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Biologically Plausible Artificial Neural Networks

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1. Introduction

Artificial Neural Networks (ANNs) are based on an abstract and simplified view of the neuron. Artificial neurons are connected and arranged in layers to form large networks, where learning and connections determine the network function. Connections can be formed through learning and do not need to be 'programmed.' Recent ANN models lack many physiological properties of the neuron, because they are more oriented to computational performance than to biological credibility [41].

According to the fifth edition of Gordon Shepherd book, *The Synaptic Organization of the Brain*, "information processing depends not only on anatomical substrates of synaptic circuits, but also on the electrophysiological properties of neurons" [51]. In the literature of dynamical systems, it is widely believed that knowing the electrical currents of nerve cells is sufficient to determine what the cell is doing and why. Indeed, this somewhat contradicts the observation that cells that have similar currents may exhibit different behaviors. But in the neuroscience community, this fact was ignored until recently when the difference in behavior was showed to be due to different mechanisms of excitability bifurcation [35]. A bifurcation of a dynamical system is a qualitative change in its dynamics produced by varying parameters [19].

The type of bifurcation determines the most fundamental computational properties of neurons, such as the class of excitability, the existence or nonexistence of the activation threshold, all-or-none action potentials (spikes), sub-threshold oscillations, bi-stability of rest and spiking states, whether the neuron is an integrator or resonator etc. [25].

A biologically inspired connectionist approach should present a neurophysiologically motivated training algorithm, a bi-directional connectionist architecture, and several other features, e. g., distributed representations.

1.1. McCulloch-Pitts neuron

McCulloch-Pitts neuron (1943) was the first mathematical model [32]. Its properties:

- neuron activity is an "all-or-none" process;
- a certain fixed number of synapses are excited within a latent addition period in order to excite a neuron: independent of previous activity and of neuron position;
- synaptic delay is the only significant delay in nervous system;
- activity of any inhibitory synapse prevents neuron from firing;
- network structure does not change along time.

The McCulloch-Pitts neuron represents a simplified mathematical model for the neuron, where x_i is the i-th binary input and w_i is the synaptic (connection) weight associated with the input x_i. The computation occurs in soma (cell body). For a neuron with p inputs:

$$a = \sum_{i=1}^{p} x_i w_i \qquad (1)$$

with $x_0 = 1$ and $w_0 = \beta = -\theta$. β is the bias and θ is the activation threshold. See figures 1 and 2. The are p binary inputs in the schema of figure 2. X_i is the i-th input, W_i is the connection (synaptic) weight associated with input i. The synaptic weights are real numbers, because the synapses can inhibit (negative signal) or excite (positive signal) and have different intensities. The weighted inputs ($X_i \times W_i$) are summed in the cell body, providing a signal a. After that, the signal a is input to an activation function (f), giving the neuron output.
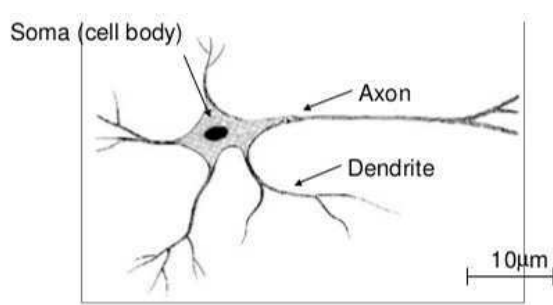

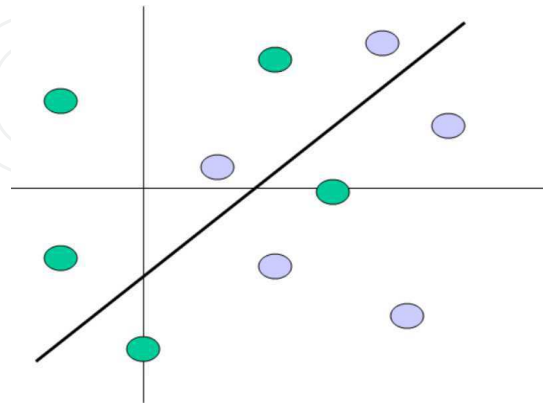**Figure 1.** The typical neuron.
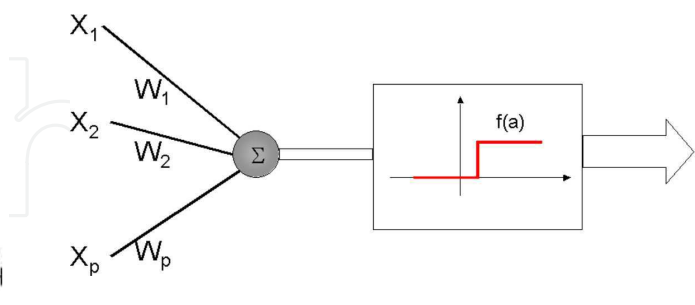
Figure 2. The neuron model.

The activation function can be: (1) hard limiter, (2) threshold logic, and (3) sigmoid, which is considered the biologically more plausible activation function.

1.2. The perceptron

Rosenblatt's perceptron [47] takes a weighted sum of neuron inputs, and sends output 1 (spike) if this sum is greater than the activation threshold. It is a linear discriminator: given 2 points, a straight line is able to discriminate them. For some configurations of *m* points, a straight line is able to separate them in two classes (figures 3 and 4).
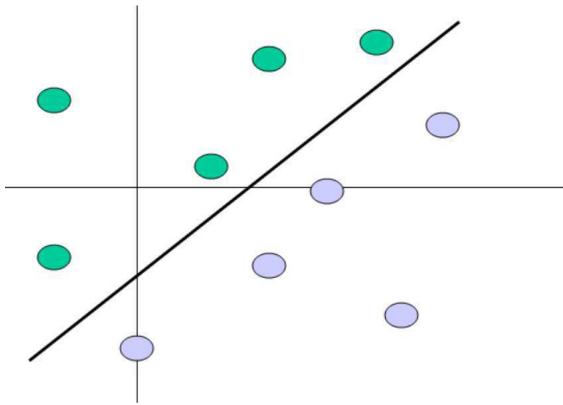
**Figure 3.** Set of linearly separable points.   **Figure 4.** Set of non-linearly separable points.

The limitations of the perceptron is that it is an one-layer feed-forward network (non-recurrent); it is only capable of learning solution of linearly separable problems; and its learning algorithm (delta rule) does not work with networks of more than one layer.

1.3. Neural network topology

In cerebral cortex, neurons are disposed in columns, and most synapses occur between different columns. See the famous drawing by Ramón y Cajal (figure 5). In the extremely simplified mathematical model, neurons are disposed in layers (representing columns), and there is communication between neurons in different layers (see figure 6).
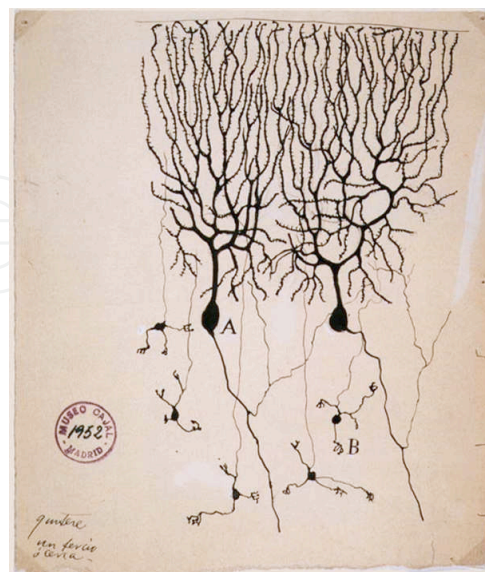
Figure 5. Drawing by Santiago Ramón y Cajal of neurons in the pigeon cerebellum. (A) denotes Purkinje cells, an example of a multipolar neuron, while (B) denotes granule cells, which are also multipolar [57].
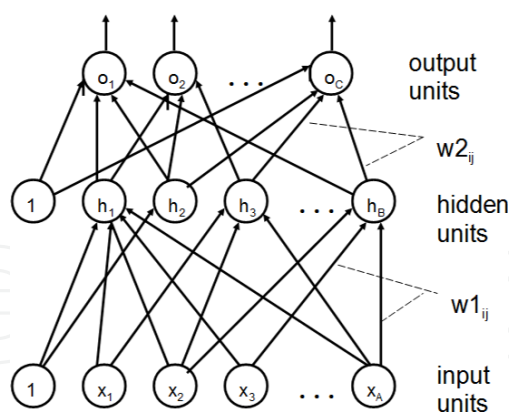
Figure 6. A 3-layer neural network. Notice that there are $A + 1$ input units, $B + 1$ hidden units, and C output units. w_1 and w_2 are the synaptic weight matrices between input and hidden layers and between hidden and output layers, respectively. The "extra" neurons in input and hidden layers, labeled 1, represent the presence of bias: the ability of the network to fire even in the absence of input signal.

1.4. Classical ANN models

Classical artificial neural networks models are based upon a simple description of the neuron, taking into account the presence of presynaptic cells and their synaptic potentials, the activation threshold, and the propagation of an action potential. So, they represent impoverished explanation of human brain characteristics.

As advantages, we may say that ANNs are naturally parallel solution, robust, fault tolerant, they allow integration of information from different sources or kinds, are adaptive systems, that is, capable of learning, they show a certain autonomy degree in learning, and display a very fast recognizing performance.

And there are many limitations of ANNs. Among them, it is still very hard to explain its behavior, because of lacking of transparency, their solutions do not scale well, and they are computationally expensive for big problems, and yet very far from biological reality.

ANNs do not focus on real neuron details. The conductivity delays are neglected. The output signal is either discrete (e.g., 0 or 1) or a real number (e.g., between 0 and 1). The network input is calculated as the weighted sum of input signals, and it is transformed in an output signal via a simple function (e.g., a threshold function). See the main differences between the biological neural system and the conventional computer on table 1.

Andy Clark proposes three types of connectionism [2]: (1) the *first-generation* consisting of perceptron and cybernetics of the 1950s. They are simple neural structures of limited applications [30]; (2) the *second generation* deals with complex dynamics with recurrent networks in order to deal with spatio-temporal events; (3) the *third generation* takes into account more complex dynamic and time properties. For the first time, these systems use biological inspired modular architectures and algorithms. We may add a fourth type: a network which considers populations of neurons instead of individual ones and the existence of chaotic oscillations, perceived by electroencephalogram (EEG) analysis. The K-models are examples of this category [30].

	Von Neumann computer	**Biological neural system**
Processor	Complex	High speed
	One or few	Simple
	Low speed	A large number
Memory	Separated from processor	Localized
	Non-content addressable	Integrated with processor
	Distributed	Content addressable
Computing	Centralized	Sequential
	Stored programs	Distributed
	Parallel	Self-learning
Reliability	Very vulnerable	Robust
Expertise	Numeric and symbolic manipulations	Perceptual problems
Operational environment	Well-defined, well-constrained	Poorly defined, unconstrained

Table 1. Von Neumann's computer versus biological neural system [26].

1.5. Learning

The Canadian psychologist Donald Hebb established the bases for current connectionist learning algorithms: "When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased" [21]. Also, the word "connectionism" appeared for the first time: "The theory is evidently a form of connectionism, one of the switchboard variety, though it does not deal in direct connections between afferent and efferent pathways: not an 'S-R' psychology, if R means a muscular response. The connections server rather to establish autonomous central activities, which then are the basis of further learning" [21].

According to Hebb, knowledge is revealed by associations, that is, the plasticity in Central Nervous System (CNS) allows synapses to be created and destroyed. Synaptic weights change values, therefore allow learning, which can be through internal self-organizing: encoding of new knowledge and reinforcement of existent knowledge. How to supply a neural substrate to association learning among world facts? Hebb proposed a hypothesis: connections between two nodes highly activated at the same time are reinforced. This kind of rule is a formalization of the associationist psychology, in which associations are accumulated among things that happen together. This hypothesis permits to model the CNS plasticity, adapting it to environmental changes, through excitatory and inhibitory strength of existing synapses, and its topology. This way, it allows that a connectionist network learns correlation among facts.

Connectionist networks learn through synaptic weight change, in most cases: it reveals statistical correlations from the environment. Learning may happen also through network topology change (in a few models). This is a case of probabilistic reasoning without a statistical model of the problem. Basically, two learning methods are possible with Hebbian learning: unsupervised learning and supervised learning. In unsupervised learning there is no teacher, so the network tries to find out regularities in the input patterns. In supervised learning, the input is associated with the output. If they are equal, learning is called auto-associative; if they are different, hetero-associative.

1.6. Back-propagation

Back-propagation (BP) is a supervised algorithm for multilayer networks. It applies the generalized delta rule, requiring two passes of computation: (1) activation propagation (forward pass), and (2) error back propagation (backward pass). Back-propagation works in the following way: it propagates the activation from input to hidden layer, and from hidden to output layer; calculates the error for output units, then back propagates the error to hidden units and then to input units.

BP has a universal approximation power, that is, given a continuous function, there is a two-layer network (one hidden layer) that can be trained by Back-propagation in order to approximate as much as desired this function. Besides, it is the most used algorithm.

Although Back-propagation is a very known and most used connectionist training algorithm, it is computationally expensive (slow), it does not solve satisfactorily big size problems, and sometimes, the solution found is a local minimum - a locally minimum value for the error function.

BP is based on the error back propagation: while stimulus propagates forwardly, the error (difference between the actual and the desired outputs) propagates backwardly. In the cerebral cortex, the stimulus generated when a neuron fires crosses the axon towards its end in order to make a synapse onto another neuron input. Suppose that BP occurs in the brain; in this case, the error must have to propagate back from the dendrite of the postsynaptic neuron to the axon and then to the dendrite of the presynaptic neuron. It sounds unrealistic and improbable. Synaptic "weights" have to be modified in order to make learning possible, but certainly not in the way BP does. Weight change must use only local information in the synapse where it occurs. That's why BP seems to be so biologically implausible.

2. Dynamical systems

Neurons may be treated as *dynamical systems*, as the main result of Hodgkin-Huxley model [23]. A dynamical system consists of a set of variables that describe its state and a law that describes the evolution of state variables with time [25]. The Hodgkin-Huxley model is a dynamical system of four dimensions, because their status is determined solely by the membrane potential V and the variable opening (activation) and closing (deactivation) of ion channels n, m and h for persistent K^+ and transient Na^+ currents [1, 27, 28]. The law of evolution is given by a four-dimensional system of *ordinary differential equations* (ODE). Principles of neurodynamics describe the basis for the development of biologically plausible models of cognition [30].

All variables that describe the neuronal dynamics can be classified into four classes according to their function and time scale [25]:

1. *Membrane potential*.
2. *Excitation variables*, such as activation of a Na^+ current. They are responsible for lifting the action potential.
3. *Recovery variables*, such as the inactivation of a current Na^+ and activation of a rapid current K^+. They are responsible for re-polarization (lowering) of the action potential.

4. *Adaptation variables*, such as the activation of low voltage or current dependent on Ca^{2+}. They build prolonged action potentials and can affect the excitability over time.

2.1. The neurons are different

The currents define the type of neuronal dynamical system [20]. There are millions of different electrophysiological spike generation mechanisms. Axons are filaments (there are 72 km of fiber in the brain) that can reach from 100 microns (typical granule cell), up to 4.5 meters (giraffe primary afferent). And communication via spikes may be stereotypical (common pyramidal cells), or no communication at all (horizontal cells of the retina). The speed of the action potential (spike) ranges from 2 to 400 km/h. The input connections ranges from 500 (retinal ganglion cells) to 200,000 (purkinje cells). In about 100 billion neurons in the human brain, there are hundreds of thousands of different types of neurons and at least one hundred neurotransmitters. Each neuron makes on average 1,000 synapses on other neurons [8].

2.2. Phase portraits

The power of dynamical systems approach to neuroscience is that we can say many things about a system without knowing all the details that govern its evolution.

Consider a quiescent neuron whose membrane potential is at rest. Since there are no changes in their state variables, it is an equilibrium point. All incoming currents to depolarize the neuron are balanced or equilibrated by hyper-polarization output currents: stable equilibrium (figure 7(a) - top). Depending on the starting point, the system may have many trajectories, as those shown at the bottom of the figure 7. One can imagine the time along each trajectory. All of them are attracted to the equilibrium state denoted by the black dot, called *attractor* [25]. It is possible to predict the itinerant behavior of neurons through observation [10].

Regarding Freeman's neurodynamics (see section 2.5) the most useful state variables are derived from electrical potentials generated by a neuron. Their recordings allow the definition of one state variable for axons and another one for dendrites, which are very different. The axon expresses its state in frequency of action potentials (pulse rate), and dendrite expresses in intensity of its synaptic current (wave amplitude) [10].

The description of the dynamics can be obtained from a study of system phase portraits, which shows certain special trajectories (equilibria, separatrices, limit cycles) that determine the behavior of all other topological trajectory through the phase space.

The excitability is illustrated in figure 7(b). When the neuron is at rest (phase portrait = stable equilibrium), small perturbations, such as *A*, result in small excursions from equilibrium, denoted by PSP (post-synaptic potential). Major disturbances, such as *B*, are amplified by the intrinsic dynamics of neuron and result in the response of the action potential.

If a current strong enough is injected into the neuron, it will be brought to a pacemaker mode, which displays periodic spiking activity (figure 7(c)): this state is called the cycle stable limit, or stable periodic orbit. The details of the electrophysiological neuron only determine the position, shape and period of limit cycle.
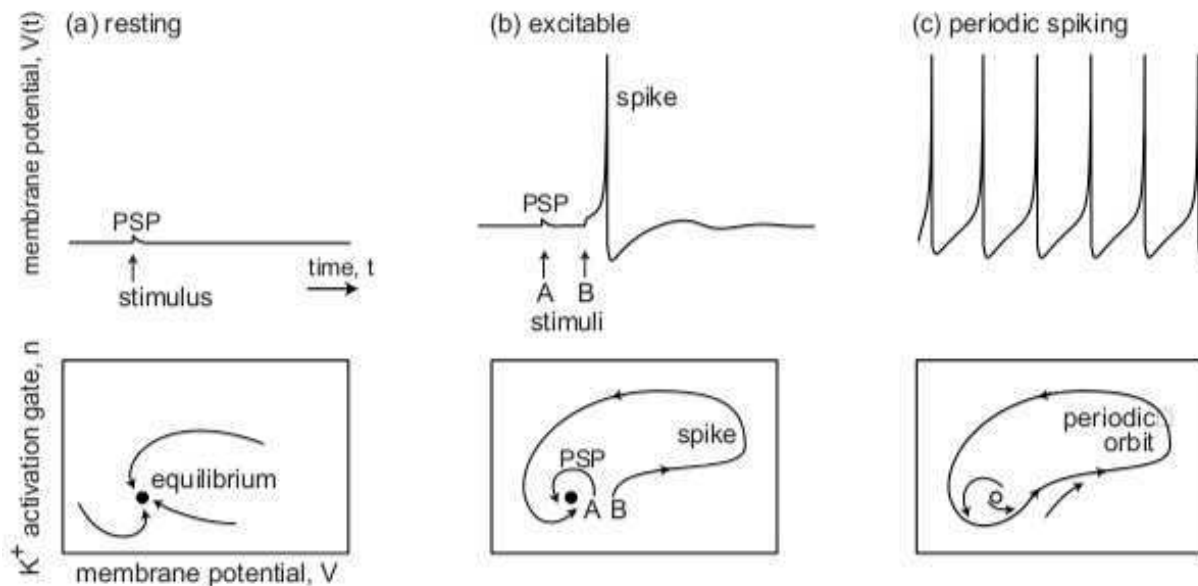
Figure 7. The neuron states: rest (a), excitable (b), and activity of periodic spiking (c). At the bottom, we see the trajectories of the system, depending on the starting point. Figure taken from [25], available at http://www.izhikevich.org/publications/dsn.pdf.
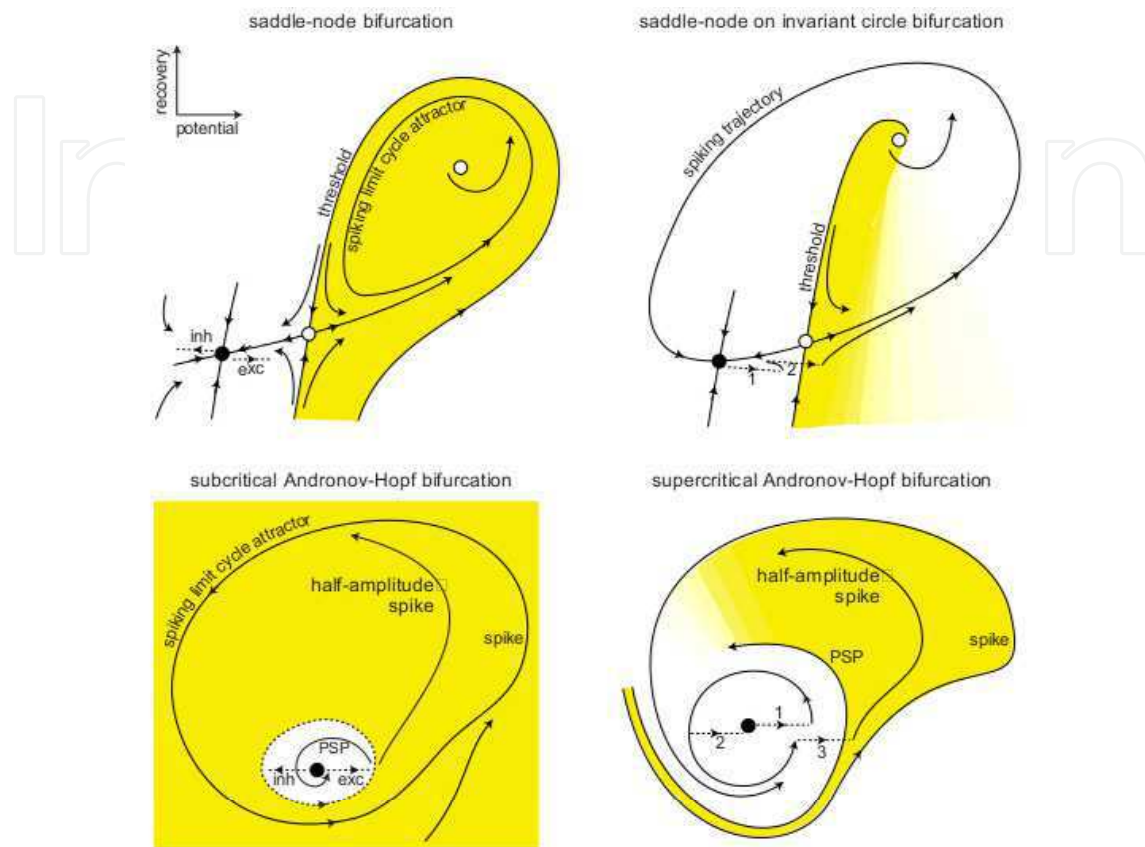
2.3. Bifurcations

Apparently, there is an injected current that corresponds to the transition from rest to continuous spiking, i.e. from the portrait phase of figure 7(b) to 7(c). From the point of view of dynamical systems, the transition corresponds to a *bifurcation* of the dynamical neuron, or a qualitative representation of the phase of the system.

In general, neurons are excitable because they are close to bifurcations from rest to spiking activity. The type of bifurcation depends on the electrophysiology of the neuron and determines its excitable properties. Interestingly, although there are millions of different electrophysiological mechanisms of excitability and spiking, there are only four different types of bifurcation of equilibrium that a system can provide. One can understand the properties of excitable neurons, whose currents were not measured and whose models are not known, since one can identify experimentally in which of the four bifurcations undergoes the rest state of the neuron [25].

The four bifurcations are shown in figure 8: saddle-node bifurcation, saddle-node on invariant circle, sub-critical Andronov-Hopf and supercritical Andronov-Hopf. In *saddle-node bifurcation*, when the magnitude of the injected current or other parameter of the bifurcation changes, a stable equilibrium correspondent to the rest state (black circle) is approximated by an unstable equilibrium (white circle). In *saddle-node bifurcation on invariant circle*, there is an invariant circle at the time of bifurcation, which becomes a limit cycle attractor. In *sub-critical Andronov-Hopf bifurcation*, a small unstable limit cycle shrinks to a equilibrium state and loses stability. Thus the trajectory deviates from equilibrium and approaches a limit cycle of high amplitude spiking or some other attractor. In the *supercritical Andronov-Hopf bifurcation*, the equilibrium state loses stability and gives rise to a small amplitude limit cycle attractor.

When the magnitude of the injected current increases, the limit cycle amplitude increases and becomes a complete spiking limit cycle [25].

Figure 8. Geometry of phase portraits of excitable systems near the four bifurcations can exemplify many neurocomputational properties. Figure taken from [25], available at http://www.izhikevich.org/publications/dsn.pdf.

Systems with Andronov-Hopf bifurcations, either sub-critical or supercritical, exhibit low amplitude membrane potential oscillations, while systems with saddle bifurcations, both without and with invariant circle, do not. The existence of small amplitude oscillations creates the possibility of resonance to the frequency of the incoming pulses [25].

2.4. Integrators and resonators

Resonators are neurons with reduced amplitude sub-threshold oscillations, and those which do not have this property are *integrators*. Neurons that exhibit co-existence of rest and spiking states, are called *bistable* and those which do not exhibit this feature are *monostable*. See table 2.

2.4.1. Neurocomputational properties

Inhibition prevents spiking in integrators, but promotes it in resonators. The excitatory inputs push the state of the system towards the shaded region of figure 8, while the inhibitory inputs push it out. In resonators, both excitation and inhibition push the state toward the shaded region [25].

sub-threshold oscillations	co-existence of rest and spiking states	
	yes (bistable)	*no* (monostable)
no (integrator)	saddle-node	saddle-node on invariant circle
yes (resonator)	sub-critical Andronov-Hopf	supercritical Andronov-Hopf

Table 2. Neuron classification in integrators-resonators/monostable-bistable, according to the rest state bifurcation. Adapted from [25].

2.5. Freeman neurodynamics

Nowadays, two very different concepts co-exist in neuroscience, regarding the way how the brain operates as a whole [55]: (1) classical model, where the brain is described as consisting of a series of causal chains composed of nerve nets that operate in parallel (the conventional artificial neural networks [20]); (2) neurodynamical model, where the brain operates by non-linear dynamical chaos, which looks like noise but presents a kind of hidden order [10].

According to Freeman [10], in order to understand brain functioning, a foundation must be laid including brain imaging and non-linear brain dynamics, fields that digital computers make possible. Brain imaging is performed during normal behavior activity, and non-linear dynamics models these data.

In a dynamicist view, actions and choices made are responsible for creation of meanings in brains, and they are different from representations. Representations exist only in the world and have no meanings. The relation of neurons to meaning is not still well understood. In Freeman's opinion, although representations can be transferred between machines, meaning cannot be transferred between brains [10]. Brain activity is directed toward external objects, leading to creation of meaning through learning. Neuron populations are the key to understand the biology of intentionality.

Freeman argues that there are two basic units in brain organization: the *neuron* and the *neuron population*. Although neuron has been the base for neurobiology, masses of interacting neurons forming neuron populations are considered for a macroscopic view of the brain. Like neurons, neuron populations also have states and activity patterns, but they do (different) macroscopic things. Between the microscopic neuron and these macroscopic things, there are *mesoscopic* populations [10].

Neurobiologists usually claim that brains process information in a cause-and-effect manner: stimuli carry information that is conveyed in transformed information. What if stimuli are selected before appearance? This view fails in this case. This traditional view allowed the development of information processing machines. This simplified, or even mistaken, view of neuronal workings, led to the development of digital computers. Artificial Intelligence artifacts pose a challenge: how to attach meaning to the symbolic representations in machines?

Pragmatists conceive minds as dynamical systems, resulted from actions into the world. How are these actions generated? According to a cognitivist view, an action is determined by the form of a stimulus. Intentional action is composed by space-time processes, called short-term

memory or cognitive maps, for materialists and cognitivists. In the pragmatism view there is no temporary storage of images and no representational map.

The neurons in the brain form dense networks. The balance of excitation and inhibition allow them to have intrinsic oscillatory activity and overall amplitude modulation (AM) [10, 55].

These AM patterns are expressions of non-linear chaos, not merely a summation of linear dendritic and action potentials. AM patterns create attractor basins and landscapes. In the neurodynamical model every neuron participates, to some extent, in every experience and every behavior, via non-linear chaotic mechanisms [10].

The concepts of non-linear chaotic neurodynamics are of fundamental importance to nervous system research. They are relevant to our understanding of the workings of the normal brain [55].

2.5.1. Neuron populations

Typical neuron have many dendrites (input) and one axon (output). The axon transmits information using *microscopic* pulse trains. Dendrites integrate information using continuous waves of ionic current. Neurons are connected by synapses. Each synapse drives electric current. The microscopic current from each neuron sums with currents from other neurons, which causes a *macroscopic* potential difference, measured with a pair of extracellular electrodes (E) as the electroencephalogram (EEG) [10, 18]. EEG records the activity patterns of *mesoscopic* neuron populations. The sum of currents that a neuron generates in response to electrical stimulus produces the post-synaptic potential. The strength of the post-synaptic potential decreases with distance between the synapse and the cell body. The attenuation is compensated by greater surface area and more synapses on the distal dendrites. Dendrites make waves and axons make pulses. Synapses convert pulses to waves. Trigger zones convert waves to pulses. See figure 9. Researchers who base their studies on single neurons think that population events such as EEG are irrelevant noise, because they do not have understanding of a mesoscopic state [10].
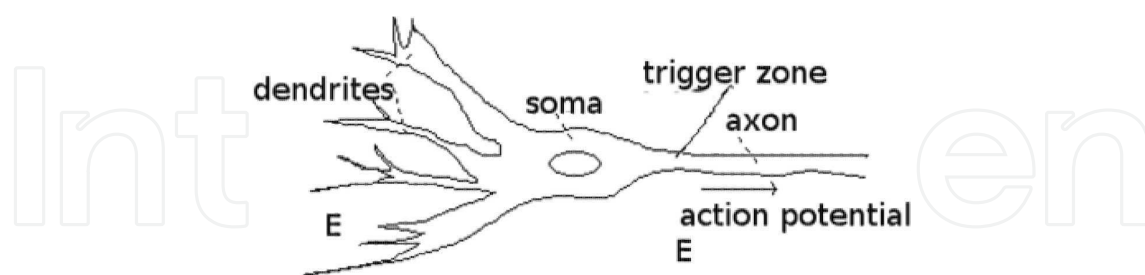
Figure 9. Typical neuron showing the dendrites (input), the soma (cell body), the axon (output), the trigger zone, and the direction of the action potential. Notice that letters "E" represent the pair of extracellular electrodes. Adapted from [45] and [10].

In single neurons, microscopic pulse frequencies and wave amplitudes are measured, while in populations, macroscopic pulse and wave densities are measured. The neuron is microscopic and ensemble is mesoscopic. The flow of the current inside the neuron is revealed by a change in the membrane potential, measured with an electrode inside the cell body, evaluating the dendritic wave state variable of the single neuron. Recall that

extracellular electrodes are placed outside the neuron (see the Es in figure 9), so cortical potential provided by sum of dendritic currents in the neighborhood is measured. The same currents produce the membrane (intracellular) and cortical (extracellular) potentials, given two views of neural activity: the former, microscopic and the latter, mesoscopic [10].

Cortical neurons, because of their synaptic interactions, form neuron populations. Microscopic pulse and wave state variables are used to describe the activity of the single neurons that contribute to the population, and mesoscopic state variables (also pulse and wave) are used to describe the collective activities neurons give rise. Mass activity in the brain is described by a pulse density, instead of pulse frequency. This is done by recording from outside the cell the firing of pulses of many neurons simultaneously. The same current that controls the firings of neurons is measured by EEG, which does not allow to distinguish individual contributions. Fortunately, this is not necessary.

A population is a collection of neurons in a neighborhood, corresponding to a cortical column, which represents dynamical patterns of activity. The average pulse density in a population can never approach the peak pulse frequencies of single neurons. The activity of neighborhoods in the center of the dendritic sigmoid curve is very near linear. This simplifies the description of populations. Neuron populations are similar to mesoscopic ensembles in many complex systems [10]. The behavior of the microscopic elements is constrained by the embedding ensemble, and it cannot be understood outside a mesoscopic and macroscopic view.

The collective action of neurons forms activity patterns that go beyond the cellular level and approach the organism level. The formation of mesoscopic states is the first step for that. This way, the activity level is decided by the population, not by individuals [10]. The population is semi-autonomous. It has a point attractor, returning to the same level after its releasing. The state space of the neuron population is defined by the range of amplitudes that its pulse and wave densities can take.

2.5.2. Freeman K-sets

Regarding neuroscience at the mesoscopic level [10, 11], theoretical connection between the neuron activity at the microscopic level in small neural networks and the activity of cell assemblies in the mesoscopic scale is not well understood [16]. Katzir-Katchalsky suggests treating cell assemblies using thermodynamics forming a hierarchy of models of the dynamics of neuron populations [29] (*Freeman K-sets*): KO, KI, KII, KIII, KIV and KV. Katzir-Katchalsky is the reason for the K in Freeman K-sets.

The KO set represents a noninteracting collection of neurons. KI sets represent a collection of KO sets, which can be excitatory (KI_e) or inhibitory (KI_i). A KII set represents a collection of KI_e and KI_i. The KIII model consists of many interconnected KII sets, describing a given sensory system in brains. A KIV set is formed by the interaction of three KIII sets [30]. KV sets are proposed to model the scale-free dynamics of neocortex operating on KIV sets [16]. See the representation of KI and KII sets by networks of KO sets in figure 10 [9].

The K-sets mediate between the microscopic activity of small neural networks and the macroscopic activity of the brain. The topology includes excitatory and inhibitory populations of neurons and the dynamics is represented by ordinary differential equations (ODE) [16].
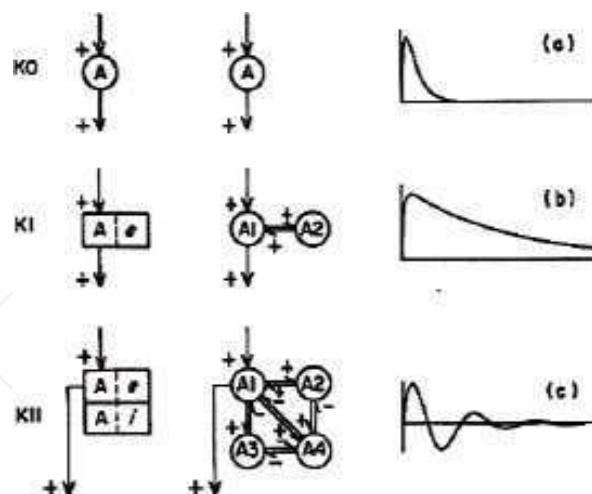
Figure 10. Representation of (b) KI and (c) KII sets by networks of (a) KO sets. Available at [9].

The advantages of KIII pattern classifiers on artificial neural networks are the small number of training examples needed, convergence to an attractor in a single step and geometric increase (rather than linear) in the number of classes with the number of nodes. The disadvantage is the increasing of the computational time needed to solve ordinary differential equations numerically.

The Katchalsky K-models use a set of ordinary differential equations with distributed parameters to describe the hierarchy of neuron populations beginning from micro-columns to hemispheres [31]. In relation to the standard KV, K-sets provide a platform for conducting analyzes of unified actions of the neocortex in the creation and control of intentional and cognitive behaviors [13].

2.5.3. Freeman's mass action

Freeman's mass action (FMA) [9] refers to collective synaptic actions neurons in the cortex exert on other neurons, synchronizing their firing of action potentials [17]. FMA expresses and conveys the meaning of sensory information in spatial patterns of cortical activity that resembles the frames in a movie [12, 13].

The prevailing concepts in neurodynamics are based on neural networks, which are Newtonian models, since they treated neural microscopic pulses as point processes in trigger zones and synapses. The FMA theory is Maxwellian because it treats the mesoscopic neural activity as a continuous distribution. The neurodynamics of the FMA includes microscopic neural operations that bring sensory information to sensory cortices and load the first percepts of the sensory cortex to other parts of the brain. The Newtonian dynamics can model cortical input and output functions but not the formation of percepts. The FMA needs a *paradigm shift*, because the theory is based on new experiments and techniques and new rules of evidence [17].

2.6. Neuropercolation

Neuropercolation is a family of stochastic models based on the mathematical theory of probabilistic cellular automata on lattices and random graphs, motivated by the structural

and dynamical properties of neuron populations. The existence of phase transitions has been demonstrated both in discrete and continuous state space models, i.e., in specific probabilistic cellular automata and percolation models. Neuropercolation extends the concept of phase transitions for large interactive populations of nerve cells [31].

Basic bootstrap percolation [50] has the following properties: (1) it is a deterministic process, based on random initialization, (2) the model always progresses in one direction: from inactive to active states and never otherwise. Under these conditions, these mathematical models exhibit phase transitions with respect to the initialization probability p. Neuropercolation models develop neurobiologically motivated generalizations of bootstrap percolations [31].

2.6.1. Neuropercolation and neurodynamics

Dynamical memory neural networks is an alternative approach to pattern-based computing [18]. Information is stored in the form of spatial patterns of modified connections in very large scale networks. Memories are recovered by phase transitions, which enable cerebral cortices to build spatial patterns of amplitude modulation of a narrow band oscillatory wave. That is, information is encoded by spatial patterns of synaptic weights of connections that couple non-linear processing elements. Each category of sensory input has a Hebbian nerve cell assembly. When accessed by a stimulus, the assembly guides the cortex to the attractors, one for each category.

The oscillating memory devices are biologically motivated because they are based on observations that the processing of sensory information in the central nervous system is accomplished via collective oscillations of populations of globally interacting neurons. This approach provides a new proposal to neural networks.

From the theoretical point of view, the proposed model helps to understand the role of phase transitions in biological and artificial systems. A family of random cellular automata exhibiting dynamical behavior necessary to simulate feeling, perception and intention is introduced [18].

2.7. Complex networks and neocortical dynamics

Complex networks are at the intersection between graph theory and statistical mechanics [4]. They are usually located in an abstract space where the position of the vertexes has no specific meaning. However, there are several network vertexes where the position is important and influences the evolution of the network. This is the case of road networks or the Internet, where the position of cities and routers can be located on a map and the edges between them represent real physical entities, such as roads and optical fibers. This type of network is called a "geographic network" or spatial network. Neural networks are spatial networks [56].

From a computational perspective, two major problems that the brain has to solve is the extraction of information (statistical regularities) of the inputs and the generation of coherent states that allow coordinated perception and action in real time [56].

In terms of the theory of complex networks [4], the anatomical connections of the cortex show that the power law distribution of the connection distances between neurons is exactly optimal to support rapid phase transitions of neural populations, regardless of how great

they are [31]. It is said that connectivity and dynamics are *scale-free* [13, 14], which states that the dynamics of the cortex is size independent, such that the brains of mice, men, elephants and whales work the same way [17].

Scale-free dynamics of the neocortex are characterized by self-similarity of patterns of synaptic connectivity and spatio-temporal neural activity, seen in power law distributions of structural and functional parameters and in rapid state transitions between levels of the hierarchy [15].

2.8. Brain-Computer Interfaces

A non-intrusive technique to allow direct *brain-computer interface* (BCI) can be a scalp EEG - an array of electrodes put on the head like a hat, which allows monitoring the cognitive behavior of animals and humans, by using brain waves to interact with the computer. It is a kind of a keyboard-less computer that eliminates the need for hand or voice interaction.

The Neurodynamics of Brain & Behavior group in the Computational Neurodynamics (CND) Lab at the University of Memphis's FedEx Institute of Technology is dedicated to research cognitive behavior of animals and humans including the use of molecular genetic or behavioral genetic approaches, to studies that involve the use of brain imaging techniques, to apply dynamical mathematical and computational models, to neuroethological studies. The research has three prongs of use for BCI: video/computer gaming; to support people with disabilities or physical constraints, such as the elderly; and to improve control of complex machinery, such as an aircraft and other military and civilian uses [24].   The direct brain-computer interface would give those with physical constraints or those operating complex machinery "extra arms" [3].

Similar to how they found seizure prediction markers, the plan is to use the data to analyze pre-motor movements, the changes in the brain that occur before there's actually movement, and apply that to someone who has a prosthetic device to allow them to better manipulate it. Since the brain is usually multitasking, the researchers will have to pick up the signal for the desired task from all the other things going on in the brain.

3. A biologically plausible connectionist system

Instead of the computationally successful, but considered to be biologically implausible supervised Back-propagation [5, 48], the learning algorithm Bio*Rec* employed in Bioθ Pred [44, 46] is inspired by the Recirculation [22] and GeneRec [33] (GR) algorithms, and consists of two phases.

In the *expectation* phase[1] (figure 11), when input x, representing the first word of a sentence through semantic microfeatures, is presented to input layer α, there is propagation of these stimuli to the hidden layer β (*bottom-up propagation*) (step 1 in figure 11). There is also a propagation of the previous actual output o^p, which is initially empty, from output layer γ back to the hidden layer β (*top-down propagation*) (steps 2 and 3).[2] Then, a hidden expectation activation (h^e) is generated (Eq. (2)) for each and every one of the B hidden units, based on

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[1] [33] employs the terms "minus" and "plus" phases to designate *expectation* and *outcome* phases respectively in his GeneRec algorithm.

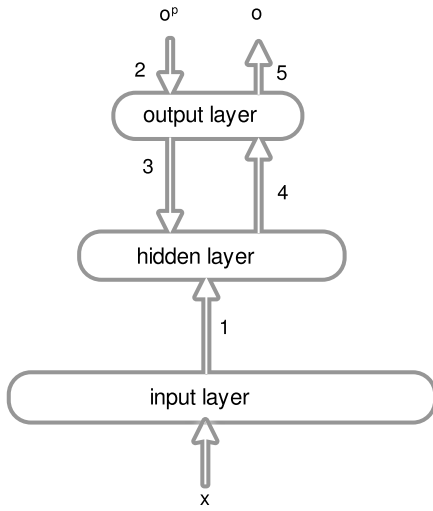[2] The superscript p is used to indicate that this signal refers to the *previous* cycle.
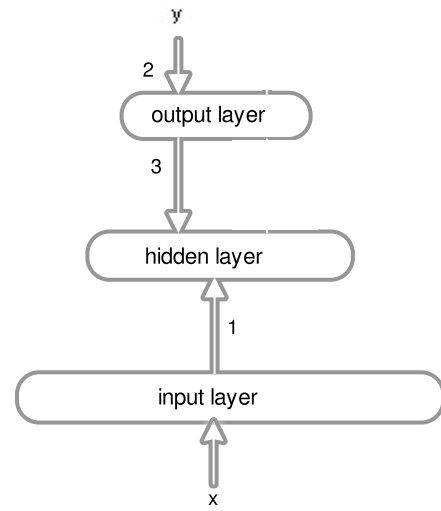
Figure 11. The expectation phase.

Figure 12. The outcome phase.

inputs and previous output stimuli o^p (sum of the bottom-up and top-down propagations - through the sigmoid logistic activation function σ). Then, these hidden signals propagate to the output layer γ (step 4), and an actual output o is obtained (step 5) for each and every one of the C output units, through the propagation of the hidden expectation activation to the output layer (Eq. (3)) [37]. w_{ij}^h are the connection (synaptic) weights between input (i) and hidden (j) units, and w_{jk}^o are the connection (synaptic) weights between hidden (j) and output (k) units[3].

$$h_j^e = \sigma(\Sigma_{i=0}^{A} w_{ij}^h.x_i + \Sigma_{k=1}^{C} w_{jk}^o.o_k^p) \qquad 1 \leq j \leq B \qquad (2)$$

$$o_k = \sigma(\Sigma_{j=0}^{B} w_{jk}^o.h_j^e) \qquad 1 \leq k \leq C \qquad (3)$$

In the *outcome* phase (figure 12), input x is presented to input layer α again; there is propagation to hidden layer β (bottom-up) (step 1 in figure 12). After this, expected output y (step 2) is presented to the output layer and propagated back to the hidden layer β (top-down) (step 3), and a hidden outcome activation (h^o) is generated, based on inputs and on expected outputs (Eq. (4)). For the other words, presented one at a time, the same procedure (*expectation* phase first, then *outcome* phase) is repeated [37]. Recall that the architecture is bi-directional, so it is possible for the stimuli to propagate either forwardly or backwardly.

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[3] i, j, and k are the indexes for the input (A), hidden (B), and output (C) units respectively. Input (α) and hidden (β) layers have an extra unit (index 0) used for simulating the presence of a bias [20]. This extra unit is absent from the output (γ) layer. That's the reason i and j range from 0 to the number of units in the layer, and k from 1. x_0, h_0^e, and h_0^h are set to $+1$. w_{0j}^o is the bias of the hidden neuron j and w_{0k}^o is the bias of the output neuron k.

$$h_j^o = \sigma(\Sigma_{i=0}^{A} w_{ij}^h.x_i + \Sigma_{k=1}^{C} w_{jk}^o.y_k) \qquad 1 \leq j \leq \text{B} \qquad (4)$$

In order to make learning possible the synaptic weights are updated through the delta rule[4] (Eqs. (5) and (6)), considering only the local information made available by the synapse. The learning rate η used in the algorithm is considered an important variable during the experiments [20].

$$\Delta w_{jk}^o = \eta.(y_k - o_k).h_j^e \qquad 0 \leq j \leq \text{B}, \qquad 1 \leq k \leq \text{C} \qquad (5)$$

$$\Delta w_{ij}^h = \eta.(h_j^o - h_j^e).x_i \qquad 0 \leq i \leq \text{A}, \qquad 1 \leq j \leq \text{B} \qquad (6)$$

Figure 13 displays a simple application to digit learning which compares BP with GeneRec (GR) algorithms.
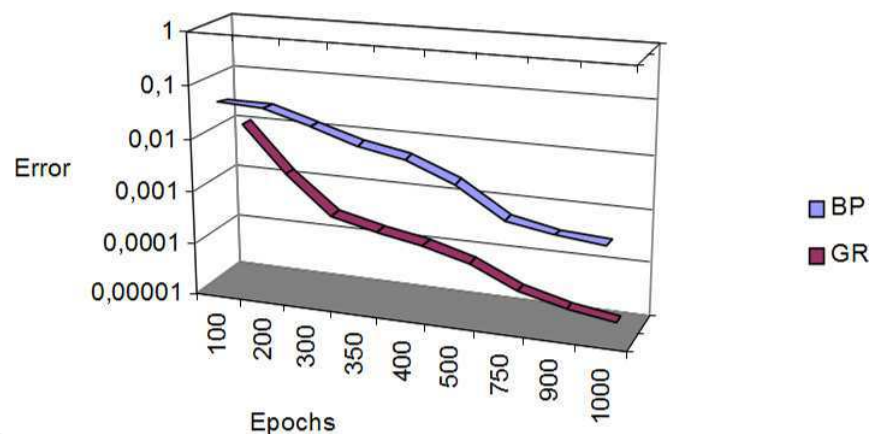
Figure 13. BP-GR comparison for digit learning.

Other applications were proposed using similar alleged biological inspired architecture and algorithm [34, 37–40, 42–44, 49].

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[4] The learning equations are essentially the delta rule (Widrow-Hoff rule), which is basically error correction: "The adjustment made to a synaptic weight of a neuron is proportional to the product of the error signal and the input signal of the synapse in question." ([20], p. 53).

3.1. Intraneuron signaling

The Spanish Nobel laureate neuroscientist Santiago Ramón y Cajal, established at the end of the nineteenth century, two principles that revolutionized neuroscience: the Principle of connectional specificity, which states that "nerve cells do not communicate indiscriminately with one another or form random networks;" and the Principle of dynamic polarization, which says "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." Intraneuron signalling is based on the principle of dynamic polarization. The signaling inside the neuron is performed by four basic elements: receptive, trigger, signaling, and secretor. The Receptive element is responsible for input signals, and it is related to the dendritic region. The Trigger element is responsible for neuron activation threshold, related to the soma. The Signaling element is responsible for conducting and keeping the signal and its is related to the axon. And the Secretor element is responsible for signal releasing to another neuron, so it is related to the presynaptic terminals of the biological neuron.

3.2. Interneuron signaling

Electrical and chemical synapses have completely different morphologies. At electrical synapses, transmission occurs through gap junction channels (special ion channels), located in the pre and postsynaptic cell membranes. There is a cytoplasmatic connection between cells. Part of electric current injected in presynaptic cell escapes through resting channels and remaining current is driven to the inside of the postsynaptic cell through gap junction channels. At chemical synapses, there is a synaptic cleft, a small cellular separation between the cells. There are vesicles containing neurotransmitter molecules in the presynaptic terminal and when action potential reaches these synaptic vesicles, neurotransmitters are released to the synaptic cleft.

3.3. A biologically plausible ANN model proposal

We present here a proposal for a biologically plausible model [36] based on the microscopic level. This model in intended to present a mechanism to generate a biologically plausible ANN model and to redesign the classical framework to encompass the traditional features, and labels that model the binding affinities between transmitters and receptors. This model departs from a classical connectionist model and is defined by a restricted data set, which explains the ANN behavior. Also, it introduces T, R, and C variables to account for the binding affinities between neurons (unlike other models).

The following feature set defines the neurons:

$$N = \{\{w\}, \theta, g, T, R, C\} \tag{7}$$

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 encode the genetic information, while T, R, and C are the labels, absent in other models. This proposal follows Ramón y Cajal's principle of connectional specificity, that states that each neuron is connected to another neuron not only in relation to $\{w\}$, θ, and g, but also in relation to T, R, and C; neuron i is only connected to neuron j if there is binding affinity between the T of i and the R of j. Binding affinity means compatible types, enough amount of substrate, and compatible genes. The combination of T and R results in C: C can act over other neuron connections.

The ordinary biological neuron presents many dendrites usually branched, which receive information from other neurons, an axon, which transmits the processed information, usually by propagation of an action potential. The axon is divided into several branches, and makes synapses onto the dendrites and cell bodies of other neurons (see figure 14). Chemical synapse is predominant is the cerebral cortex, and the release of transmitter substance occurs in active zones, inside presynaptic terminals. Certain chemical synapses lack active zones, resulting in slower and more diffuse synaptic actions between cells. The combination of a neurotransmitter and a receptor makes the postsynaptic cell releases a protein.
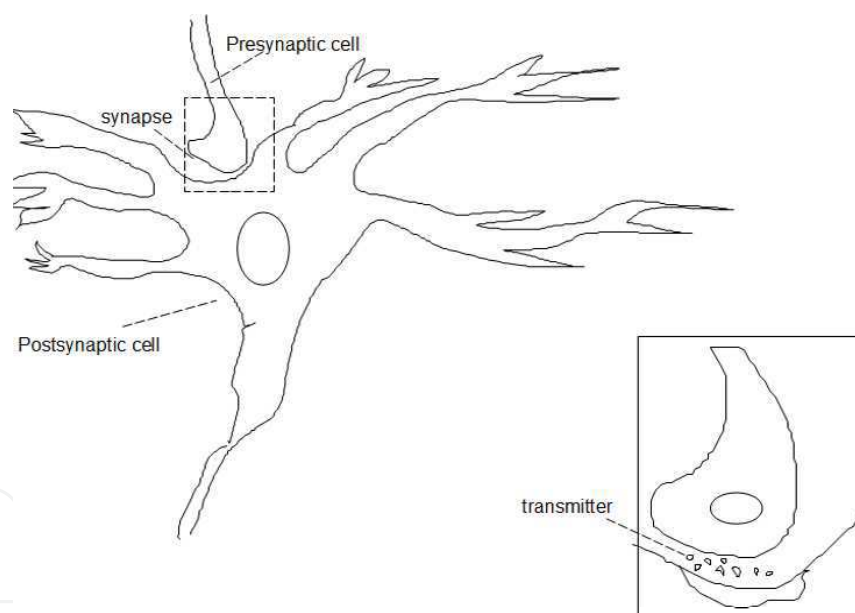

Figure 14. The chemical synapse. Figure taken from [45].

Although type I synapses seem to be excitatory and type II synapses inhibitory (see figure 15), the action of a transmitter in the postsynaptic cell does not depend on the chemical nature of the neurotransmitter, instead it depends 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 an ion channel will be activated directly by the transmitter or indirectly through a second messenger.

Neurotransmitters are released by presynaptic neuron and they combine with specific receptor in membrane of postsynaptic neuron. The combination of neurotransmitter with
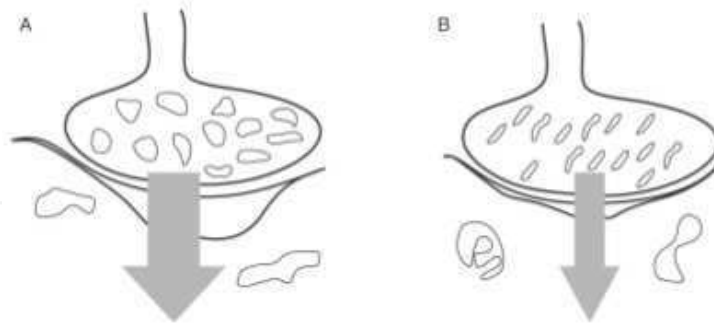
Figure 15. Morphological synapses type A and type B. In excitatory synapse (type A), neurons contribute to produce impulses on other cells: asymmetrical membrane specializations, very large synaptic vesicles (50 nm) with packets of neurotransmitters. In inhibitory synapse (type B), neurons prevent the releasing of impulses on other cells: symmetrical membrane specializations, synaptic vesicles are smaller and often ellipsoidal or flattened, contact zone usually smaller. Figure taken from [45].

receptor leads to intracellular release or production of a second messenger, which interacts (directly or indirectly) with ion channel, causing it to open or close. There are two types of resulting signaling : (1) propagation of action potential, and (2) production of a graded potential by the axon. Graded potential signaling does not occur over long distances because of attenuation.

Graded potentials can occur in another level. See, for instance, figure 16. Axon 1 making synapse in a given cell can receive a synapse from axon 2. Otherwise, the presynaptic synapse can produce only a local potential change, which is then restricted to that axon terminal (figure 17).
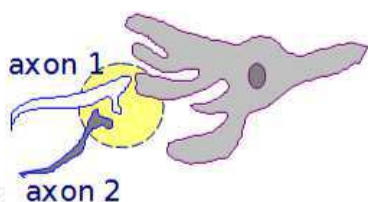
Figure 16. An axon-axon synapse [6].
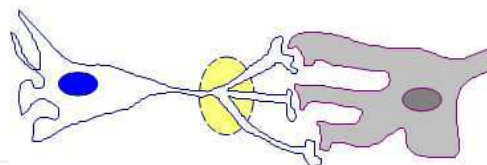
Figure 17. A local potential change [6].

In view of these biological facts, it was decided to model through labels T and R, the binding affinities between Ts and Rs. And label C represents the role of the "second messenger,", the effects of graded potential, and the protein released by the coupling of T and R.

Controller C can modify the binding affinities between neurons by modifying the degrees of affinity of receptors, the amount of substrate (amount of transmitters and receptors), and gene expression, in case of mutation. The degrees of affinity are related to the way receptors gate ion channels at chemical synapses. Through ion channels transmitter material enters the postsynaptic cell: (1) in direct gating: receptors produce relatively fast synaptic actions, and (2) in indirect gating: receptors produce slow synaptic actions: these slower actions often serve to modulate behavior because they modify the degrees of affinity of receptors.

In addition, modulation can be related to the action of peptides[5]. There are many distinct peptides, of several types and shapes, that can act as neurotransmitters. Peptides are different from many conventional transmitters, because they "modulate" synaptic function instead of activating it, they spread slowly and persist for some time, much more than conventional transmitters, and they do not act where released, but at some distant site (in some cases).

As transmitters, peptides act at very restricted places, display a slow rate of conduction, and do not sustain the high frequencies of impulses. As neuromodulators, the excitatory effects of substance P (a peptide) are very slow in the beginning and longer in duration (more than one minute), so they cannot cause enough depolarization to excite the cells; the effect is to make neurons more readily excited by other excitatory inputs, the so-called "neuromodulation." In the proposed model, C explains this function by modifying the degrees of affinity of receptors.

In biological systems, the amount of substrate modification is regulated by the acetylcholine (a neurotransmitter). It spreads over a short distance, toward the postsynaptic membrane, acting at receptor molecules in that membrane, which are enzymatically divided, and part of it is taken up again for synthesis of a new transmitter. This will produce an increase in the amount of substrate. In this model, C represents substrate increase by a variable acting over initial substrate amount.

Peptides are a second, slower, means of communication between neurons, more economical than using extra neurons. This second messenger, besides altering the affinities between transmitters and receptors, can regulate gene expression, achieving synaptic transmission with long-lasting consequences. In this model, this is achieved by modification of a variable for gene expression, mutation can be accounted for.

3.3.1. The labels and their dynamic behaviors

In order to build the model, it is necessary to set the parameters for thew connectionist architecture. For the network genesis, the parameters are:

- number of layers;
- number of neurons in each layer;
- initial amount of substrate (transmitters and receptors) in each layer; and
- 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)).

For the evaluation of controllers and how they act, the parameters are:

- Controllers can modify:
    - the degree of affinity of receptors;
    - the initial substrate storage; and
    - the gene expression value (mutation).

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[5] Peptides are a compound consisting of two or more amino acids, the building blocks of proteins.

The specifications stated above lead to an ANN with some distinctive characteristics: (1) each neuron has a genetic code, which is a set of genes plus a gene expression controller; (2) the controller can cause mutation, because it can regulate gene expression; (3) the substrate (amount of transmitter and receptor) is defined by layer, but it is limited, so some postsynaptic neurons are not activated: this way, the network favors clustering.

Also, 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) [36]. 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.

3.3.2. A network simulation

In table 3, a data set for a five-layer network simulation is presented [36]. For the specifications displayed in table 3, the network architecture and its activated connections are shown in figure 18. 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).

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: *res* = new degree of affinity of receptor (target), for *t* = s: *res* = substrate increasing (origin), for *t* = e: *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.)

Table 3. The data set for a five-layer network. Adapted from [36].

In figure 18, 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.

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
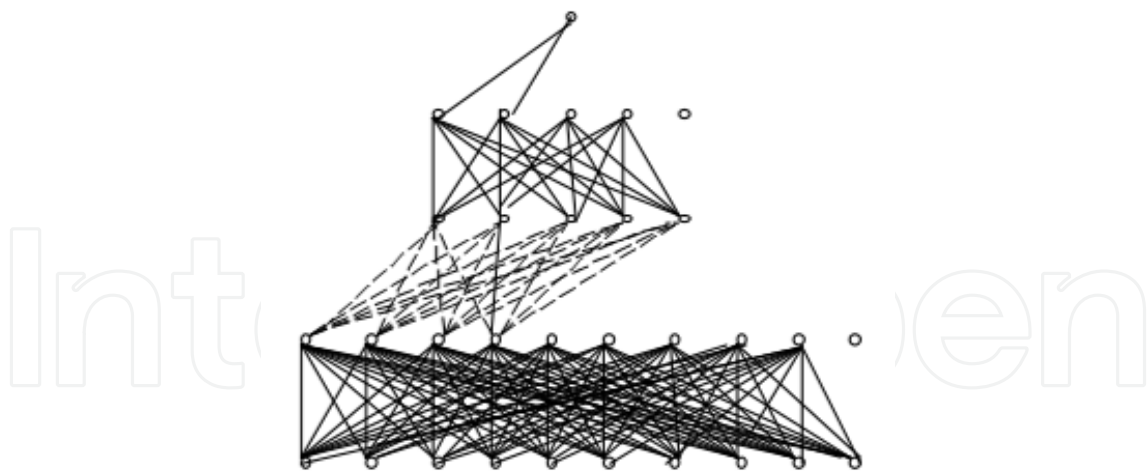
Figure 18. A five-layer neural network for the data set in table 3. In the bottom of the figure is the layer 1 (input layer) and in the top is the layer 5 (output layer). Between them, there are three hidden layers (layers 2 to 4). Figure taken from [36].

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.

Although the architecture shown in figure 18 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 [7]. 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.

3.4. Other models

Other biological plausible ANN models are concerned with the connectionist architecture; related directly to the cerebral cortex biological structure, or focused on the neural features and the signaling between neurons. Always, the main purpose is to create a more faithful model concerning the biological structure, properties, and functionalities, including learning processes, of the cerebral cortex, not disregarding its computational efficiency. The choice of the models upon which the proposed description is based takes into account two main criteria: the fact they are considered biologically more realistic and the fact they deal with intra and inter-neuron signaling in electrical and chemical synapses. Also, the duration of action potentials is taken into account. In addition to the characteristics for encoding information regarding biological plausibility present in current spiking neuron models, a distinguishable feature is emphasized here: a combination of Hebbian learning and error driven learning [52–54].

4. Conclusions

Current models of ANN are in debt with human brain physiology. Because of their mathematical simplicity, they lack several biological features of the cerebral cortex. Also, instead of the individual behavior of the neurons, the mesoscopic information is privileged. The mesoscopic level of the brain could be described adequately by dynamical system theory (attractor states and cycles). The EEG waves reflect the existence of cycles in brain electric field. The objective here is to present biologically plausible ANN models, closer to human brain capacity. In the model proposed, still at the microscopic level of analysis, 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 ANN would be closer to human evolutionary capacity, that is, it would represent a genetically well-suited model of the brain. The hypothesis of the "dynamic core" [7] is also contemplated, that is, the model allows reentrancy in its architecture connections.

Acknowledgements

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