Abstraction of the structure and dynamics of the biological neuron for a formal study of the dendritic integration

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Abstract

Understanding how neurons integrate the thousands of inputs they receive is a fundamental issue of neuroscience research. For this purpose, we define a new model for studying the impact of the dendritic morphology on the neuronal function. Following the Cable Theory application to neuron modelling, we propose relevant abstractions to reduce the number of parameters while keeping biophysical accuracy. This allows us to demonstrate a theorem characterizing structural equivalence classes of neurons sharing the same input/output (I/O) function. The theorem implies that the dendritic morphology is, surprisingly, not as critical as expected with respect to the I/O function of the neuron.

1 Introduction

Understanding brain organization and the way it processes neuronal information is an interdisciplinary worldwide challenge [6]. Here we focus on the I/O function at the single neuron scale with a particular emphasis on neuron morphology. It is known since decades that dendritic arborization is the part of the neuron where most of the neuronal computation is performed. However, it has been largely neglected up to know in computational neuroscience, faced with the difficulty to reduce its complexity. For this purpose, we decided to systematically use the remarkable abstraction capabilities of theoretical computer science. We propose the first neuron model integrating dendritic morphology, based on formal methods. It permits to prove rigorous properties about the role of the dentrites morphology in the I/O function of the neuron.

An input signal received on the dendritic tree far from the soma can easily undergo a 40-fold attenuation [19]. It follows that strong distal excitatory signals may be annihilated by a weak inhibitory signal received closer to the soma. In accordance, inhibitory synapses seem to be mostly located on proximal dendrites in some cell types [12, 1].

Section 2 introduces the basics about the neuron biology. Section 3 quickly describes the existing neuron models and introduces our framework. Formal methods commonly separate static descriptions from dynamics ones. Section 4

thus defines the static description of our framework, mainly allowing to describe the structure of a neuron. Section 5 defines the dynamic description, allowing to rigorously link any input signal to its output signal for any given neuron. Our formal approach allows establishing a necessary and sufficient condition for two different neurons to have the same I/O function. Section 6 describes this result. We finally discuss the impact of neuron morphology on its function in light of our result.

2 Archetypical biological neuron

2.1 Structure-function relationship

An archetypical neuron consists of a cell body called the *soma* and of two kinds of extensions: *dendrites* on the one hand and an *axon* on the other hand (cf. Figure 1). Nervous signals travel from dendrites to the axon passing through the soma. Dendrites are tree structures which can be highly branched. Nervous impulses are received at specific points called *synapses*, mostly located all over the dendrites. These input signals then propagate along the dendrites and accumulate in the soma which behaves as a bath with tap turned on. If soma potential exceeds a given threshold, a nervous impulse is generated and transmitted to adjacent neurons through the axon, partially emptying the bath. It should be noted that there are different types of neurons whose structure may widely vary from the archetypical one [17].

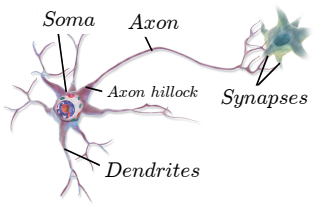

Figure 1: *Structure of archetypical biological neuron.*

2.2 Resting potential

The neuron, as any cell, is delimited by a membrane. It is a lipid bilayer pierced by channels confering a selective permeability to specific ions. At rest, the neuronal membrane has a polarity: the interior of the cell is negatively charged compared to the extracellular medium. This difference of potential of about –70mV is called the *resting potential*, due to an unequal distribution of ions on both sides of the membrane. When the permeability is disrupted, ionic flows are generated leading the membrane potential to deviate from the resting

value. A depolarization is an increase in the potential beyond its resting value and a hyperpolarization is a decrease.

2.3 Action potential

An *action potential* (AP) is the physiological support of what we call neuronal information. It is a sudden and transient reversal of the membrane potential. It is generated at the axon hillock where the membrane is rich in Na^+ voltage-dependent channels (cf. Figure 1). As the extracellular media is highly concentrated in Na^+, the opening of these channels causes a massive inflow of Na^+ resulting in a strong depolarization. These channels are rapidly inactivated and closed. At the same time, voltage-dependent K^+ channels open, leading to a K^+ ouflow, returning the potential to its resting value. This repolarization is usually followed by a slight hyperpolarization. The amplitude and duration of APs are constant parameters for a given neuron type. Therefore both duration and amplitude cannot encode the nervous signal. Therefore, the intensity depends only on APs frequency.

2.4 Synaptic transmission

Although there are electrical synapses, most of them are chemical. Incoming APs trigger the release of neurotransmitters in the synaptic cleft. These small molecules bind to receptors on the postsynaptic neuron membrane causing ionic channels to open. This results in a membrane potential change, called a *postsynaptic potential* (PSP), whose voltage is proportional to the intensity of the stimulation. The PSP can be either a depolarization or a hyperpolarization being thus respectively excitatory (EPSP) or inhibitory (IPSP). If the APs frequency is sufficiently high, their individual effects (PSP) are added: it is called *temporal summation*.

2.5 Propagation through the dendritic tree

PSPs generated at synapses propagate along the dendrites towards the soma. As thousands of synapses are distributed over the dendritic tree, the signals are combined all along: it is called *spatial summation*. The spreading of the electrical signal in dendrites is decremental: the potential tends to return to its resting value due to leak channels allowing ions to cross the membrane following the electrochemical gradient.

2.6 Integration by the soma and axon transmission

The soma accumulates all the signals having undergone both temporal and spatial integrations. There is a threshold below which the potential change at the soma has no consequence. However, if the depolarization is strong enough, a new AP is generated to be transmitted to other neurons via the axon. The reaching of this threshold actually triggers the opening of voltage-dependent Na^+ channels at the origin of the APs (Section 2.3). The maximal theoretical APs frequency is imposed by the *refractory period* in which no AP can be generated despite a suprathreshold potential. It is due to a transient inactivation of the Na^+ voltage-dependent channels just after each AP beginning. This absolute refractory period is followed by a relative one during which the stimulation must be stronger than usual to trigger the opening.

The newly generated APs propagate along the axon towards their output synapses in a regenerative way. It means that, contrary to the signal conduction in dendrites, the APs do not undergo any alteration.

3 Single neuron models

Neuron models described in the literature can be categorized according to their goal of modelling, separating computational from biophysical models.

3.1 Computational neuron models

Computational models are considered as computational units thus highly idealized. The first example is the formal neuron proposed by McCulloch and Pitts in 1943 [11]. It is a binary neuron which performs a weighted sum of its inputs. A Heavyside function is applied to calculate its output: 1 if the sum exceeds the threshold and 0 otherwise. It allows elementary logic calculations and it is mostly used as part of networks for artificial intelligence purposes. Basically, it is a bio-inspired calculation and it should not be used for a deep understanding of the neuronal functioning.

3.2 Biophysical neuron models

Biophysical models are mostly used to understand the neuronal behaviors. They allow to focus on specific mechanisms and usually consider the membrane potential as the key variable. The potential depends on the electrical membrane properties. One of the most famous biophysical model was presented by Hodgkin and Huxley in 1952 [7]. It describes the dynamics of ion channels governing the initiation of the AP by a set of non linear differential

equations. This model is accurate and compatible with experimental observations. However, it is very complicated and difficult to validate because of its large number of parameters [13]. This model thus led to simplified models such as the FitzHugh-Nagumo model [5].

3.2.1 The Integrate-and-Fire model

The *Intergrate-and-Fire (I&F)* model, proposed by Lapicque in 1907, does not focus on the molecular mecanisms governing the AP. It focuses on the I/O function of the neuron [10, 3]. The membrane is described as an electrical circuit constituted of a capacitor in parallel with a resistor. In the *leaky-I&F* model, an additional term is added to take into account the leak of ions through the membrane: $C\frac{dV}{dt} = -g_l(V - V_0) + I(t)$, where V is the membrane potential, C the capacitance, V_0 the resting potential, g_l the leak conductance and $I(t)$ the injected current (external or synaptic). This differential equation describes the potential dynamics below the threshold. The AP initiation is not explicitly represented: in addition, when V reaches the threshold, an AP is generated. At the same time, the potential is reset and the threshold value is updated to take into account the refractory period [18]. There are other extensions of the I&F model such as quadratic or exponential models [2].

3.2.2 Cable Theory applied to dendrites

Most of the existing neuron models are punctual, meaning that the neuron is equivalent to a point, ignoring its morphology. However, there are biophysical models focusing on the influence of structural characteristics on the nervous signal propagation. One of them is the Cable Theory applied to dendrites, proposed by Rall, a pioneer of dendrites modelling, in the 1960's [14, 15]. *Cable Theory* is based on a second order partial differential equation developed by Lord Kelvin in 1850 to describe the attenuation of the electrical signal spreading along a submarine cable. This concept was later applied to dendrites [15]. The dendrites are considered as cylindrical cables along which an electrical signal passively propagates. This phenomenon is described as follows: $\lambda\frac{\partial^2 V}{\partial x^2} = \tau\frac{\partial V}{\partial t} + V$, where V is the membrane potential variation from the resting value, x is the traveled distance over the cylinder, t is the time, $\lambda = \sqrt{\frac{r_m}{r_i}}$ is the space constant and $\tau = r_m \times c_m$ is the time constant where r_i is the intracellular resistance, r_m the membrane resistance and c_m the membrane capacitance.

Given an initial condition, the equation is solved analytically for different boundary conditions, assuming an infinite or a finite cable. The equation describes the attenuation undergone by the nervous signal during its conduction,

taking into account the length of dendrites, their diameter and the composition of the membrane. It is the basis of essentially all simulators taking the neuronal morphology explicitly into account such as Neuron or GENESIS [1]. However, the main drawback of this approach is the number of parameters involved [13].

3.3 A framework dedicated to the study of dendritic integration

In the remainder of this chapter, we study the impact of the morphology of the dendritic trees on the neuron I/O function. For this purpose, we have developed the first formal neuron model integrating dendrites morphology. We are at the interface between computational and biophysical models as we are not directly interested in the cellular mechanisms involved in neuron properties but rather in the "computational properties" of the neuron. Notwithstanding, our parameters can always be correlated with observable biophysical entities. Our major contribution is to model this process by mixing discrete and continuous modelling. Since the AP is known for its speed and its stereotypical properties, it is reasonable to consider it as an instantaneous event identified by its time of occurrence (conventionally called *spike*). More precisely, in our model a neuron receives spikes sequences at synapses. Those discrete inputs are immediately converted into continuous signals which then conduct through the dendritic tree towards the soma. The soma integrates all the signals and when a threshold is reached, a spike is emitted on the output. Our soma modelling is inspired by the leaky-I&F model. To investigate the dendritic integration, we focus on dendrites modeling, choosing Cable Theory as a basis. Although based on noticeable hypotheses [4], Cable Theory is credible and it allows very efficient abstractions. We have reduced the number of parameters while keeping the biophysical accuracy. Thanks to this approach, we are able to study equivalent classes of dendritic integration.

It is worth noting that our framework will include an *abstract* modelling of the ionic charge flows in the excited neuron. These charges are directly related to the membrane potential difference compared to the resting value, depending on the local membrane properties. Charges travel through the dendritic tree and they are summed at branching points. Finally, they control the soma potential and consequently the spike emissions. From our abstract point of view, we will make no difference between "charges" and "potentials.".

4 Static description of a neuron

According to computer science, a *tree* is recursively defined as a root node to which is attached a *forest* of children trees. Our neuron model consists of a forest of dendritic trees connected to a root soma and we ignore the axon as it

transmits the signal from the soma without any loss (cf. Figure 2) . Synapses on the dendritic forest are the input ports of the system. They receive sequences of spikes and each of them triggers a local change of the electrical potential. The potential reaches a maximal absolute value after a short period of time and then returns progressively to its resting value. The kinetic is proper to each synapse and characterized by the parameters given in Definition 1.

Definition 1. [Synapse] *A* synapse *is a triplet s = (ν_s, $\hat{\tau}_s$, $\check{\tau}_s$) where:*

- *ν_s is a non-zero real number called* maximal potential of a spike *for s. If $\nu_s > 0$ then s is said* excitatory*, otherwise it is said* inhibitory*;*
- *$\hat{\tau}_s$ and $\check{\tau}_s$ are strictly positive real numbers respectively called* rise time *and* descent delay *of the potential.*

Continuous signals generated at synapses then propagate in the dendritic trees towards the soma. We choose to divide dendrites into homogeneous elementary compartments delimited by branching points and synapses (Definition 4). In agreement with Cable Theory, we hypothesize a passive signal propagation with leakage. Whatever the boundary conditions, the analytical solution of the linear cable equation is of the form $V(x) = V_0 \times \alpha$ where α, is a variable depending on x. Therefore, we decided to describe the potential at the output of a compartment as equal to the potential at its input attenuated by a coefficient α after a delay δ (Definition 2). Grouping the parameters of the Cable Theory into those two abstract parameters greatly simplifies our model.

Definition 2. [Compartment] *A* compartment *is a couple c = (δ_c, α_c) where:*

- *δ_c is a real number greater or equal to zero called the* crossing delay *for c;*
- *α_c is a real number such that $\alpha_c \in]0, 1]$, called the* attenuation *at the end of c. If $\delta_c = 0$, then $\alpha_c = 1$.*

The soma accumulates input signals coming from the dendrites making its potential changing gradually. At the same time, there is a leak of charges making the potential slowly returning to its resting value. The soma is also characterized by its activation threshold from which it can emit a spike, and by the duration of the refractory periods (Definition 3). Note that the relative refractory period can be technically expressed by an augmented threshold.

Definition 3. [Soma] *A* soma *is a tuple ∇ = (θ, $\hat{\theta}$, ρ, $\hat{\rho}$, γ) where all the parameters are strictly positive real numbers: θ is called the* activation threshold*, $\hat{\theta}$ the* threshold augmentation*, ρ the* absolute refractory period*, $\hat{\rho}$ the* relative refractory period *and γ is called the* leak*.*

Definition 4. [Neuron] *A* neuron *is a labelled rooted tree satisfying the following conditions: any non-root node having 0 or 1 child is labelled by a synapse, any branch is labelled by a compartment and any non-root node having at least 2 children is called a branching (and is not labelled). The root of the tree is labelled by a soma. Given a neuron N, we note $Sy(N)$ the set of its synapses and $Co(N)$ the set of its compartments. Moreover, the direct children of the soma are called* dendritic trees*. Finally, we note $\mathcal{N}$ the set of all neurons.*

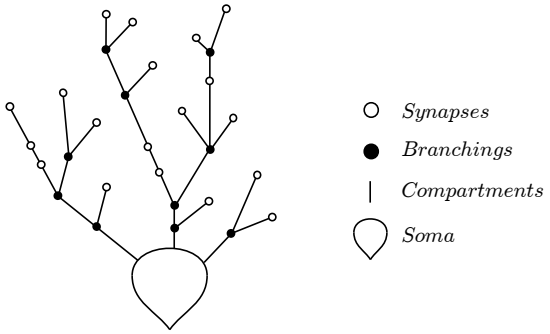

Figure 2: *Schematic representation of our neuron model structure.*

In computer science, the parent of a node is its only neighbour on the way to the root. The root is the only parentless node. Children of a node are its neighbours except its parent node. In the neuron model, the information goes from the leaves (synapses) to the root (soma). To avoid any confusion, we use the terms input/output and predecessor/successor to replace the couple child/parent. Given a neuron N and a compartment $y \xrightarrow{c} z$: y is called the *input node* of c, z the *output node* of c, c the *output compartment* of y and c the *input compartment* of z. We note $In(z)$ the set of the input compartments of z. A *predecessor* of $y \xrightarrow{c} z$ is a compartment in N of the form $x \xrightarrow{c'} y$ and we note $Pred(c)$ the set of the predecessors of c. Also, we call contributor compartments of c ($CC(c)$), the compartments at delay 0 from y. We define the synaptic contributors ($CS(c)$) in a similar way. More formally:

Definition 5. [Contributors] *Given a neuron N and a compartment $y \xrightarrow{c} z$, $\forall c' \in Pred(c)$, the set of the* contributor compartments *of c noted $CC(c)$ is defined inductively by:*

- *If $\delta_{c'} \neq 0$ then $c' \in CC(c)$;*
- *If $\delta_{c'} = 0$ then $CC(c') \subset CC(c)$.*

Moreover, the set of the synaptic contributors *of $y \xrightarrow{c} z$ noted $CS(c)$ is the subset of $Sy(N)$ defined inductively by:*

- *If* $y \in Sy(N)$ *then* $y \in CS(c)$*;*
- $\forall c' \in Pred(c)$*, if* $\delta_{c'} = 0$ *then* $CS(c') \subset CS(c)$*.*

5 The state of a neuron and its dynamics

To describe the state of a neuron and its dynamics, we need to introduce the notion of "segment":

Notation 1. *Given a set* E*, a* segment *with values in* E *is an application* $\omega : [0, t] \to E$ *where* $t \in \mathbb{R}^+ \cup \{+\infty\}$ *with the convention that* $[0, +\infty] = \mathbb{R}^+$*. We note Sgt the set of all the segments and we endow* $Sgt(E)$ *with a partial internal law of concatenation as follows:*
if $\omega_1 : [0, t_1] \to E$ *and* $\omega_2 : [0, t_2] \to E$ *are two segments such that* $t_1 \in \mathbb{R}^+$ *and* $\omega_1(t_1) = \omega_2(0)$*, then* $\omega_1 \cdot \omega_2$ *is the concatenated segment* [1] $\omega_1 \cdot \omega_2 : [0, t_1 + t_2] \in E$ *such that:*

- $(\omega_1 \cdot \omega_2)(t) = \omega_1(t)$ *if* $t \leqslant t_1$
- $(\omega_1 \cdot \omega_2)(t) = \omega_2(t - t_1)$ *if* $t \geqslant t_1$

We note $\bullet_{i=1}^{n} \omega_i$ *for* $\omega_1 \cdot \omega_2 \cdot ... \cdot \omega_n$*, the concatenation of the n segments.*
Moreover, if $\square$ *is a binary operation on* E*, it can be extended as follows: if* ω_1 *and* ω_2 *are segments of the same length* t_0*, we note* $\omega_1 \square \omega_2$*:* $[0, t_0] \to E$ *the segment such that* $(\omega_1 \square \omega_2)(t) = \omega_1(t) \square \omega_2(t)$ *for all* $t \in [0, t_0]$*. When* $\square = +$*, we accept the notation* $\sum_{i=1}^{n} \omega_i$ *for* $\omega_1 + \omega_2 + ... + \omega_n$*.*

The input signals are received at the synapses in the form of infinite segments taking value 1 at the times of the spikes and 0 otherwise (Definition 6). The output signal will be of the same type so that our modelling opens a way of building a network where the input of a neuron would come from the output of other ones.

Definition 6. [Signal] *A* signal *is a segment* $\omega : \mathbb{R}^+ \to \{0, 1\}$ *such that:*

$$\exists r \in \mathbb{R}^{*+}, \forall t \in \mathbb{R}^+, (\omega(t) = 1 \ \Rightarrow \ (\forall t' \in]t, t + r[, \omega(t') = 0))$$

The carrier of ω *is defined by:* $Car(\omega) = \{t \in \mathbb{R}^+ | \omega(t) = 1\}$*. A signal such that* $Car(\omega)$ *is a singleton* $\{u\}$ *is called a* spike *at the time u, noted* ω^u*.*

We call "trace", the potential change triggered by a spike. It directly depends on the synapse parameters (Definition 1). This continuous variation

[1] by convention $(t_1 + \infty) = +\infty$

is exponential from a biophysical point of view [15, 16]. For the sake of simplicity, we approximate it as linear (Definition 7). It seems reasonable as the experiments do not always follow the theoretical model and show a significant variability [8]. The temporal summation observed in biology is reproduced by making the sum of the respective traces of the successive spikes. It gives what we call the trace of the signal (cf. Figure 3).

Definition 7. [Trace of a signal] *The* trace of a spike ω^u *on a synapse* s *is the segment* v_{s,ω^u} *defined by:*

- *If* $t \leqslant u$ *then* $v_{s,\omega^u}(t) = 0$*;*
- *If* $u \leqslant t \leqslant u + \hat{\tau}_s$ *then* $v_{s,\omega^u}(t) = \frac{\nu_s}{\hat{\tau}_s}(t - u)$*;*
- *If* $u + \hat{\tau}_s \leqslant t \leqslant u + \hat{\tau}_s + \check{\tau}_s$ *then* $v_{s,\omega^u}(t) = \frac{\nu_s}{\check{\tau}_s}(u + \hat{\tau}_s + \check{\tau}_s - t)$*;*
- *If* $u + \hat{\tau}_s + \check{\tau}_s \leqslant t$ *then* $v_{s,\omega^u}(t) = 0$

Moreover, given an input signal ω_s *on a synapse s, the trace of* ω_s *is defined by the real segment* $v_{s,\omega_s} = \sum_{u \in Car(\omega_s)} v_{s,\omega^u}$.

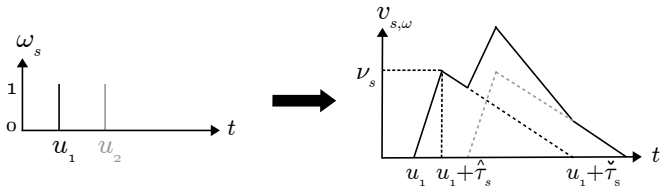

Figure 3: *Trace of a signal.* The trace of a signal is the sum of the traces of its spikes. Each spike received at a synapse s causes a maximum variation ν_s of the potential after a time $\hat{\tau}_s$ followed by a return to the resting potential with a delay $\check{\tau}_s$.

The state of a neuron at a given time is the value of the potential at every point of it. It includes the state of the soma and the state of all the compartments (Definition 9).

The value of the potential at the soma is not sufficient to characterize its state. One must also know the time elapsed since the last emmited spike to manage refractory periods. We thus define the state of the soma as a couple (e, p) where e is the elapsed time since the last spike and p is the current soma potential. Due to the biological properties of the neuron, there is a constraint on this couple which has to be nominal (i.e. normal): when p is above the threshold, e is necessarily less than the refractory period duration (Definition 8). Indeed,

when p exceeds the threshold, there are only two possibilities: either e is in the refractory period, or a spike is emitted and thus e is reset to 0.

Definition 8. [Nominal] *Given a soma ∇ = (θ, $\hat{\theta}$, ρ, $\hat{\rho}$, γ), a couple (e, p) where $e \in \mathbb{R}^+$ and $p \in \mathbb{R}$, is* nominal *if ($e < \rho$) or ($p < \theta$) or ($p < \theta + \hat{\theta} + \frac{\hat{\theta}(\rho - e)}{\hat{\rho}}$). We note Nominal($\nabla$) the set of all the nominal couples.*

Based on the compartment definition (Definition 2) and knowing the potential at its input node, it is easy to calculate the potential at its output after its crossing delay. The potential at each point of a compartment at a given time is deduced from this relationship. We thus define the state of a compartment c at a time h as a segment $v_c^h(t)$ which describes the evolution of the potential at its output between h and $h + \delta_c$ (cf. Figure 4).

Definition 9. [State of a neuron] The state of a neuron *N is a triplet $\eta = (V, e, p)$ where:*

- *V is a family of segments, indexed by $Co(N)$, the set of the compartments of N; each segment is of the form $v_c : [0, \delta_c] \to \mathbb{R}$ where δ_c is the crossing delay of the compartment c. For each compartment c:*

$$v_c(\delta_c) \sim \left(\sum_{c' \in CC(c)} v_{c'}(0) \right) \alpha_c$$

  where by convention the comparator "$\sim$" is: "=" if its input node is a branching, "$\geqslant$" if its input node is an exitatory synapse or "$\leqslant$" if its input node is an inhibitory synapse;

- *$e \in \mathbb{R}^+$ represents the* elapsed time since the last spike *and $p \in \mathbb{R}$ is called the* soma potential *such that the couple (e, p) is nominal for the soma of N.*

We note ζ_N the set of all the states of the neuron N.

The compartments dynamics is done by "segments sliding" by an arbitrary timestep Δ (cf. Figure 5). The potential at the input of c is calculated from the potential at the output of its contributor compartments while taking into account the spikes received at synaptic contributors. More formally:

Theorem 1. [Dynamics of the compartments]

Let a neuron N, an initial state $\eta^I = (V^I, e^I, p^I)$ and an input signal $\mathscr{S} = \{\omega_s\}_{s \in Sy(N)}$. There exists a unique application $\mathscr{V} : \mathbb{R}^+ \times Co(N) \to Sgt(\mathbb{R})$, which associates a segment $v_c^h(t) : [0, \delta_c] \to \mathbb{R}$ for each couple $(h, c) \in \mathbb{R}^+ \times Co(N)$ and such that, for any $\Delta \leqslant inf(\{\delta_c | c \in N \ \wedge \ \delta_c > 0\})$ and for each couple (h, c):

1. $v_c^0 = v_c^I$
2. *for any* $t + \Delta \leqslant \delta_c$, $v_c^{h+\Delta}(t) = v_c^h(t + \Delta)$,
3. *for any* t *such that* $\delta_c - \Delta \leqslant t \leqslant \delta_c$, $v_c^{h+\Delta}(t) = \left(\sum_{c' \in CC(c)} v_{c'}^h(t - \delta_c + \Delta) + \sum_{s \in CS(c)} v_{s,\omega_s}^h(t - \delta_c + \Delta) \right) \alpha_c$.

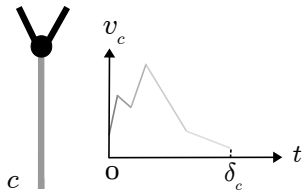

Figure 4: *The state of a compartment.* The state of a compartment c is the potential at its output between $t = 0$ ($v_c(0)$) and the crossing delay of c ($v_c(\delta_c)$).

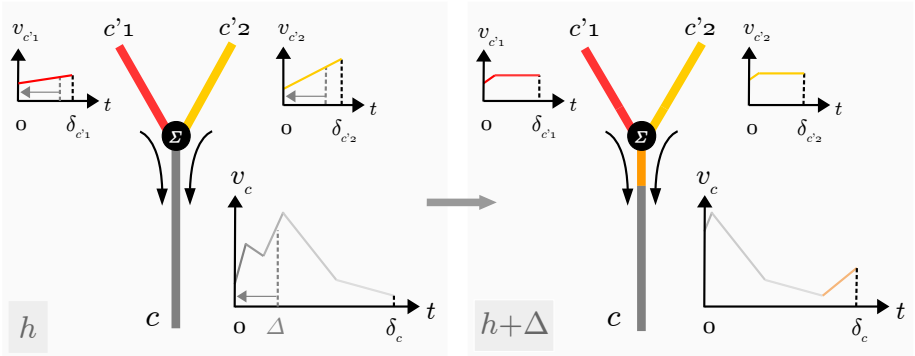

Figure 5: *Dynamics of the compartments.* The state of the compartments at time $h + \Delta$ can be calculated from the state of the compartments at time h by "sliding." The potential at the input of a compartment is the sum of the potentials at the output of its contributors.

Lastly, the soma dynamics is inspired by the leaky-I&F model. At a time t, the soma potential depends on inputs coming from the dendritic trees (denoted $F(t)$) and on the leak γ applied to the current value of the potential. From an initial condition (e_0, p_0), we can define the P_F function which describes the evolution of the soma potential over time (Lemma 1). To account for the refractory period, the couple (e, p) is forced to remain nominal (Definition 8). So when p reaches the threshold, its value is reduced (by θ) and e is reset to 0. P_F is hence discontinuous at this set of times that defines the carrier of the output signal (Definition 6).

Lemma 1. [Technical lemma] *Given a soma $\nabla = (\theta, \hat{\theta}, \rho, \hat{\rho}, \gamma)$, there exists a unique family of functions $P_F : Nominal(\nabla) \times \mathbb{R}^+ \to \mathbb{R}$ indexed by the set of continuous and lipschitzian functions $F : \mathbb{R}^+ \to \mathbb{R}$, such that for any couple $(e_0, p_0) \in Nominal(\nabla)$, P_F satisfies:*

- $P_F(e_0, p_0, 0) = p_0$
- $\forall t \in \mathbb{R}^+$ *the right derivative* $\frac{dP_F(e_0,p_0,t)}{dt}$ *exists and is equal to* $F(t) - \gamma.P_F(e_0, p_0, t)$
- $\forall t \in \mathbb{R}^+$, $\ell = \lim_{u \to t^-} (P_F(e_0, p_0, u))$ *exists and:*
  - *if* $(t + e_0, \ell) \in Nominal(\nabla)$ *then* $P_F(e_0, p_0, t)$ *is continuous and is derivable if* $t > 0$ *therefore* $P_F(e_0, p_0, t) = \ell$,
  - *otherwise, for any* $u \geqslant t$, $P_F(e_0, p_0, u) = P_G(0, \ell - \theta, u - t)$ *where for any* $x \in \mathbb{R}^+$, $G(x) = F(x + t)$.

The lemma hereinabove allows us to define the dynamics of a neuron which associates a state to each time.

Definition 10. [Dynamics of a neuron] *Given a neuron N, an initial state $\eta^I = (V^I, e^I, p^I)$ and an input signal $\mathscr{S} = \{\omega_s\}_{s \in Sy(N)}$, the* dynamics *of N is the infinite segment $d : \mathbb{R}^+ \to \zeta_N$ defined by:*

- $d(0) = \eta^I$
- $\forall h \in \mathbb{R}^+$, $d(h) = \eta = (V, e, p)$ *where:*
  - $V = \{\mathscr{V}(h, c)\}_{c \in Co(N)} = \{v_c^h\}_{c \in Co(N)}$ *where the application $\mathscr{V}$ is the one from Theorem 1;*
  - *Consider beforehand* $F(\hbar) = \sum_{c \in In(\nabla)} v_c^{\hbar}(0)$.
    F is lipschitzian at $\hbar$ as in any point its derivative is between $\left(\sum_{s \in Sy(N)} \frac{-\nu_s}{\hat{\tau}_s} \right)$ *and* $\left(\sum_{s \in Sy(N)} \frac{\nu_s}{\hat{\tau}_s} \right)$. *According to Technical lemma 1, there exists a unique function P_F such that $P_F(e^I, p^I, 0) = p^I$ and $\forall \hbar$, $\frac{dP_F(e^I,p^I,\hbar)}{d\hbar} = F(\hbar) - \gamma.P_F(e^I, p^I, \hbar)$. Therefore, if $P_F(e^I, p^I, \hbar)$ is continuous on the $]0, \hbar]$ interval, then $e = e^I + \hbar$. Otherwise, let $\hbar'$ be the greatest $\hbar$ such that $P_F(e^I, p^I, \hbar)$ is discontinuous, then $e = h - \hbar'$.*
  - *Considering the previous P_F function, $p = P_F(e^I, p^I, h)$.*

6 Remarkable properties

We found that any neuron can be reduced to a *pin-holder*. A "pin-holder" is a neuron where each dendritic tree is simply one synapse linked to the soma by a single compartment. The pin-holder corresponding to a neuron can be obtained by applying the decomposition function defined below:

Definition 11. [Decomposition function] *We note $\mathscr{P}$, the set of all the pin-holders. The* decomposition function *$fd : \mathscr{N} \to \mathscr{P}$ is the application which associates to any neuron $N \in \mathscr{N}$ the neuron from $\mathscr{P}$ built as follows:*

- *$fd(N)$ has the same soma than N: ∇,*
- *$fd(N)$ has the same set of synapses than N: $Sy(fd(N)) = Sy(N)$,*
- *For each synapse $s \in Sy(N)$, there exists a unique path made of compartments $c_1, ..., c_n$ (where for all i, $c_i = (\delta_{c_i}, \alpha_{c_i})$) from ∇ to s in N. Therefore, the compartment linking ∇ to s in $fd(N)$ is the couple (δ_s, α_s) such that $\delta_s = \sum_{i=1}^{n} \delta_{c_i}$ and $\alpha_s = \prod_{i=1}^{n} \alpha_{c_i}$.*

The definition of the state of a pin-holder differs from the state of its corresponding neuron only at the compartments level, as the soma remains the same.

Definition 12. [State of a pin-holder] *Given the state of a neuron N, we note $\overline{fd_N} : \zeta_N \to \zeta_{fd(N)}$ the application which associates to each state $\eta = (V, e, p)$ of N, the state $\overline{fd_N}(\eta) = (\overline{V}, e, p)$ of $fd(N)$ such that for each synapse s of $fd(N)$ and c its output compartment:*

$$\overline{v_c} = v_{c_1} \cdot (v_{c_2} \times \alpha_1) \cdot ... \cdot (v_{c_n} \times \prod_{i=1}^{n-1} \alpha_i) = \bullet_{i=1}^{n} \left(v_{c_i} \times \prod_{j=1}^{i-1} \alpha_j \right)$$

such that $c_1, ..., c_n$ is the path of compartments from ∇ to s in N where for each i, $c_i = (\delta_{c_i}, \alpha_{c_i})$.

Thanks to the pin-holder concept we have introduced, we demonstrated the following theorem:

Theorem 2. [The pin-holder theorem] *Let N_1 and N_2 be two neurons. If $fd(N_1) = fd(N_2)$ then, for any input signal $\mathscr{S}$, N_1 and N_2 have the same soma dynamics meaning that the evolution of the (e, p) couples with time is the same after a certain delay. Therefore, they have the same output signal (ω_∇) after a certain delay.*

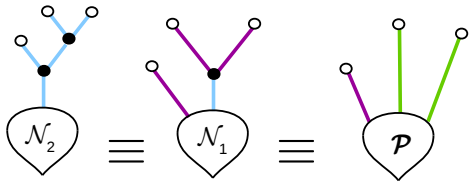

Figure 6: *The pin-holder theorem*. The theorem defines equivalent classes of structure and demontrates that the exact shape of dendritic trees is not critical in the I/O function of the neuron. The neurons $\mathcal{N}_1$, $\mathcal{N}_2$ and P represented here belong to the same class. The parameters are ($\alpha = 0.1$, $\delta = 1$) for the blue compartments, ($\alpha = 0.25$, $\delta = 2$) for the purple compartments and ($\alpha = 0.125$, $\delta = 3$) for the green compartments.

The interesting result is that a pin-holder is the canonical representative of a large set of neurons (cf. Figure 6). According to Theorem 2, these neurons have the same soma dynamics and therefore the same output for a given input. This result shows that the dentrites morphology is, surprisingly, not critical when only taking into account the I/O function of the neuron. Instead of the precise morphology, attenutation and delay are the key parameters in this function.

Moreover, the normalization into a pin-holder is an important reduction of the neuron complexity and this is very efficient for computational purposes.

Notice that the pin-holder is a sort of extension of the classical formal neuron (Section 3). Indeed, the α parameter of the compartments in our model is comparable to the weights applied to the inputs in the classical version. The main difference is the delay brought by our δ parameter.

7 Conclusion

The complex neuronal information processing emerges from an appropriate arrangement of a large number of neurons, each behaving as a device with potentially rich computational capabilities. Focusing on the I/O function at the scale of an individual neuron is thus a fundamental step in understanding the whole brain function. Cable Theory provides a good approximation to link the structure of the neuron to its function. As we are interested in the impact of dendrites morphology, we decided to base our work on this founding model. We proposed relevant abstractions to reduce the number of parameters while keeping the biophysics relevance. This enabled us to demonstrate the *pin-holder theorem* showing that a very large number of different dendtritic tree structures share the same I/O function. Consequently, and unexpectedly,

under the assumptions of Cable Theory, it implies that the precise morphology does not have a critical impact on the neuron I/O function, only delays and attenuations matter. It then comes that the dendritic morphology is probably essentially driven by the fact that neuronal structure is the result of a progressive development during neuroembryogenesis.

Among the basic Cable Theory assumptions, the passive conduction of the signals through the dendrites is a strong one: Some neurons exhibit active mechanisms at the level of dendrites [8]. Another assumption is that synapses parameters do not noticeably vary in time although inhibitory synapses seem to be more effective when an excitatory signal passes close to them [9, 17]. Taking into account these properties to model more complex neurons would probably modify our basic results, but we provide a first track on how the inputs distributed over the dendritic tree interact in time and space to determine the I/O function of the neuron.

A future direction is to take benefit from the *pin-holder theorem* in order to study interconnected neurons. Our framework opens up perspectives for precise network studies for two principal reasons. First, thanks to our hybrid approach, we would only need to work on discrete signals as the continous part of the system is restricted to the "internal" part of the neuron. Also, the *pin-holder theorem* allows to drastically reduce the neuron model complexity and this is a strong advantage for simulating and formally reasoning on networks.

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