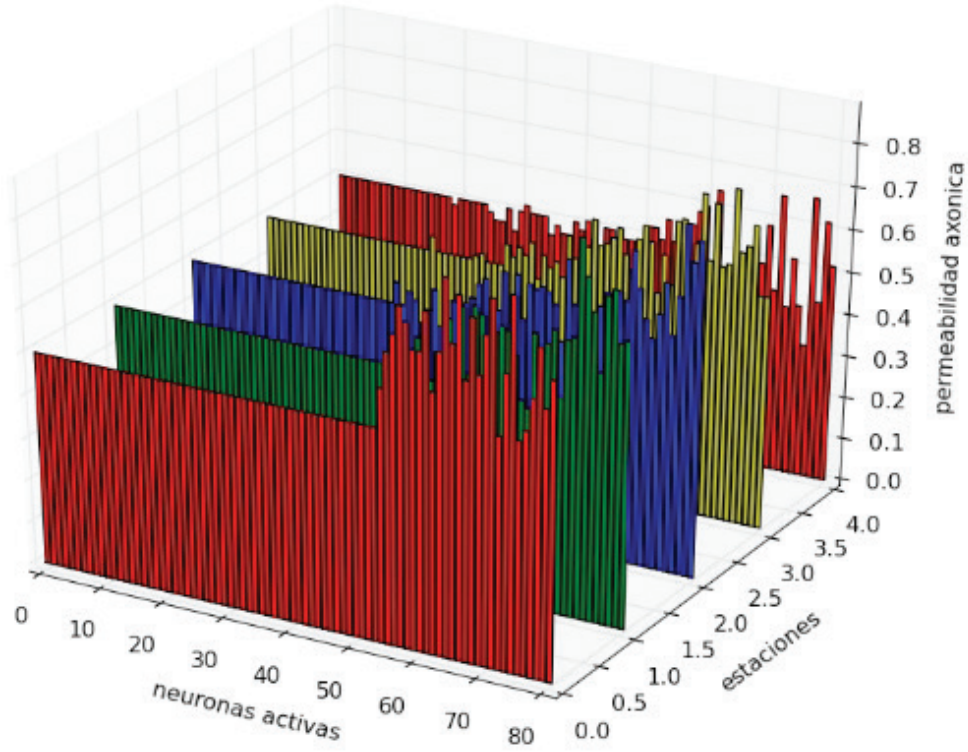

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Figure 6.

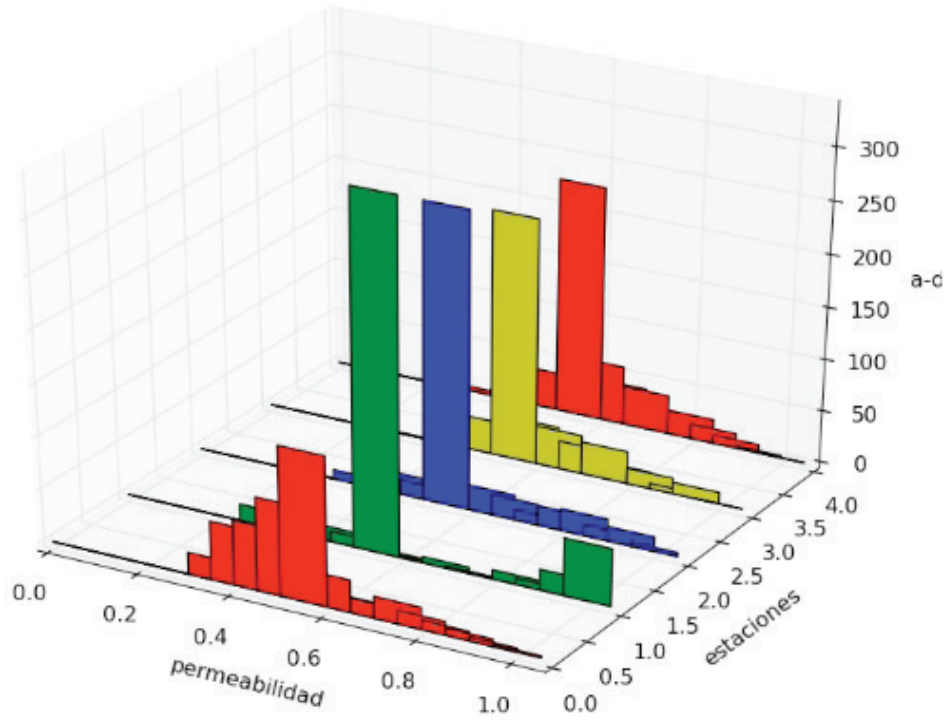

Figure 7.

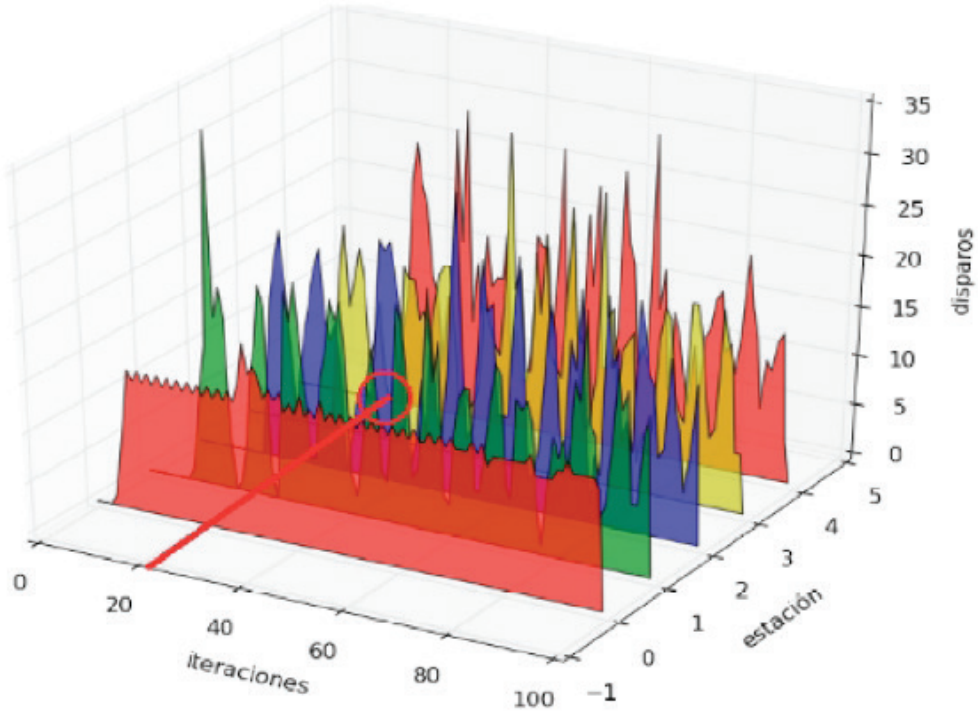

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Figure 8.

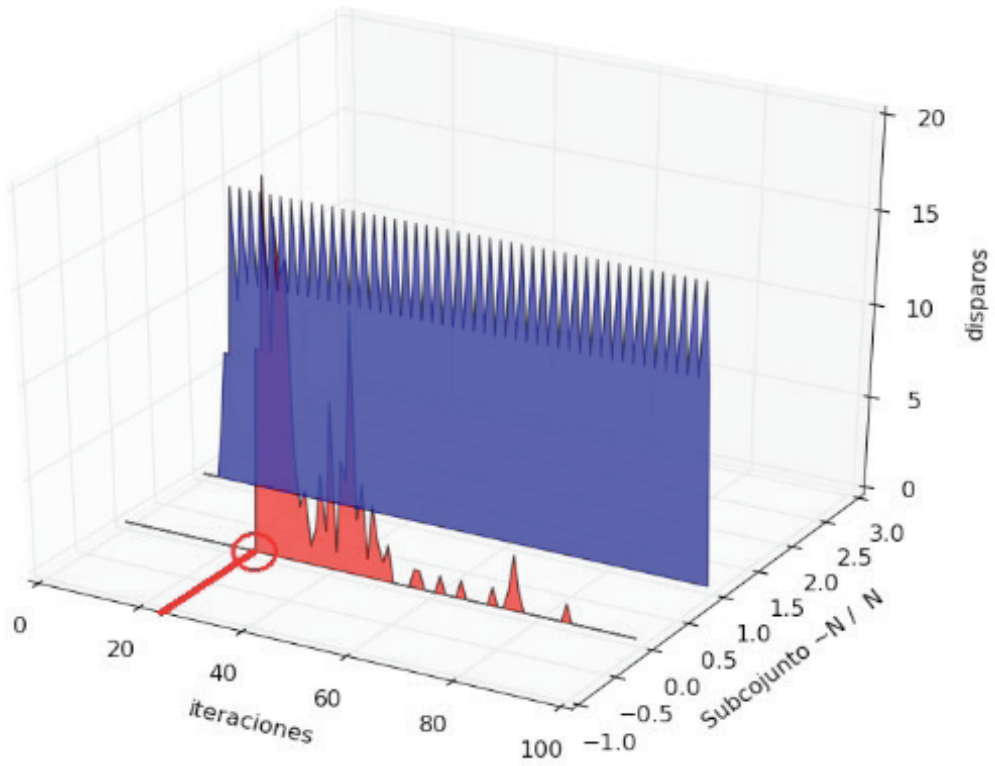

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Figure 9a.

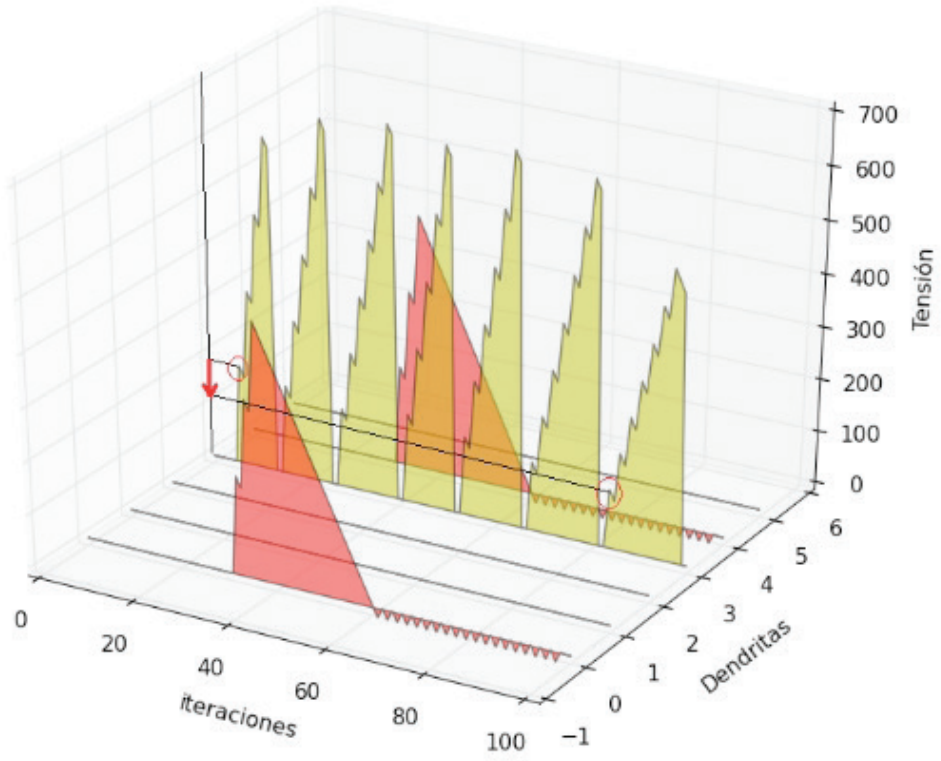
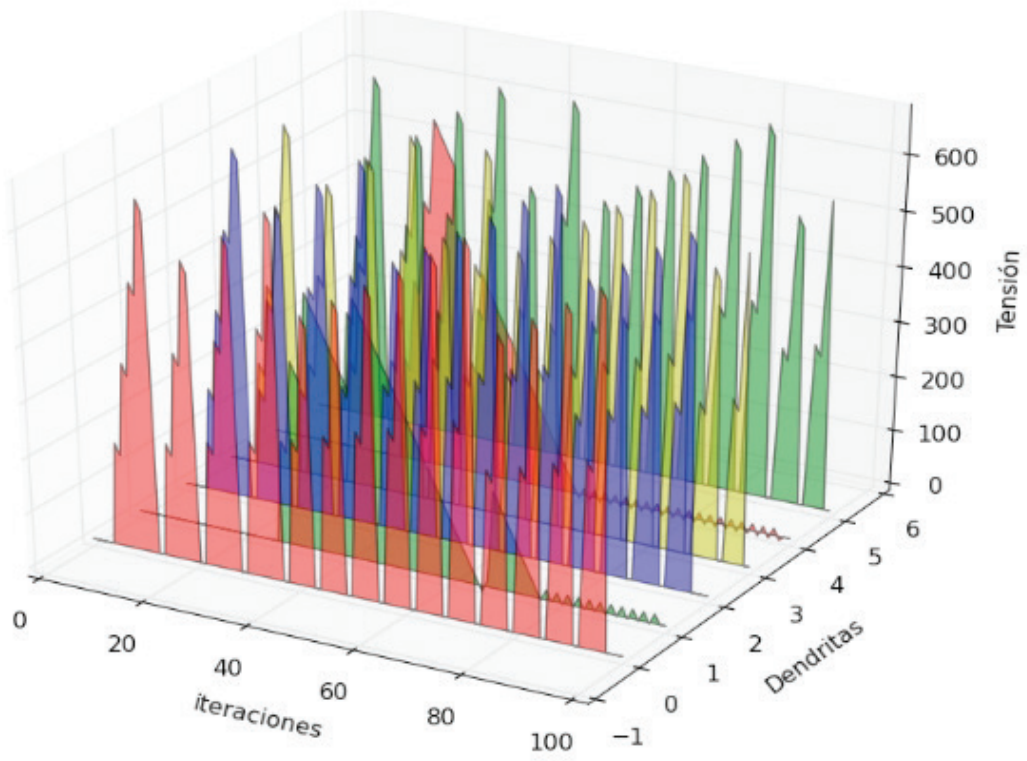


Figure 9b.



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Acronyms used for the modeling

d	Number of dendrites in each neuron
a	Number of dendrites of the next station with which a neuron is connected
dPp	Media of the permeability of dendrites connected with *P* neurons during a pulse
dnPp	Media of the permeability of dendrites connected with *nP* neurons during a pulse
E	Station
I	Iteration
ICd	Codification index
ICf	Configuration index
L	Pulse
N	Number of neurons per station
nP	Set of neurons firing less during a pulse
nPp	Media of *nP* neurons firing during a pulse
P	Set of neurons firing more during a pulse
Pp	Media of *P* neurons firing during a pulse
p	Permeability factor of the synapse
r	Rate of decreasing to zero of the dendritic voltage variation
T	Inactivity period of the axon, during which a neuron cannot fire again
t	Delay between the axon firing and the dendritic voltage change of the postsynaptic neuron
$\mathbf{U_a}$	Threshold value to V_c, after which the activation of the neuron is produced
$\mathbf{U_d}$	Upper threshold to V_d, whose synaptic facilitation depends, for the excitatory synapse
$\mathbf{U_{-d}}$	Lower threshold to V_d, whose synaptic facilitation depends, for the inhibitory synapse
$\mathbf{V_A}$	Intensity of neural firing
$\mathbf{V_a}$	Intensity of the voltage on each dendrite from the presynaptic neuron
$\mathbf{V_c}$	Sum of the voltages in each dendrite of a neuron
$\mathbf{V_d}$	Voltage of a dendrite
$\mathbf{V_{ext}}$	Voltage on dendrites of neurons which are sensory stimulated
x	Increasing or decreasing value for the *p* factor in synaptic facilitation of depression respectively

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