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Neural Networks for Computational Neuroscience

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Abstract.
Computational neuroscience is an appealing interdisciplinary domain, at the interface between biology and computer science. It aims at understanding the experimental data obtained in neuroscience using several different kinds of models, one of which being artificial neural networks.

In this tutorial we review some of the advances neural networks have achieved in computational neuroscience, and in particular focusing on spiking neural networks. Several artificial neuron models, that are able to account for the temporal properties of biological neurons, are described. We also describe briefly data obtained using conventional neuroscience methods, and some artificial neural networks developed to understand the mechanisms underlying these experimental data.

1 Introduction

Computational neuroscience aims at creating models from experimental data obtained in neurophysiology and neuro-imaging experiments. There exists a wide range of symbolic supports for modelling, including Markov chains, bayesian networks, mean-field equations, and oscillator synchronisation. The present tutorial focuses on models based on artificial neural networks, and in particular, on spiking neuron networks (SNN).

Several models illustrate the classification we propose, starting from microscopic properties: single neuron behaviour, to mesoscopic properties: behaviour of a population of neurons, in a cortical column or in a brain area, and finally to macroscopic properties: observable behaviour of the brain, through one or other cognitive process.

2 Microscopic models

2.1 Computational properties of single biological neurons

One of the key problems in computational neuroscience is defining the operations that represent single neurons. Neurons have been considered to encode their outputs by their average firing rates. As an example, Hubel & Wiesel's experiments [1] proved the specificity of primary visual cortex (V1) neurons for a prefered

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orientation. For example, such a behaviour of biological neurons is reproduced by the threshold or sigmoidal neuron models, in multi-layer perceptrons.

However in the late 80's, some experiments raised doubts on this behaviour of single neurons. In a seminal paper, Gray and Singer [2] showed that correlations between spike timings of primary visual cortex of the cat are related to the nature of presented stimulus. If two stimuli share common properties (e.g. a common motion), then the spike timings of the neurons in different visual fields are correlated. However, the correlation does not appear if the stimuli have different properties e.g. different motions.

Another article by Thorpe and Imbert [3], based on theoretical assumptions, also raised some doubt regarding rate coding in the nervous system. From experimental data, Thorpe and Imbert showed that the speed of computation in the visual system is too fast to allow the integration of more than one spike, at each processing step. Since all spikes share the same properties of shape and temporal course, only the *timing* of spikes can bear relevant information.

In the 90's, several authors studied the computational properties of spike timing. Considering random incoming signals, for instance gaussian noise generated signals, balanced between excitatory and inhibitory PSPs [1], they observed that spike timing encodes a significant higher proportion of excitatory PSPs incoming simultaneously at the neuron soma [4]. It may be that there exists a code based on synchrony detection, and acting on a very short time-scale, that acts in complement with the rate-based code [5].

Computational properties of a neuron also depend on its physiological parameters. Using a temporal neuron model, König et al. [6] showed that, depending on the time constant of the voltage leak of a neuron, a single neuron can act either as a temporal integrator or as a coincidence detector. A temporal integrator behaviour means that the neuron fires a spike whenever a given count of excitatory PSPs has reached the neuron, whatever the timing of their arrivals. When the neuron acts as a coincidence detector, the neuron fires a spike when several EPSPs (even a small number) reach simultaneously the neuron soma.

Destexhe et al. [7] have shown that a model neuron, randomly reached by a very high number of inputs, can behave exactly as observed in vivo. They show that the model reproduces the amplitude and spectral properties observed in experiments. Furthermore, the presence of background activity can enhance the responsiveness of a neuron.

All these observations have raised the possibility of developing several artificial neuron models, where time is an intrinsic property.

2.2 Temporal models of neurons

2.2.1 Hodgkin-Huxley model

The fathers of the spiking neurons are the conductance-based models, such as the electrical model defined by Hodgkin and Huxley [8, 9] in 1952. The basic

[1] PSPs = Post-Synaptic Potentials

idea is to model electro-chemical information transmission of natural neurons by electric circuits made of capacitors and resistors: C is the capacitance of the membrane, the g_i are the conductance parameters for the different ion channels (sodium Na, potassium K, etc.) and the E_i are the corresponding equilibrium potentials. Variables m, h and n describe the opening and closing of the voltage dependent channels.

$$C\frac{du}{dt} = -g_{Na}m^3h(u - E_{Na}) - g_K n^4(u - E_K) - g_L(u - E_L) + I(t) \qquad (1)$$

$$\tau_n\frac{dn}{dt} = -[n - n_0(u)] \qquad \tau_m\frac{dm}{dt} = -[m - m_0(u)] \qquad \tau_h\frac{dh}{dt} = -[h - h_0(u)]$$

The **Hodgkin-Huxley model** (HH) is realistic but far too complex for the simulation of SNNs. Although ODE[2] solutions can be applied directly to the system of HH differential equations, it would be intractable to compute temporal interactions between neurons in a large network of Hodgkin-Huxley models.

The HH model has been compared successfully - with appropriate calibration of parameters - to numerous data from biological experiments on the giant axon of the squid. More generally, the HH model is able to model biophysically meaningful variations of the membrane potential, as recorded from neurons in-vivo: An abrupt, high increase at firing time, followed by a short time where the neuron is unable to spike again, the *absolute refractoriness*, and a further time range where the membrane is underpolarized, which makes a new firing more difficult, i.e. the *relative refractory period*.

2.2.2 Integrate-and-Fire model

More tractable models are the **Integrate-and-Fire** (I&F) neurons ([10] cited by [11]). The most important simplification implies that the shape of the action potentials is neglected and every spike is considered as a uniform event defined only by the time of its appearance. The basic circuit consists of a capacitor C in parallel with a resistor R driven by an input current $I(t)$. The dynamics of the membrane potential can be described by a single first-order linear differential equation: $RC\frac{du}{dt} = RI$. Defining the time constant of the neuron membrane as $\tau_m = RC$, for modelling the voltage leakage, the usual formula for the **Leaky Integrate-and-Fire** neuron (LIF), can be written as follows:

$$\tau_m\frac{du}{dt} = u_{rest} - u(t) + RI(t) \qquad (2)$$

In addition, the firing time $t^{(f)}$ of the neuron is defined by a threshold crossing equation $u(t^{(f)}) = \vartheta$, under the condition $u'(t^{(f)}) > 0$. Immediately after $t^{(f)}$, the potential is reset to a given value u_r. An absolute refractory period can be modelled by forcing the neuron to a value $u = -u_{abs}$ during a time d_{abs} after a spike emission, and then restarting the integration with initial value $u = u_r$.

[2]ODE = Ordinary Differential Equations

There exist many variations between the HH and LIF models, with decreasing biophysical plausibility, but also with decreasing computational cost (see [12] for a review) for in-depth comparison of HH and LIF subthreshold dynamics).

Note that the same neuron cannot be simultaneously an "integrator" and "resonator" since the properties are mutually exclusive, but the same neuron model can simulate all of them, with different choices of parameters. In the class of spiking neurons controlled by differential equations, the two-dimensional **Izhikevich neuron model** [13] defined by the coupled equations

$$\frac{du}{dt} = 0.04u(t)^2 + 5u(t) + 140 - w(t) + I(t) \qquad \frac{dw}{dt} = a\left(bu(t) - w(t)\right) \quad (3)$$

with after-spike resetting: if $u \geq \vartheta$ then $u \leftarrow c$ and $w \leftarrow w + d$

is a good compromise between biophysical plausibility and computational cost.

2.2.3 Spike Response Model

More simple to understand and to implement is the **Spike Response Model** (SRM) defined by Gerstner et al. [14, 15]. The model expresses the membrane potential u at time t as an integral over previous times, including a model of refractoriness, but without differential equations. SRM is a phenomenological model of a neuron, based on the occurence of spike emissions.

Let $\mathcal{F}_j = \{t_j^{(f)}; 1 \leq f \leq n\} = \{t \mid u_j(t) = \vartheta \ \wedge \ u_j'(t) > 0\}$ denote the set of all firing times of neuron N_j, and $\Gamma_j = \{i \mid N_i \text{ is presynaptic to } N_j\}$ define its set of presynaptic neurons. The state $u_j(t)$ of neuron N_j at time t is given by

$$u_j(t) = \sum_{t_j^{(f)} \in \mathcal{F}_j} \eta_j\left(t - t_j^{(f)}\right) + \sum_{i \in \Gamma_j} \sum_{t_i^{(f)} \in \mathcal{F}_i} w_{ij}\epsilon_{ij}\left(t - t_i^{(f)}\right) + \underbrace{\int_0^\infty \kappa_j(r)I(t-r)dr}_{\text{if external input current}} \quad (4)$$

with the following kernel functions: η_j is non-positive for $s > 0$ and models the potential reset after a spike emission, ϵ_{ij} describes the response to presynaptic spikes, and κ_j describes the response of the membrane potential to an external input current.

Kernel ϵ_{ij} describes the generic response of a neuron N_j to spikes coming from presynaptic neurons N_i. For the sake of simplicity, $\epsilon_{ij}(s)$ can be assumed to have the same form $\epsilon(s - d_{ij}^{ax})$ for any pair of neurons, only modulated in amplitude and sign by the weight w_{ij} (excitatory EPSP for $w_{ij} > 0$, inhibitory IPSP for $w_{ij} < 0$).

A short term memory variant of SRM results from assuming that only the last firing $\hat{t}_j$ of N_j contributes to refractoriness, $\eta_j\left(t - \hat{t}_j\right)$ replacing the sum in formula (4). Moreover, integrating the equation on a small time window of $1ms$ and assuming that each presynaptic neuron emits at most once in the time window (reasonable since refractoriness of presynaptic neurons), we obtain the again simplified formula of model $\mathbf{SRM}_0$, which is very close to the actual expression of neural activity, in rate coding

$$v_j(t) = \sum_{i \in \Gamma_j} w_{ij}\epsilon(t - \hat{t}_i - d_{ij}^{ax}) \quad (5)$$

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$$\text{with next firing time} \quad t_j^{(f+1)} = t \Longleftrightarrow v_j(t) = \vartheta - \underbrace{\eta_j\left(t - \hat{t}_j\right)}_{threshold\ kernel}$$

Despite its simplicity, the Spike Response Model is more general than the Integrate-and-Fire neuron and is often able to compete with the Hodgkin-Huxley model for simulating complex neuro-computational properties.

2.3 Synaptic plasticity

From the early work presented by Hebb in 1949 [16], **synaptic plasticity** has been the main basis of learning rules, by weight updating in artificial neural networks. However Hebb's ideas are poorly exploited by most of the current algorithms [3].

Novel tracks for setting algorithms that control the synaptic plasticity are derived from both a deeper understanding of Hebb's lesson and from a bank of recent results in neuroscience, following the advances of experimental technology. Innovative principles are often referred as **temporal Hebbian rules**. In the biological context of natural neurons, the changes of synaptic weights with effects lasting several hours are referred as LTP[3] if the weight values (also named *efficacies*) are strengthened, and LTD if the weight values are decreased. In the second or minute timescales, the weight changes are designed by STP and STD[4].

A good review of the main synaptic plasticity mechanisms for regulating levels of activity in conjunction with Hebbian synaptic modification has been developed by Abbott and Nelson in [17]:

- *Synaptic scaling:* For example, cortical neurons actively maintain an average firing rate by scaling their incoming weights. Synaptic scaling is multiplicative, in the sense that synaptic weights are changed by an amount proportional to their strength, and not all by the same amount (additive / subtractive adjustment).

- *Synaptic redistribution:* Markram and Tsodyks' experiments [18] have critically challenged the conventional assumption that LTP reflects a general gain increase. The phenomenon of "Redistribution of Synaptic Efficacy" (RSE) designs the change in frequency dependence they have observed during synaptic potentiation. Synaptic redistribution could enhance the amplitude of synaptic transmission for the first spikes in a sequence, but with transient effect only.

- *Spike-timing dependent synaptic plasticity*[5]*:* **STDP** is far from being the most popular synaptic plasticity rule (first related articles [19, 20, 15]). STDP is a form of Hebbian synaptic plasticity sensitive to the precise

[3]LTP = Long Term Potentiation - LTD = Long Term Depression
[4]STP = Short Term Potentiation - STD = Short Term Depression
[5]STDP = Spike-Time Dependent Plasticity

timing of spike emission. It relies on local information driven by backpropagation of the timing of action potential through the dendrites of the postsynaptic neuron. The type and amount of long-term synaptic modification induced by repeated pairing of pre- and postsynaptic action potential as a function of their relative timing varies from an experiment to another, in neuroscience. In a computational model, however, a basic principle has emerged: A maximal increase of synaptic efficacy occurs on a connection when the presynaptic neuron fires a short time before the postsynaptic neuron, whereas a late presynaptic spike (just after the postsynaptic firing) leads to a decrease in the weight. If the two spikes (pre- and post-) are very distant in time, then the weight remains unchanged. This form of potentiation/depression timing dependency reflects a form of causal relationship in information transmission through action potentials.

Intrinsic plasticity is another form of synaptic plasticity that has been recently introduced in spiking neuron network models [21]. Intrinsic plasticity is a persistent modification of a neuron's intrinsic electrical properties by neuronal or synaptic activity. It is mediated by changes in the expression level or biophysical properties of ion channels in the membrane, and can affect diverse processes, like synaptic integration, subthreshold signal propagation, spike generation, spike backpropagation, and meta-plasticity.

3 Mesoscopic models

The mesoscopic level includes models reproducing the behaviour of a given cerebral structure, and modelling the properties of the neurons in this structure. The purpose is to describe how a collective behaviour, observed at this level by neuroscientists, can emerge from the interaction between populations of neurons, starting from the behaviour of single neurons.

3.1 Perception

3.1.1 Fast visual processing

Experiments using human subjects [22] have shown that even the processing of complex pictures can be achieved extremely quickly. For pictures shown only a very short presentation time 50ms, the subjects are still able to achieve a very simple categorization i.e. is there an animal or not in the picture, faster than 150ms. "SpikeNet" model [23] reproduces experimental observations with a physiologically plausible model. In this model, the processing is based on rank-order coding, taking into account the timing of the first spikes only. With only one percent of neurons of the higher-level layer firing a spike, the system is already able to recognize the rough shape of the picture.

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3.1.2 Temporal binding

Feature binding is a problem studied by psychologists [24, 25], where the fact that different attributes of the same object are processed in different areas of the nervous system e.g. shape in area V1 and V2, and colour in area V4. The feature binding problem can be formulated in the following way: "At a higher level, how are the different attributes of a same object processed as belonging to the same object, without confusion, if several objects are displayed together?".

The temporal binding hypothesis addresses this problem: the spike timings of the neurons processing attributes of the same object are correlated, and thus the system is able to tag them as being part of the same object [26, 27]. This hypothesis has received a number of experimental confirmations, in both animals [2, 28] and humans [29].

Several models have been built to address this problem. In [30], the different areas where the attributes are processed, are linked to higher processing levels by means of bidirectional connections. Watanabe et al. show that the system is able to maintain correlated activity between the different levels of processing, due to reverberating activities. Cell assemblies are measured by means of functional connectivity: a stimulus presentation induces the formation of a dynamic cell assembly, with a composition that may vary in time, without being completely determined by the underlying anatomical connectivity.

3.2 Activity persistence

In [31], Amit and Brunel study the conditions of formation of a stimulus-dependent cell assembly being able to self maintain in the system after the stimulus has disappeared. The authors show that the persistent activity is due to the selection of transmission delays between neurons. If the synaptic plasticity, which is responsible for this selection, is not strong enough, the stimulus-induced activity is unable to be persistent. Furthermore, the selective formation of the cell assembly is faster when the network displays background activity before the presentation of the stimulus (compared to the situation where there is no activity at all). The spontaneous activity thus allows the system to be stronger reactive to processed inputs.

Following the previous work, Mongillo et al. [32] were interested in the persistent activity occuring in infero-temporal cortex of the monkey, as observed experimentally by Nakamura and Kubota [33]. In the working memory task, the monkey has to remember a given face during a time period, and to compare it with another face presented after this time. Experimental data showed that during the time period of remembering, the neurons keep a high level of activity, even in the presence of distractors during the time range. Mongillo et al. describe a model that can produce these experimental results. The persistent activity is increased as the number of trials increases, and is related to the ratio of fast (AMPA receptors) and slow (NMDA receptors) synaptic plasticity. If the

recurrent excitation is fast, the transition is abrupt. If the recurrent excitation is slow, then the synaptic plasticity increases gradually, reproducing experimental data.

3.3 Oscillations

Oscillations have been used to study aspects in neuroscience, since oscillations are supposed to be the code allowing several structures in the brain to interact [34, 35]. The way oscillations are generated have been studied by computational models.

One way to describe the emergence of oscillations is to study the conditions under which oscillations appear. Therefore, neuron parameters can be studied using a mean-field approach: each element is considered to be stochastically linked with the others, and the behaviour of the whole population is studied via the average behaviour of single elements. Some models reproduce the mechanisms of oscillation generation in a given brain structure, e.g. rat hippocampus [36, 37, 38, 39, 40], insect antennal lobe [41, 42]. These models describe interactions inside a population of neurons [43, 44, 36], or between several populations with different properties - usually, one population of excitatatory neurons and one population of inhibitory neurons [37, 39, 40].

For instance, Whittington et al. [37] describe a model that is able to explain the co-existence of "gamma" (30-80Hz) and "beta" (10-20 Hz) oscillations in CA1, one structure of the rat hippocampus. The authors show that the introduction of synaptic plasticity makes the system able to maintain a "beta" oscillation when the system is stimulated. This oscillation disappears in absence of stimulation, corresponding to what is observed experimentally.

In [40], Brunel et al. describe a model reproducing very closely the properties of the neurons in the rat hippocampus. They show that the average activity of the population can achieve a rythmic oscillation (close to "gamma" oscillations), although each neuron has itself a very low activity (about 2Hz). This work is a good example of how models can help in solving some apparently antagonist experimental observations (high average activity and low single neuron activity).

4 Macroscopic models

In mesoscopic models described in Section 3, the influences coming from other structures are modelled by means of stochastic spikes (e.g., following a Poisson distribution). There is a paradox in these models: why the spike timings inside the structure should be completely determined, whereas the spikes coming from outside the structure are random? The only way to overcome this problem would be to build a model including all the structures interacting in the nervous system. However building, and simulating on a computer, a model of the whole nervous system, with comparable numbers of neurons, is at present clearly unrealistic.

4.1 *A priori* models

Some models have been designed for simulating large-scale interactions between distinct areas of the nervous system. For example, thalamo-cortical interactions have been simulated [45, 46], to study the conditions of emergence of gamma oscillations and the way lesions in various parts of the system may alter the gamma oscillation generation.

In [47], Krichmar and Edelman have designed a biologically-inspired system to control a robot interacting with the environment. The model is composed of two perceptual systems (audition and vision), a memory system, and a motor system. The system as a whole implements about 16000 neurons, and more than one million synapses. The authors also implement conditionning mechanisms. The aim of the experiments has been to study simultaneously different levels of interactions (microscopic to macroscopic) in the system.

More recently, a similar model was used to study the emergence of place cells i.e. neurons whose activity is specifically enhanced when the individual is in a given place, in the modelled hippocampus of the system [48]. The authors focus on a specific substructure of the whole system, but the originality of their work is that the spike timings coming from the other sub-structures are completely defined, and are influenced by stimuli perceptions.

4.2 *A posteriori* models

An other way to study how several structures interact is to build an *a posteriori* model. In such an approach, the design of the neural network model is not based on the reproduction of known neural architectures or dynamics. The design is rather based on the ability of the model to cope with a given behavioural task.

In [49], Meunier describes a model (EvoSNN, for Evolutionary Spiking Neuron Network) composed of several randomly interacting populations of neurons. The topology of the network is adapted through an evolutionary algorithm. The fitness criterion of the evolutionary algorithm is based on the survival behaviour of a virtual individual controlled by EvoSNN and moving in a virtual preys-predators environment.

The topological and dynamical properties of the network are not taken into account in the fitness criterion. However, these properties are studied *a posteriori*, after the evolutionary process. Topological and dynamical properties of initial (before evolution) and evolved (after evolution) neural networks are compared. Evolved individuals have better learning capacities than initial ones [50]. Another key result, at the topological level, is that the wiring cost is optimized by the evolutionary process. Furthermore, the evolved network connectivity shows a specific kind of modular organisation [51]. At the level of dynamics, the author observes the emergence of gamma oscillations when a stimulus is presented to the network [52]. Moreover, the difference between temporal assemblies activated by two different stimuli appears at the spike timing level, but not in the spatial composition (subsets of neurons) of the assembly [53]. All these properties are coherent with experimental observations.

5 Conclusion

Although far from being exhaustive, we have presented some neuroscience results and introduced several computational models based on artificial neural networks that have been able to explain some of these results. Computational models present the possibility to validate or invalidate the functionality of hypothesis suggested by experiments in neuroscience and neuro-imaging. In addition they often suggest new hypotheses and new experiments. The models range from the microscopic level, i.e. at the single neuron level, to the macroscopic level, i.e. interactions between several cerebral structures. Other excellent examples can be found in books such as [54] or [55].

Building specific neural networks for modelling experimental results on cognitive processing or neural structures and dynamics in the brain is a very appealing domain. We hope this short presentation of several models as case studies will raise the interest of the atificial neural network community for the computational neuroscience research area.

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