# A dynamic neuro-synaptic hardware platform for Spiking Neural Networks

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Digital computers are built with two types of elements, bits which interact via logic gates. Quantum computers use *qubits* which are coupled through *quantum qates*. Similarly, a neurocomputer requires two hardware elements: *neurons* interconnected with *synapses*. We introduce a bio-inspired spiking neuro-synaptic unit built with conventional electronic components. Our hardware is based on a text-book theoretical model of a spiking neuron and its synaptic and membrane currents. All model parameters are tunable, and the timescales are bio-compatible. The spiking neuron is fully *analogue*, and its excitability is implemented with a memristor. The synaptic and membrane currents are both excitatory and inhibitory with tunable intensity and bio-mimetic dynamics. We demonstrate various basic neuro-computing primitives, and how to combine basic network motifs to achieve more complex neuro-computing functions. The neurosynaptic unit can be considered as the building blocks for constructing neural networks of arbitrary geometry. Its compact and simple design, along with the wide accessibility of ordinary electronic components, make our methodology an attractive platform to build neural interfaces for medical devices, robotics and artificial intelligence systems such as reservoir computing. We also discuss other possible extensions of our work: (i) to use the neuro-synaptic circuit as a template to incorporate oxide memristors that are under intense research in material science; (ii) to port the design to very large scale integration (VLSI) electronics to implement massive networks that may be less affected by the mismatch issue. Our device is a general-purpose neuromorphic building block which allows to implement a neuro-computer operating in real continuous time with perfect scaling. It may open the way to run theoretical neuroscience network models, beyond what is currently possible in digital computer simulations, thus achieving *Neuro-Computing Supremacy*. We provide a bill of materials and printed circuit board designs to implement the device.

#### I. INTRODUCTION

#### A. Neuro-Computing paradigms

Neuromorphic Computing (NC) is a field aimed at implementing systems that can perform functions taking inspiration from nervous systems and brains. Examples range from face recognition to autonomous navigation to robotic motion, and many more that are revolutionizing modern life. To implement those systems there are two main approaches. One is based on software running on digital computers, and the other is fabricating neuromorphic hardware that mimics the components of a neural network. In both cases, the NC systems rely on models from theoretical neuroscience, computer science or physics. Every model has a definition of its components, called neurons and synapses. Neurons are the degrees of freedom of a neural network, since they can be in different states. Synapses describe the couplings and interactions between them (see Fig. 1).

We may recall a paradigmatic example of an NC system, the Hopfield model, to clarify the roles of neurons and synapses, which are often a matter of confusion. This is a model for associative memory, introduced in the 80s [1]. It bears close similarity with magnetic spin models in Physics (see Fig. 1). The neurons are defined as discrete variables, where  $S_i = \pm 1$  denotes the  $i^{th}$  member of the network. The synapses are the interactions between neurons, where the real parameters  $J_{ij}$  denotes the interaction between neurons i and j.

The model has an energy given by the expression  $E = 1/N \sum J_{ij}S_iS_j$ , where N is the number of neurons. Each memory corresponds to a local minimum of the energy landscape. The genius of Hopfield was to formulate a simple recipe to choose the  $J_{ij}$  couplings, which provide such a landscape. The model can recall a memory by association. Namely, given an arbitrary initial state  $S_i$  for i = 1 to N, an algorithm of energy minimization will retrieve its associated memory.

This example also allows us to illustrate the case of



FIG. 1. Schematic view of the pre-synaptic (red) and postsynaptic (blue) neurons. The neurons are coupled by a synapse. The action potential reaching the end of the axon terminals of the pre-neuron induces a synaptic input current in the post-neuron, which can be excitatory (positive) or inhibitory (negative). Inset: analogy with Hopfield model, where neurons are spins  $S_i$ , and synapses are the couplings  $J_{ij}$ .

a software NC system. In fact, the Hopfield model, as any of the modern Deep Neural Networks for Artificial Intelligence, are algorithms running on digital computers. The computers may be conventional or the so-called "neuromorphic chips", such as TrueNorth or Loihi, which are digital processors with an optimized architecture to run those types of models [2]. Bit-like variables and  $J_{ij}$ couplings are the simplest modelization for neurons and synapses and allow for computation with massive neural networks, such as those used in Artificial Intelligence. However, severe limitations emerge quickly if one wants to adopt a more bio-mimetic spiking neural network (SNN) model.

Neurons are a special type of cells in the body that are characterized by having electric activity. They are not just active or inactive, but emit action-potentials that are fast voltage spikes. Synapses involve the dynamical process of transferring neurotransmitters between a presynaptic and a post-synaptic neuron, so they are not just real-valued parameters (see Fig. 1) [3].

The dynamical models of spiking neurons range from schematic to bio-realistic. In the first case, the most paradigmatic one is the leaky-integrate-and-fire (LIF) [3, 4]. For the latter, the most famous one is the Hodgkin-Huxley model which describes the generation of action potential in the giant axon of the sound [5]. These models are described by multiple non-linear differential equations. Similarly, to describe the synaptic couplings further differential equations are needed. Hence. to numerically study an SNN requires the solution of a challenging system of multiple coupled non-linear differential equations. The scaling of these SNNs is typically bad, as the number of differential equations grows fast with the size of the network and its synaptic connectivity. Possibly an even more serious problem is the scaling of the simulation time. An action potential spike typically lasts 1 ms, and the firing rate of a neuron is roughly between 1 and 100 Hz. Then a time-step of 0.1 ms is often adopted. The timescale associated with the global behavior can be considered of the order of minutes, so there is at least a  $10^6$  factor for the number of simulation time-steps, just for one neuron. However, this scaling quickly worsens with a growing number of neurons, at least by a factor of N, in the most favorable case. Nevertheless, there are several implementations of useful numerical simulators of SNNs available (see [6] for a recent review and benchmark).

It is not an easy question what is the largest SNN size that current simulators can handle [6], as this depends on the model, the connectivity, the computer architecture and power, etc. Whatever this estimate may be, it would become severely reduced if one considers continuous models, without hard-resets of variables, where the spikeemission is not described by time-stamps and synapses are dynamical. Namely, where the full non-linearity of the problem is properly treated.

Finally, another severe limitation imposed on software approaches is the numerical and electric power

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requirements needed to carry out the computation. Power efficiency becomes a relevant aspect, specially for practical applications such as navigation control, neuroprostheses, wearable implants, brain-machine interfaces, etc [7, 8].

#### B. Neuro-Computing in hardware

A very different approach to NC is based on the idea of implementing neurons and synapses directly in hardware. In fact, a hardware implementation has the very attractive feature of *ideal scaling* because *time represents itself*. In other words, the system evolves in real time and the time evolution occurs irrespective of the number of neurons in the network. If it takes one minute to simulate the behavior of one neuron, it takes the same one minute to simulate the behavior of a network of size N.

Modern silicon electronic technology based on complementary metal-oxides systems (CMOS) can implement, a priori, almost any desired mathematical model. This approach has several decades of development, starting with the pioneering work of C. Mead, who coined the term Neuromorphic Electronics [9]. He observed that transistors operating in the subthreshold regime, i.e. not as digital switches but as variable conductances, show a behavior analogous to the activation of ionic channels in neuron membrane models. There are currently many electronic chips that integrate millions of *silicon neurons* which typically require tens of transistors each [10, 11]. Surprisingly, the implementation of dynamical synapses in hardware has received comparably less attention [12], although they are a key feature in theoretical neuroscience [13]. The connectivity of those large-scale SNNs is usually handled by the so-called Address Event Representation (AER), where each spike emission is represented by a time-stamp and the address of the pre-synaptic neuron [11]. Then, the connectivity in an AER system is implemented by a digital system that generates and inputs a stereotyped signal of a certain intensity, representing the synaptic strength, at the address of the corresponding postsynaptic neuron.

A remarkable example of this type of hardware implementation is the DYNAP-SE chip, which counts  $4 \times 256$  neuro-synaptic computing units [14]. Each one implements an adaptive-exponential integrate-andfire neuron [3] that generates discrete spike events, plus a dynamical synapse that can implement biomimetic currents, which are routed via an AER system. This chip exhibits the appealing feature of ultra lowpower consumption. However, as all other electronic implementations that work in the transistor subthreshold regime, it suffers from the device mismatch problem. This remains a significant challenge, although there are interesting ideas proposed to mitigate it [15]. Another inconvenience of this approach is that it requires a high level of technical expertise and fabrication capabilities [11]. Moreover, it takes a rather long time to design and implement each electronic chip, which then cannot be modified.

Here we propose to consider a drastically alternative approach, introducing a spiking neuro-synaptic (NS) unit of unprecedented simplicity, having the ideal scaling property of hardware implementations, being easily reproducible, at a very low cost [16]. Moreover, our neuromorphic circuit realizes standard neuro-synaptic mathematical models, having fully tunable parameters. The circuit generates biomimetic spiking traces that resemble those of biological systems and can work at biorealistic timescales. It produces electric signals that are well adapted to build brain-machine interfaces, hence, is an ideal methodology for the implementation of neuro-Our NS unit is fully analog, including prostheses. the spike emission mechanism and the synaptic current injections that are not handled by an AER module. Unlike electronic chips, the spikes are produced by thyristors, which are not in the sub-threshold regime. Hence, our method mitigates the issues of device mismatch mentioned before.

With our approach, we may implement up to hundreds of units, which may be sufficient for research, flexible and tunable brain-machine-interfaces, robotics, and artificial intelligence, such as reservoir computing. Nevertheless, if a massive number of neuro-synaptic units were required, the present methodology can be ported to CMOS very large scale integration (VLSI). [17].

The present paper is organized as follows: We shall first describe the NS model and its circuit implementation, emphasizing the connection to mathematical models and its parameter tunability. Then, we shall demonstrate the implementation of the simplest motifs of spiking neural networks, that realize neuromorphic primitives, i.e. basic neuro-computing functions. We shall discuss how our work may open the way for a new type of neuro-computer. We pose an interesting question lying ahead: whether such a neurocomputer may achieve computational neuro-supremacy, i.e., the ability to perform computations beyond what is possible for software running on conventional (digital) This is a notion that is analogue to computers. that of quantum-supremacy in the field of quantum computation. Finally, we provide technical details on all our circuits and a list of materials, which are available upon reasonable request.

# II. NEURO-SYNAPTIC UNIT: THEORETICAL MODEL AND ITS PHYSICAL EMBODIMENT

# A. Theoretical model

The NS hardware unit takes inspiration from a basic model in Wilson's textbook on theoretical neuroscience (cf. Ch.12 of [13]). It is a set of differential equations for a spiking neuron of type-I supplemented with synaptic currents. A type-I neuron characteristically fires action potential spikes at arbitrary low rates, such as cortical neurons [13, 18].

When a pre-synaptic (or upstream) neuron emits a spike, which is a very fast event, typically lasting 1 ms, the action potential liberates a certain amount of neurotransmitters (see Fig. 1) into the synaptic cleft, which is about 20 nanometers wide [19]. Those molecules reach the dendrites of the post-synaptic (or downstream) neuron where they open ionic channels that induce synaptic currents integrated in the cell body. The neurotransmitters can either be excitatory or inhibitory, depending on whether they contribute or prevent the emission of a spike by the post-synaptic neuron. This depends on the sign of the synaptic current: if positive, or depolarizing, brings the potential of the neuron closer to the firing threshold; if negative, or hyperpolarizing, it has the opposite effect.

This synaptic current process typically occurs in the range of tens to hundreds of milliseconds. Thus, at a much longer timescale than the spike emission and has its own dynamics. This introduces an additional and relevant timescale in SNN dynamics, which is well recognized to have significant consequences [20].

In the 60's Rall introduced a set of differential equations to model this phenomenon, which is widely adopted [3, 13, 21, 22]. When applied to a delta function representing the sharp spike emission, they provide as a solution a synaptic current with the functional form known as Rall's alpha function  $\alpha(t) = t/\tau_s^2 \exp(-t/\tau_s)$ . Thus, the sharp (~ 1 ms) spike emitted by the pre-neuron is perceived by the post-neuron as a broad synaptic current input that peaks and decays in a typical timescale  $\tau_s$  (~ 10 - 100 ms).

Rall's differential equations for the synaptic current are incorporated in Wilson's book model (cf Eq. 12.18 of [13]). Those equations, along with the expression for a general integrate-and-fire spiking neuron from Gerstner's book (cf Eq. 5.2 of [3]), conform to the mathematical model that underlies our hardware implementation. The set of equations reads,

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$$C_m \frac{dV_m}{dt} = f(V_m) + I_\alpha + I_0 \tag{1}$$

$$\tau_{\rm s} \frac{dI_{\rm e}}{dt} = -I_{\rm e} + I_{\delta} H(V_{pre} - \Omega) \tag{2}$$

$$\tau_{\rm s} \frac{dI_{\alpha}}{dt} = -I_{\alpha} + I_{\rm e} \tag{3}$$

where  $V_m$  is the neuron's membrane potential,  $C_m$  is the membrane capacitance,  $\tau_s = R_s C_s$  is the synapse timeconstant with  $C_s$  and  $R_s$  being the capacitance and leak resistance of the synapse, respectively.  $I_0$  is an external input current to the neuron.  $I_e$  and  $I_\alpha$  are dynamical synaptic currents (analog to f and g in Wilson's book [13]). Also closely following the notation of Wilson, Hdenotes a step-function (or Heaviside function) that is unity if the pre-synaptic voltage  $V_{pre}$  is larger than the synaptic threshold  $\Omega$ , or zero otherwise [13]. Thus, the pre-synaptic action potential is modeled as a narrow pulse of rectangular shape that represents a  $\delta(t)$ -like spike. The condition  $(V_{pre} > \Omega)$  sets the width of the narrow pulse, that we call  $\tau_a$ , and its magnitude we denote  $I_{\delta}$ . Hence, this spike carries a charge

$$Q_N = I_\delta \tau_a \tag{4}$$

that physically may represent the amount of neurotransmitter liberated by the pre-neuron into the synapse.

The current  $I_{\rm e}$  results from the first leaky-integration (2) of the sharp rectangular current pulse. Hence, with  $\tau_a \ll \tau_s$  one obtains the exponential current,

$$I_{\rm e}(t) = \frac{Q_N}{\tau_s} \, \exp(-t/\tau_s) \tag{5}$$

This current is the input to the third equation (3), so after this second leaky-integration one gets the alphafunction [3, 13, 22]

$$I_{\alpha}(t) = \frac{Q_N}{\tau_s} \frac{t}{\tau_s} \exp(-t/\tau_s)$$
(6)

These currents have bio-mimetic line shapes [3, 13]. They realize the synaptic currents of four basic types, namely, fast exponential excitatory (AMPA) or inhibitory (GABA<sub>a</sub>), and slow alpha-function excitatory (NMDA) or inhibitory (GABA<sub>b</sub>) (cf Ch.3 of [3]).

#### B. Analogue Leaky-Integrate-and-Fire

To fully specify the model, we need to define the nonlinear function  $f(V_m)$  in Eq. 1 that generates the spikes. In the interest of simplicity, we start by recalling the leaky-integrate-and-fire (LIF) model, which is the most widely used. In the LIF, shown in Fig. 2, the body (soma) of the neuron is represented as a capacitor  $C_m$  that charges by integrating the input ionic currents arriving at its dendrites. This integration suffers leakage losses which are represented by the membrane resistor  $R_m$ . The third component of the LIF model is a voltage-dependent switch S that is normally open, but closes when the potential  $V_m(t)$  on the capacitor reaches a threshold  $V_{th}$ . At that point, one says that a spike is "fired" at a time  $t = t_{fire}$ , the voltage  $V_m$  is reset to a low resting value and the switch is set back to open. It is important to realize that the LIF is a mathematical model and the spike is an abstract "event" defined by the condition  $V_m = V_{th}$  [3]. In other words, there is no dynamical description of the spike [23]. In this modelization, the non-linear function  $f(V_m)$  is implemented by the Vdependent switch.

Here, we shall adopt the above idealized description as a basis for our hardware implementation, which we shall also augment to provide an embodiment of the physical emission of an action-potential spike (see Fig. 2). The first step is, as shown in Fig. 2, to add a small "load" resistor with  $R_a \ll R_m$ . Then, when S closes at  $V_m = V_{th}$ , the  $C_m$  quickly discharges on  $R_a$  and the current on this resistor produces a voltage spike. The fast discharge time is  $\tau_a = R_a C_m$ , which provides a definite parametrization to the width of the  $\delta$ -like spike that we mentioned above (4). Interestingly, we



FIG. 2. aLIF model: (a) The leaky-integrate-and-fire model with a voltage dependent switch S is augmented with a discharge resistance  $R_a$  to produce an explicit action potential (left). The switch can be implemented by a thyristor T with a resistor  $R_m$  between its gate and anode. This resistor also provides the leak resistance of the model while the thyristor is not conducting (mid panel). The combination of  $T+R_m$ realizes the concept of a memristor, with the functionality of a voltage-gated conductance, central to all neuron models (right panel). (b) LIF behavior: input current pulses (lower panel) are leaky-integrated producing the increase of the membrane voltage  $V_m$  (mid panel). When  $V_m$  reaches the dynamical threshold  $V_{th}$  (vertical arrows), the forward resistance of the T collapses and there is the emission of an output of a continuous action-potential spike  $V_{ap}$  (top panel). Circuit technical details and parameter values for all figures are provided in the Appendix.

may give this spike generation mechanism a biological interpretation as the action potential that is initiated in the axon hillock [24]. The axon hillock is characterized by a high concentration of Na channels, over 100 times more than in the cell body (soma) [25]. We can thus think of the closing of the V-dependent switch as representing the massive opening of those channels and the enhanced membrane conductance at the axon hillock described by the small resistance  $R_a$ . Despite the extended use of the LIF model, we are not aware of this simple extension. The second step to specify the embodiment of the model is to implement the V-dependent switch. The simplest circuit to do this exploits the concept of a *volatile memristor*, which is V-dependent resistance (or V-gated conductance) [26]. We have recently, demonstrated that a combination of a thyristor and a resistor can implement such a memristive two-terminal device that provides the desired functionality [27, 28].

A voltage-gated conductance is in fact a key ingredient common to all conductance-based neuron models, such as Hodgkin-Huxley, Morris-Lecar, Hindmarsh-Rose, etc, [13]. Those theoretical models are formulated with differential equations that describe the spike emission as a *continuous* dynamical process. It results from the strong non-linearity of the equations, namely the property of excitability [13]. We note that simplified models, such as Izhikevich and AdEx [3], which are popular for numerical simulations, do not fall in that category. This is because they require a discontinuous reset, for the sake of their numerical simplicity [3, 23].

Our memristor spiking model implementation is shown in Fig. 2 and it is described by the following equations,

$$C_m \frac{dV_m}{dt} = -\frac{V_m}{R_m[S] + R_a} + I_0$$
(7)

$$V_{ap} = \frac{V_m}{R_m[S] + R_a} R_a \tag{8}$$

where  $R_m[S]$ , with S=hi,lo, denotes a volatile memristor whose resistance can switch between two states: a high resistance  $R_m[hi]$  and low resistance  $R_m[lo]$ . The commutation depends on the applied voltage, thus S = $S(V_m)$ , implementing a V-dependent conductance [28]. The second equation (8) is a voltage divisor providing the action-potential  $V_{ap}$  on the axon-hillock resistor  $R_a$ . When the memristor is in the  $R_m[hi] \gg R_a$  (i.e. open switch) then  $V_{ap}$  is negligible. When it commutes to  $R_m[lo] \ll R_a$  (i.e. closed switch) there is an actionpotential spike explicitly described by  $V_{ap}(t)$  (see Fig. 2).

Notice that the first differential equation above may seem linear, but it is not. In fact, the non-linearity, necessary for the excitability of the model, is provided by the commutation property of the memristor device [28].

The circuit implementation of the LIF model that we just described features a fully continuous spike generation mechanism. Therefore, our circuit-model, although not bio-realistic but bio-mimetic, can be considered as a member of the class of *conductance-based* neuron models and we call it the analog-LIF (aLIF) model. In Fig. 2 we show the measured traces of the neuron circuit, which shows the leaky-integration of input current pulses and the resulting output emitted spikes  $V_{ap}(t)$ .

# C. Physical embodiment of the neuro-synaptic model equations

One of the main goals of the present work is to provide a simple and transparent physical instantiation of the set of equations (1-3), which our the mathematical reference. We would like that each circuit component could be directly related to the theoretical model parameters. Furthermore, we would like the hardware implementation to be modular, such that the sub-circuit units can be associated to build arbitrary spiking neural networks, and eventually a neuro-computer. We shall see in the rest of the paper that all these goals are accomplished by the NS unit that we describe below.

For the spiking generation mechanism, which is encoded in the first equation (7), we adopt the aLIF circuit model discussed in the previous section. Thus, we rewrite below the reference model (1,3) including the aLIF and renaming some variables for convenience,

$$C_m \frac{dV_m}{dt} = -\frac{V_m}{R_m[S] + R_a} + I_{s2} + I_0 \tag{9}$$

$$V_{post} = \frac{V_m}{R_m[S] + R_a} R_a \tag{10}$$

$$\tau_s \frac{dI_{s1}}{dt} = -I_{s1} + I_\delta H(V_{pre} - \Omega) \tag{11}$$

$$\tau_s \frac{dI_{s2}}{dt} = -I_{s2} + I_{s1} \tag{12}$$

where  $V_{post} = V_{ap}$  denotes the output post-synaptic action-potential, and  $I_{s1}$  and  $I_{s2}$  denote the synaptic currents of the two leaky-integration stages. Similar to provided for the spike generation via the aLIF circuit, the synaptic current equations (11, 12) also have a concrete physical embodiment as we show in the circuit in Fig. 3. We indicate in the figure the correspondence between the equations of the model and the circuit blocks.

We shall now describe the implementation in detail. For the sake of clarity, we shall explain the circuit in Fig. 3 from input to output, i.e. from the pre-synaptic neuron on the left  $(V_{pre})$  to post-synaptic neuron on the right  $(V_{post})$ .

#### 1. Neuro-Synaptic circuit blocks

The first block (A) is an aLIF representing the presynaptic neuron. It generates the action potential spike  $V_{pre}$  that is the input to the NS unit. It was described in detail before.

The second block (B) implements the differential equation (11) of our model. The current term  $I_{s1}(t)$  is the dynamical variable and the  $I_{\delta}H(V_{pre} - \Omega)$  is an independent term.

The  $I_{\delta}(t)$  current are rectangular-pulses emitted by each spike  $V_{pre}$ . Their width is  $\tau_a$  (see II B) and their intensity is controlled by a resistor  $R_W$  in a conventional current-source implementation. The technical details are



FIG. 3. (a) Schematic diagram of the neuro-synaptic unit circuit blocks and their relation with the biological neuron. (b) Physical embodiment of model Eqs. 9-12 with sub-circuit blocks denoting the pre- and post-synaptic neurons and the synaptic block in-between. The synaptic block has two stages that perform a leaky-integration each. The input pre-synaptic potential produces a  $\delta(t)$ -like current spike. A double leakyintegration of pulse provides an  $\alpha(t)$ -like synaptic current into the post-synaptic neuron, which also receives the external input  $I_0$ .

provided in the Appendix. Here, it is important to mention that  $R_W$  controls the synaptic coupling that is denoted  $W_{ij}$  between neurons *i* and *j*, in the terminology of Artificial Neural Networks.

These  $W_{ij}$  couplings are key parameters for networks that implement synaptic learning, such as through spike time-dependent plasticity [3]. In that case, one may adopt non-volatile memristors for the  $R_W$ , as we shall discuss elsewhere.

The synaptic current pulse  $I_{\delta}$  has a short duration, so it approximates a delta function  $I_{\delta}$  that is leakyintegrated by the  $R_s C_s$  pair. This circuit provides the time constant  $\tau_s$  and implements the differential equation in the current  $I_{s1}(t)$ , which is the output of the second block.

The third circuit block (C) implements (12) in a similar fashion. There, the current  $I_{s2}(t)$  is the dynamic variable and the current  $I_{s1}$  enters as the independent term. To do this we need to take  $I_{s1}$  output from the second block and input it to the third one. The tricky part is to "copy the current" from one to the other, independently of the load represented by the latter. We resolve this by a standard electronic circuit, aptly called a "current mirror" (CM), and provide the technical details in the Appendix.

We note a useful feature of our CM implementation, namely, given the input, the output can be chosen *either* positive or negative in polarity. This feature is extremely convenient, as we can implement either excitatory or inhibitory synaptic currents. In other words, this feature allows to choose the sign of the synaptic coupling parameters  $W_{ij}$  in an artificial neural networks.

This circuit block leaky-integrates the input current

 $I_{s1}$  with timescale constant  $\tau_s = R_s C_s$ , similarly as the previous stage. The output of this block is the current signal  $I_{s2}(t)$  and the same CM solution is adopted to input it (or its negative copy) to the last block which is the aLIF circuit of the post-synaptic neuron.

The last circuit block (D) of Fig. 3 implements Eqs. 9 and 10, i.e. the aLIF neuron model [28]. This sub-circuit has already been discussed in detail above. Here we just note that it corresponds to the *post-synaptic* neuron, so the action-potential spike emitted by this last block is denoted by  $V_{post}(t)$ .

# 2. Synaptic current forms

The reader may have realized that our implementation provides the freedom to adopt either one or two leakyintegration synaptic blocks. If only one (yellow) block is used, the single spike input  $I_{\delta}$  is transformed into the  $I_{s1}(t) \sim \pm \exp(-t/\tau_s)$ . While, if two blocks are adopted, the resulting output to a single input  $I_{\delta}$  spike is  $I_{s2}(t) \sim \pm \alpha(t/\tau_s)$ .



FIG. 4. Traces at the different stages of the NS unit. (a) The pre-synaptic neuron action-potential spike is the input to the NS unit. (b) The action-potential is transformed into a  $\delta(t)$ -like pulse with an intensity  $I_{\delta}$  (modulated by  $R_W$ ) and a duration given by the spike emission time  $\sim \tau_a$ . The pulse carries a total "neurotransmitter" charge  $Q_N \approx I_{\delta}\tau_a$ . (c) first leaky-integration stage (Eq. 11) which provides and exponential current  $I_e = I_{s1}$  with timescale  $\tau_s = RsCs$ .  $Q_N$  is the time-integral of the current (d) second leaky-integration stage (Eq.12) which provides an  $\alpha(t)$ -like synaptic current  $I_{\alpha}$  $= I_{s2}$ . Note that, as in the mathematical model, both  $Q_N$ and  $\tau_s$  remain constant (to a reasonable approximation) after each successive leaky-integration. They are also in reasonable good agreement with the nominal parameter values  $Q_N \approx 80$ nC (calculated from Eq. 15) and  $\tau_s = 1.6$  ms.

In the panels of Fig. 4, we show an example of the temporal traces of the currents at different points of the NS unit circuit of Fig. 3. The first one is the presynaptic action potential spike, the output of the box A in Fig. 3, which shows the triangular shape produced by the fast discharge of  $C_m$  on  $R_a$  with  $\tau_a \sim ms$ , as we described before. This pre-synaptic potential, through the condition  $V_{pre} > \Omega \approx 0.7 V$  in our implementation (see Appendix), produces the  $I_{\delta}(t)$  current-pulse, which is input to the box B. It has duration  $\tau_a$  and intensity  $I_{\delta} \approx V_{+}/R_{W}$ , where  $V_{+} (\approx 4V)$  is set by the circuit voltage source (see the Appendix). The sharp pulse is then leaky-integrated (cf Eq.11) to produce the synaptic current  $I_{s1}(t)$  with the exponential form as in Eq.11, at the output of the box B. The second leaky-integration produces the synaptic current  $I_{s2}(t)$  with the alpha-form as in Eq.12, at the output of the box C. As shown by the good quality of the fits in the Fig. 4, the NS faithfully implements those functional forms and keeps constant the normalization of the integrated charge  $Q_N$ to a reasonably good approximation.

#### 3. Synaptic current versus conductances

There is one important point about our implementation that is worth clarifying here. Theoretical models are formulated in terms of either membrane conductances or synaptic currents (see Ch.3 and Fig.3.2 in [3]). However, we shall show that these two options are essentially the same.

The key point is to realize that neurons spend most of their time at and around their resting potential,  $V_m \approx V_{rest} \approx -70$  mV. The potential  $V_m$  slowly drifts up or down under the influence of synaptic currents. Eventually, a small upward change of about 10 mV, drives the neuron to emit a short action potential spike and quickly returns to  $V_m \approx V_{rest}$ . Thus, since the general expression of synaptic currents in theoretical models reads [3]

$$I_{syn}(t) = -g_{syn}(t)(V_m(t) - E),$$
(13)

where  $g_{syn}(t)$  is the synaptic conductance and E is a constant potential parameter, which is chosen either higher or lower than  $V_{rest}$  for excitatory or inhibitory currents, respectively [3, 13]. Hence, from  $V_m(t) \approx V_{rest}$  all the time except in brief firing events lasting a few ms, to a good approximation the temporal dependence of  $I_{syn}$  is the same as  $g_{syn}(t)$ , so one can model either. This feature is also the reason why one often reads in neuroscience literature expressions such as "to inject a conductance" [29].

# D. Synaptic current timescales: spike-by-spike to rate coding

An important question in neuroscience is what is the nature of the neural code. In neural networks, do neurons code through the timing between spikes or by the neuron spiking rate? Our neuro-synaptic unit is wellequipped to implement both of these representations. The key parameters to compare are the timescale for successive spike emission, i.e. the membrane timescale  $\tau_m \sim R_m C_m$ , versus the synaptic integration timescale  $\tau_s \sim R_s C_s$ .

If  $\tau_s \leq \tau_m$  then the synaptic leaky-integration can follow individual spike emission and spike-by-spike time coding is possible. On the other hand, if  $\tau_s \geq \tau_m$ , the effect of the successive spikes gets accumulated and a synaptic current  $I_s$  proportional to the firing rate f results. In Fig. 5 we illustrate the different coding regimes. We show the input spikes  $V_{pre}$  of a pre-synaptic neuron acting on the synaptic unit (cf Fig. 3) and the resulting output synaptic currents  $I_{s2}$  that it generates.



FIG. 5. Left: Spike-by-spike coding. Each emitted presynaptic spike produces individual  $\alpha$ -type spikes of synaptic current. Center and right: Rate coding. The pre-synaptic emitted spikes have a high rate and produce a synaptic current build-up by accumulation or leaky-integration. The current intensity encodes the spiking frequency rate,  $I_s = I_s(f)$ .

To determine whether the system is in the spikeby-spike or rate-code regime, we need to consider the activation function of the neuron model  $f(I_{in})$  that is shown in Fig. 6. The frequency, or equivalently, the interspike interval (ISI=1/f) depends on the external input current excitation, so it is more accurate to compare this timescale (instead of  $\tau_m$ ) with the leakyintegration timescale  $\tau_s$ . Hence, per our previous discussion, we have that  $1/f > \tau_s$  would correspond to spike-by-spike, while  $1/f < \tau_s$  to the rate-code mode. Thus, higher spiking frequencies naturally correspond to the rate-code regime, and this is well illustrated in Fig. 5

We may make here a side comment on the activation function  $f(I_{in})$ . As shown in the Fig. 6 this function also exhibits a maximal current threshold  $I_{max}$ , where the firing frequency suddenly decreases down to zero [28]. This is actually a biomimetic property of our neuron model. The eventual decrease of the spiking frequency at high current excitation is a phenomenon that is observed in biological neurons, where is known as "depolarization block" [30]. It is often considered relevant for the understanding of epileptic seizures [31].



FIG. 6. Response functions of Neuron and Synapse subcircuit units. (a) The spiking rate as a function on the input current  $f(I_{in})$  produced by the aLIF neuron sub-circuit (box D in Fig. 3)  $I_{min}$  denotes the threshold of excitation and  $I_{max}$ the value where the neuron stops spiking. (b) The synaptic current  $I_s(f)$  of the leaky-integrator sub-circuit (boxes B and C in Fig. 3) in the spike rate regime. The slope is given by the neurotransmitter charge  $Q_N$ , as in (14). In dashed line, the theoretical  $I_s(f)$  for the nominal value of  $Q_N$  is computed with (15).

It is an important question to determine what controls the intensity of the synaptic current in the rate-coding mode. We have discussed in Section II C 2 how the intensity of the synaptic current that is produced by a single action potential spike is controlled by the resistor  $R_W$ . More specifically, we showed that for spike-byspike coding,  $R_W$  controls the total charge due to a single spike, i.e.,  $Q_N = \int I_s dt$ . This charge per spike is *conserved* across the leaky-integration stages, i.e. there is no current amplification.

For the rate-coding case, the successive spikes are accumulated, hence we expect that the synaptic current will be given by  $Q_N f$ . A subtle point to note, however, and as seen in Fig. 5, is that the accumulation takes a certain time to reach a steady-state, which is the leaky-integration time-constant  $\tau_s$ . Therefore, after the transient integration time, we have,

$$I_{se} \approx Q_N f \tag{14}$$

for the excitatory case.

This simple relation is in good agreement with our circuit measurements shown in Fig. 6, including the quantitative value of the slope  $Q_N \approx 80$  nC (see Fig. 4).

We may provide the explicit dependence of  $Q_N$  with the circuit model parameters. Recalling that the expression for the charge contribution of each spike, we have,

$$Q_N \approx I_\delta \tau_a \approx (V_+/R_W) R_a C_m \tag{15}$$

Finally, we should note that all the previous considerations remain also valid for the case of inhibitory synaptic currents (since we just change the polarity of the last current mirror). Hence, in that case the  $I_s(f)$  response function is simply the reflection of Eq. 14, namely,

$$I_{si} \approx -Q_N f \tag{16}$$

## III. RESULTS

# A. Basic spiking neural network motifs and neuro-computational primitives

The methodology that we described above, in terms of circuit blocks is modular, therefore, is well adapted to implement arbitrary biomimetic SNNs. In the next sections, we shall illustrate how one can establish basic network motifs, such as a single neuron with synaptic self-excitation or self-inhibition, and then two mutually inhibiting neurons. The spiking states of these motifs realize neuro-computational functions or primitives.

The basic motifs that we shall consider are shown in Fig. 7. The simplest motif is, of course, an isolated spiking neuron. We have already discussed its behavior in Section IIB. The first non-trivial motifs display self-feedback, which is the simplest case of a recursive SNN. The self-excitatory case provides an instantiation of an important neuro-computing primitive, called dynamical memory, short-term memory or simply activity "bump". The second case is a self-inhibitory neuron, which implements two other important neural primitives: spiking adaptation and bursting [3, 13].

Then, we shall leverage those basic primitives to build more complex two-neuron SNNs. We shall consider two different motifs, and implement three different types of central pattern generators (CPG) [13, 32]. These are also fundamental neuronal functions that are widely studied as they broadly appear in neuroscience [33, 34] and robotics [35, 36]. While in the present introductory work, we shall stop at two-neuron motifs, it should be clear that the present methodology is general and permits building larger SNNs with arbitrary architecture.

# B. Self-excitation: Activity bump and dynamical memory

In this section, we begin with the simplest motif: a self-excitatory neuron (see Fig. 7), which can implement a basic neuro-computational primitive, namely, a dynamical-memory. This reverberating self-sustained state is also of central importance in neuroscience, where it is called short-term memory (cf. Ch.6 in [13]) or working memory [37]. The excitation "bump" neuro-computational function occurs in models of great current interest, such as place and grid cells [38] and in head direction systems [39]. It also appears in models of the oculomotor system, which allows to fix the gaze in a given direction [40].

In a biological neuron, the self-excitatory current may represent the intrinsic transient inward Ca<sup>2</sup>+ ionic current, known as  $I_T$  (see Ch.10 in [13]). If, alternatively, the self-synaptic connection is extrinsic, such as due to the neuron's own axon connecting the dendrites, it is called an autapse, which is a topic of current interest [41–44]. This may be relevant to epilepsy, where it was



FIG. 7. SNN motifs: (a) Single spiking neuron. (b) Single spiking neuron with excitatory feedback. This motif represents both an autapse (self axon-dendrite connection) or an intrinsic excitation current, like the inward  $\operatorname{Ca}^{2+}$ ,  $I_T$ . (c) Single spiking neuron with inhibitory feedback. This motif represents both an autapse or an intrinsic adaptation current, like the hyperpolarizing  $I_{AHP}$  that is also mediated by  $\operatorname{Ca}^{2+}$ (see 10.3 of [13]). (d) CPG master-slave motif with a burster neuron (master) that inhibits a regular spiking neuron (slave). (e) CPG of mutually inhibiting neurons with self-adaptation. The same motif has two cases: two bursters or two frequencyadaptation neurons, which give qualitative different traces.

reported that in epileptic humans about 30% of cortical neurons of layer V form autapses [45]. Moreover, very recently, it was observed that the ictal source point of epileptic seizures occurs in layers IV, V, and VI [46].

For a self-excitatory synaptic current, the system of equations of our model (9-12) becomes,

$$C_m \frac{dV_m}{dt} = -\frac{V}{R_m[S] + R_a} + I_{sse} + I_0$$
(17)

$$V_{ap} = \frac{V_m}{R_m[S] + R_a} R_a \tag{18}$$

$$\tau_s \frac{dI_{s1}}{dt} = -I_{s1} + I_\delta H(V_{ap} - \Omega) \tag{19}$$

$$\tau_s \frac{dI_{sse}}{dt} = -I_{sse} + I_{s1} \tag{20}$$

where for the self-excitation, the spike potentials become  $V_{pre} = V_{post} = V_{ap}$ . Additionally, to indicate the synaptic self-excitatory current,  $I_{s2}$  is renamed  $I_{sse}$ . The system of equations above corresponds to the neuro-computing circuit shown in Fig. 8. The key feature of the activity bump is that the neuron can reach a spiking state, and remain self-excited, despite the input external current  $I_0$  being *sub-threshold*, namely  $I_0 < I_{min}$ . As we shall discuss below, the self-excitation state is maintained by the build-up of the self-synaptic current  $I_{sse}$ .

To start the bump of activity it is necessary to apply a short excitatory external current pulse  $I_{\Delta}$  on top of the constant sub-threshold current  $I_0$ , as shown in Fig. 9. Since  $I_0$  is sub-threshold, we observe that there is no activity during the first 2 seconds. Then an excitatory



FIG. 8. Self-synaptic current circuit.  $I_0$  is the external current input,  $V_{ap}$  is the output train of action potential spikes. The output spikes are feedback to the neuron through the self-synaptic current  $I_{sse}$ .

pulse is applied, that fulfills two requirements: (i) it has to drive the neuron to the excitatory regime above threshold, i.e.  $I_{min} < I_0 + I_\Delta < I_{max}$ ; (ii) its duration  $\tau_\Delta$ has to allow for the build-up of the self-synaptic current  $I_{sse}$ , i.e.  $\tau_\Delta > \tau_s$ .

We observe in Fig. 9 that the spiking starts as soon as  $I_{\Delta}$  begins and within the next second  $\sim \tau_s$  the  $I_{sse}$  starts to build up and the frequency rate increases. Within the next second by the time  $I_{sse}$  is terminated, the self-feedback is well in place. The persistent state is realized as  $I_0 + I_{sse} > I_{min}$ . The spike rate of the bump of activity stabilizes at the self-consistent rate  $f^*$  as we discuss below.

We can recall the two response functions that characterize the neuro-synaptic unit (see Fig. 6) and use them to formulate the bump state as a self-consistent problem. On one hand, we have  $f(I_{in})$ , the neuron activation function. On the other, the synaptic current  $I_s(f)$  that results from the synaptic circuit, given an input spike-rate f from a neuron. We should also note from Fig. 9 that  $\tau_s > 1/f$ , so we are indeed in the rate coding regime.

In the self-excitatory motif, the synaptic current is feedback to the neuron, hence  $I_s = I_{see}$  (see Fig. 6. Therefore, we can cast the bump as a self-consistent problem: (i) the total input current is  $I_{in} = I_0 + I_{sse}[f]$ , where  $I_0$  denotes a constant sub-threshold external current and  $I_{sse}$  is the self-excitatory contribution that depends on the spiking frequency; (ii) the total  $I_{in}$ produces a spike-rate  $f(I_{in})$ , which itself produces the feedback current  $I_{sse}$ . Therefore, the problem of simultaneously fulfilling (i) and (ii) is to find the selfconsistent frequency  $f^*$  such that,  $f^* = f[I_0 + I_{sse}(f^*)]$ .

The solution to this problem can obtained by a geometrical construction. It corresponds to the crossing point  $(I_{in}^*, f^*)$  between the two response functions measured on the NS unit (see Fig. 6). From the first response, we call  $f_1 = f_1(I_{in})$ , which is shown by the blue line in Fig. 9. From the second response, we invert the variables to get,  $f_2 = f_2(I_s) = f_2(I_{sse})$ , shown in



FIG. 9. (a) Top: Spike trace of the excitation bump. The blue line schematically denotes the applied external subthreshold current  $I_0$  with the short excitation  $I_{\Delta}$  that starts the bump, and inhibition that terminates it. Bottom: The instantaneous spike-rate emission f of the neuron (black line). The self-synaptic current  $I_{sse}$  build-up during excitation and relaxation during inhibition (red line). The state has an external continuous sub-threshold  $I_0 = 44.7\mu A$ . (b) Geometrical construction to determine the self-consistent frequency  $f^*$ . Insets:  $f^*$  as function of  $R_W$  and  $I_0$ . The parameters are  $\tau_s = 0.47$  s,  $I_0 = 44.7\mu A$ ,  $+I_{\Delta} = 22.9\mu A$ ,  $-I_{\Delta} = -24.4\mu A$ . For clarity, the number of spikes is reduced to 1/10 of the originally measured amount.

dotted yellow line in Fig. 9 (and compare to Fig. 6). From (i) above we have  $I_{in} = I_0 + I_{sse}$ , then we get  $f_2 = f_2(I_{in} - I_0)$ , which is the yellow solid line in Fig. 9. The geometrical solution is obtained requiring  $f_1 = f_2$ , which gives the crossing point  $(I_{in}^*, f^*)$ , as shown as a red cross in Fig. 9.

We can further explore how the self-consistent firing rate  $f^*$  systematically depends on two main parameters of the model, as we show in Fig. 9. One is the synaptic weight  $R_W$ , that controls the intensity of the feedback current. This intensity is controlled by the "neurotransmitter" charge  $Q_N$ , which is modulated by the synaptic resistor  $R_W$  (see Fig. 4 and Section II D). The other parameter is the external current intensity  $I_0$ , which is always kept *sub-threshold* for the activity bump. One could in principle set  $I_0 = 0$  and still realize a bump. The requirement is a strong enough feedback, such that at  $I_{sse} > I_{min}$ .

The continuous dependence of the  $f^*$  on the model parameters is shown in the insets of Fig. 9.

One of the long-standing issues in theoretical models of this type of dynamical attractors concerns their stability with respect to perturbations, since it is based on a positive feedback loop [19, 40]. Therefore, it is important to note a conceptual point in this regard. The methodology that we adopt in the present work is based on the *physical* implementation of a theoretical model. Thus, the stability of the dynamical attractor model is directly demonstrated by construction. Namely, if it works, therefore it must be stable. Nevertheless. we may further demonstrate this feature by explicitly applying strong external perturbations to the activity bump state. This is shown in Fig. 10. Despite these strong perturbations, even halting the spikes in the inhibitory case, the dynamical attractor return promptly to its stable state, as shown in the inset. A key feature for the stability, however, is that the perturbations should not last much longer than the synaptic timescale  $\tau_s$ . Hence, we may say that this state can keep a shortterm memory encoded by  $f^*$  during that characteristic timescale.



FIG. 10. Stability of the bump attractor state. Top panel: an excitatory current pulse of intensity  $+I_{\Delta} = 22.9\mu$ A (blue line) is applied during 1.2s, producing an increase in the firing rate, which rapidly relaxes after the perturbation stops. Bottom panel: an inhibitory perturbation  $-I_{\Delta} = -24.4\mu$ A is applied during 0.8s producing a halt on the spiking. The bump recovers rapidly after the perturbation stops. Inset: evolution of the synaptic currents  $I_{s1}$  and  $I_{s2} = I_{sse}$  during the perturbation. The parameters are  $\tau_s = 0.47$  s,  $I_0 =$  $44.7\mu$ A. The number of spikes is reduced to 1/5 for the sake of clear display.

Finally, from these stability considerations, it follows that to stop the active bump state one needs to apply an inhibitory current pulse  $-I_{\Delta}$ , which is sufficient long and strong. More precisely: (i) its magnitude has to be such that the total input current  $I_{in} = I_0 + I_{sse} - I_{\Delta} < I_{min}$ , i.e. falls below the onset of excitability so stops the firing and allows the feedback to relax; (ii) its duration, therefore, should be longer than  $\tau_s$ , such that the self-synaptic current  $I_{sse}$  relaxes sufficiently, as shown in Fig. 10, such that the condition (i) is fulfilled.

#### C. Self-inhibition: Adaptation and Bursting

In this section, we turn to another minimal motif, namely that of neuron self-inhibition (see Fig. 7). As we shall see, this motif produces two important spiking modes: adaptation and bursting. These modes will be the basis for the multi-neuron motif for central pattern generators, which we shall describe later in the next section.

#### 1. Adaptation

Adaptation is a relevant function in neuroscience. An example of this type of self-inhibition is the afterhyperpolarization current  $I_{AHP}$ , which is mediated by Ca<sup>2+</sup> (see Ch.10 in [13]). Similarly, as mentioned in the previous section, this motif may also be realized by an external connection between the neuron's own axon and its dendrites. This case is called an inhibitory autapse, which is now known to be a common feature of some neocortical and PV neurons, with the function of regulating the rate of spike emission [42, 45, 47, 48]. Self-inhibition is also a main ingredient of two of the most popular schematic theoretical models, Izhikevich and AdEx [3, 49]. In both models, the self-inhibition is described by a second dynamical equation for the adaptation or recovery variable.

Below, we shall explore the dynamical behavior of this important motif by means of our hardware implementation. In this case, the system of equations of our theoretical model (9-12) becomes,

$$C_m \frac{dV}{dt} = -\frac{V}{R_m[S] + R_a} - I_{ssi} + I_0$$
(21)

$$V_{pre} = \frac{V}{R_m[S] + R_a} R_a \tag{22}$$

$$\tau_s \frac{dI_{s1}}{dt} = -I_{s1} + I_\delta H(V_{ap} - \Omega) \tag{23}$$

$$\tau_s \frac{dI_{ssi}}{dt} = -I_{ssi} + I_{s1} \tag{24}$$

where we now need to excite the aLIF neuron with a supra-threshold external current  $I_0$ , unlike in the previous section. Also, we rename the synaptic current  $I_{s2}$  as  $-I_{ssi}$  to denote the synaptic self-inhibition. The circuit configuration is identical to that shown in Fig. 8 for the self-excitation, with the sole difference that the mirrored current  $I_{s2}$  is now of inverted polarity.

While it is possible to have some degree of adaptation in the spike-by-spike regime, when  $\tau_m > \tau_s$ , the most natural, interesting, and biologically relevant case is the rate-code regime, i.e. for  $\tau_m \sim 1/f \ll \tau_s$ . Hence, we focus here on the latter. In Fig. 11 we show the behavior of the aLIF with a self-adapting current. The adaptation results from the build-up of the inhibitory current intensity  $I_{ssi}$  on the synaptic sub-circuit, which occurs within the synaptic timescale  $\tau_s$ . The total current exciting the neuron gets reduced to  $I_{in} = I_0 - I_{ssi}$ , which leads to the reduction of the firing rate with respect to the original one  $f(I_0)$ . This phenomenon of spiking rate reduction is called frequency adaptation.

As we did before, we may also consider this state as a self-consistent problem, where we need to find the spike rate  $f^*$ . Now, the self-consistent condition is given by  $f^* = f^*(I_{in}) = f^*[I_0 - I_{ssi}(f^*)]$ . As we discussed before, in Section IID Eq. 16, the response function  $I_{ssi}(f) \approx -Q_N f$ , i.e., identical to the response function  $I_{sse}(f)$  with a change of sign. So the inhibitory current represents a discharge of  $C_m$ .

In Fig. 11 we show the geometrical construction for the case of a negative self-synaptic current, which follows the same steps outlined before.

Similarly, as in the case of self-excitation, the selfconsistent frequency can be controlled by modulating the intensity of the feedback current. This can be achieved by varying the model parameters, such as  $R_W$  and  $I_0$ , as shown in the insets of Fig. 11.

Also similarly as done before for the bump, the stability of the adapted self-consistent state can also be tested by applying perturbations. Our results are shown in Fig. 12, where we demonstrate very good stability.

Before leaving this section we should make a final point. As already mentioned before, it is one's choice to adopt one or two leaky-integration blocks. This choice can be made in both, the excitatory or the present inhibitory case. Below we explicitly write the model equations, where we adopt the simplified notation of Eq. 1, for the sake of clarity, and adapt them for the case of one inhibitory leaky-integration stage. The system reads,

$$C_m \frac{dV_m}{dt} = f(V_m) - I_{ssi} + I_0 \tag{25}$$

$$\tau_{\rm s} \frac{dI_{ssi}}{dt} = -I_{ssi} + I_{\delta} H(V_{ap} - \Omega) \tag{26}$$

The reader may verify that these equations now share the same form as the Izhikevich and AdEx models [3, 49]. The  $I_{\delta}$  term represents the spikes due to the action potentials  $V_{ap}(t)$ , which are analog to the potential spikes of fast variable V(t) in the theoretical models. In all cases, these spikes are identically leaky-integrated by the recovery variable.

#### 2. Bursting

We have adopted the intuitive perspective of considering states as the solution of self-consistent problems. Therefore, it is interesting to pose the following question: what would happen if we increase the intensity of the adaptation, so that the resulting total



FIG. 11. Adaptation traces. (a) Top: Spiking trace showing the progressive reduction of the frequency. The blue line indicates the external current input  $I_0 = 92.8\mu$ A. Bottom: The instantaneous spike-rate f. The red line shows the selfsynaptic adaptation current  $I_{ssi}$  builds-up over a timescale  $\tau_s \approx 1$  s.  $f^*$  denotes the self-consistent asymptotic frequency. (b) Geometrical construction to determine the self-consistent frequency  $f^*$ . Insets:  $f^*$  as function of  $R_W$  and  $I_0$ . The number of spikes is reduced to 1/20 for the sake of clear display.

input current to the neuron falls below the excitability threshold? Namely,  $I_{ssi}$  is such that  $I_{in} = I_0 - I_{ssi}(f) < I_{min}$ , so that the strong self-inhibition would stop the firing, hence the self-consistent solution is no longer possible.

Let's analyze what would be the expected behavior. The self-inhibition takes a time  $\tau_s$  to build up and stop the firing. However, this quiescence provokes the relaxation decay of the feedback current, within a timescale  $\tau_s$ . Recalling that  $I_0 > I_{min}$ , the total input  $I_{in} = I_0 - I_{ssi}$  will grow as  $I_{ssi}$  decreases and, eventually, the neuron will get re-excited and start spiking again. Thus, this dynamical bi-stability points to periods of spike emission alternating with quiescent ones. Such a state is called bursting, which is another basic neuro-computing primitive of the self-adaptation motif.

The bursting state is implemented by the NS unit as



FIG. 12. Stability of the frequency-adaptation state. Top panel: an excitatory perturbation current pulse of intensity  $+I_{\Delta} = 22.6\mu A$  (blue line) is applied during a short period. Produce an increase in the firing rate, which rapidly relaxes after the perturbation stops. Bottom panel: an inhibitory perturbation  $-I_{\Delta} = -24\mu A$  (red line) is applied producing a halt on the spiking. The adapted state recovers rapidly after the perturbation stops. Inset: evolution of the synaptic currents  $I_{s1}$  and  $I_{s2} = I_{ssi}$  during the perturbation. The parameters are  $\tau_s = 1$  s,  $I_0 = 92.8\mu A$ . The number of spikes is reduced to 1/20 for clarity.

we show in Fig. 13. From the qualitative discussion, it should be clear that the bursting state is controlled by the two timescales  $\tau_m$  and  $\tau_s$ . The latter controls the alternation period or burst cycle, and the former the inter-spike intervals during each burst. Hence, we have  $\tau_m \ll \tau_s$  so the neuron is in the rate-coding regime. Moreover, from the previous discussion, one should expect that the intra-burst spiking frequency has to evolve around the excitability threshold  $I_{min}$ . As shown in Fig. 13 this is indeed the case, with  $I_{ssi}$ oscillating around the threshold  $I_{min}$  within a fraction of a  $\mu$ A.

We may gain further and deeper analytic understanding of those small oscillations. This is revealed by a simple analysis of the model equations, which to our knowledge has not been done before. We consider the model equations and take the time derivative of (24) and replace it into (23) to obtain,

$$\tau_s^2 \frac{d^2 I_{ssi}}{dt^2} + 2\tau_s \frac{dI_{ssi}}{dt} + I_{ssi} = I_\delta[f(t)] \tag{27}$$

This differential equation is analogue to the familiar Driven Damped Harmonic Oscillator model [50] with resonant frequency  $1/\tau_s$ . The "friction" or damping is the second term and "external driving force"  $I_{\delta}[f(t)]$ . From the behavior of this well-known system, we may expect two different states: one where the damping dominates and another where the oscillations persist. Indeed, as shown in Fig. 13 the synaptic current  $I_{ssi}(t)$  exhibits a beautiful oscillatory behavior with a period close to  $\tau_s = 1$  s, as predicted by (27).

However, our Driven Damped Oscillator has a twist, since the driving force is not arbitrary but depends on the neuron's spiking trace through the feedback loop. So we can discuss the nature of the oscillation in more



FIG. 13. Burster or pace-maker state. Top: Spike traces showing bursts of activity separated by quiescent states. Middle: The total input current  $I_{in}$  is oscillating narrowly around the threshold  $I_{min}$ . Bottom: The self-inhibitory current  $I_{ssi}$  resembling a harmonic oscillation with period  $\approx \tau_s = 1$ s, as discussed in the text.

detail. Let's assume that the system is bursting and show that this assumption is consistent. The current  $I_{\delta}[f(t)]$  consists of trains of fast spikes at frequency f separated by silent periods, thus having a rhythmic time dependence with period  $\tau_s$ . The trains of current spikes are twice leaky-integrated with timescale  $\tau_s$ , therefore  $I_{ssi}(t)$  builds up within the same timescale as the period. Similarly, during the quiescent phase, the inhibitory current relaxes on the same timescale. Then, the periodic build-up and relaxation of  $I_{ssi}(t)$  modulates the firing behavior of f(t), hence of  $I_{\delta}$ , and the bursting state is consistent. The point to make is that the "driving force"  $I_{\delta}(t)$  is in resonance, as has the trains of spikes are emitted at a frequency  $1/\tau_s$  which is the natural frequency of the oscillator (27). Within the same line of reasoning the simple adaptation can be understood as an over-damped oscillation.

# IV. CENTRAL PATTERN GENERATORS

We are now ready to start building general functional spiking neural networks. We shall begin by considering the simplest multi-neuron motif, namely two neurons coupled by dynamical synaptic currents [20]. We shall focus on implementing an SNN whose neurons emit periodically alternating bursts. Such a 2-neuron network is called a Central Pattern Generator (CPG) and has a very important role in neuroscience [32]. This type of spiking activity is fundamental for motor systems with periodic dynamics, such as heartbeats, walking, eating, etc. Moreover, CPGs are also a key component in robotics, self-propelled systems, and biomedical devices. Therefore, our simple and systematic methodology may open an interesting avenue for applications.

The CPGs involve an external excitation that induces the basic tonic spiking of each neuron, which is then sculpted by inhibitory synaptic currents acting on different timescales. As this is a vast field, we shall adopt the review of Marder and Bucher [32] as guidance to select a few paradigmatic systems to build. Nevertheless, in doing that we shall also obtain new insights provided by the present hardware-based approach.

We shall consider three different basic CPGs [32] that correspond to two different two-neuron motifs (d) and (e) in Fig. 7. One has master-slave or feed-forward inhibition architecture, while the other has recurrent connections of mutual inhibition. In the model circuits here, we adopt all synaptic currents, intra- and inter-neuron, as inhibitory  $\alpha(t)$ -type functions.

#### 1. Master-Slave CPG

We start with the simplest case, motif (d) in Fig. 7. Such CPG has a bursting neuron (N1), acting as a pacemaker, that sculpts the firing rate of a regular spiking neuron (N2) by projecting inhibition. The pace-making neuron is realized by a bursting neuron, as discussed in the previous section. The slave neuron shows regular spiking when uncoupled, as shown in Fig. 14. When the inhibitory projection is introduced, the slave neuron develops a burst spiking pattern that is the inverse of its master's (see Fig. 14). Hence, the two-neuron system displays a sequence of alternating bursts which is characteristic of CPGs.

A requirement to achieve this state is that the interneuron synaptic projection from the master has to be strong enough to silence the slave, i.e. to drive the slave under its excitation threshold. So for the currents acting on N2, we have

$$I_{in} = I_0 - I_{spi} < I_{min} \tag{28}$$

where  $I_{spi}$  denotes the synaptic projected-inhibitory of N1 on N2. The intensity of the projection can be easily controlled by the resistance  $R_W$  of the  $I_{spi}$  synaptic circuit.

#### 2. Mutual-Inhibitory CPG

We turn now to the second motif, with mutual inhibition. In this case, we shall consider two qualitatively different possibilities: two coupled bursters and two coupled adaptive neurons. For simplicity, we shall consider symmetric systems. However, it is important to acknowledge that inherent variability exists in all hardware as a result of manufacturing tolerances. Nevertheless, as with biological neural neurons that are also non-identical, the emergent function has to be robust to that type of variability.



FIG. 14. CPG master-slave: (top) Uncoupled case: N1 is a pace-making burster and N2 is a regular tonic spiking neuron. (bottom) Coupled case: the N2 slave is inhibited by the activity of N1 creating a complementary bursting pattern. Parameters are  $\tau_{ssi} = 1$ s for the pace-making and  $\tau_{spi} = 1$ ms for the fast inhibitory projection.

Two mutually coupled pacemakers present a more straightforward case; therefore, we begin our discussion with this scenario. We consider two neurons that, if decoupled, both produce the same bursting (cf III C 2), up to the intrinsic variability just mentioned. Therefore, the actual observed behavior is of two similar periodic bursting but not identical, as shown in Fig. 15. By introducing coupling in the form of projection of mutualinhibition (see motif (e) in Fig. 7), we create anticorrelation in the respective firing patterns. As we can see in the second panel of Fig. 15 the state of the twoneuron network finds a compromise where they burst in anti-phase. Therefore, they both lock into a single common frequency. This emerging frequency is close to the uncoupled ones, but relatively reduced due to the global effect of additional inhibition.

In this case, it is interesting to observe the synaptic currents, which we show in Fig. 15. They bring interesting insights. Following the green dotted line we can correlate the burst of each neuron with the respective effects on their synaptic currents. The first thing to realize is that the burst induces  $\alpha(t)$ -type currents, which can be clearly appreciated. The maximum of the  $\alpha(t)$  indicates the respective time-constants, which we denote  $\tau_{ssi} = 0.47$  s and  $\tau_{spi} = 0.1$  s for synaptic self-inhibitory (red) and synaptic projected-inhibitory (blue).

If we focus on the synaptic currents of N1, shown in the third panel of Fig. 15, we can see that the burst starts when the self-inhibitory  $I_{ssi}$  decreases to almost its lowest intensity. Conversely, the burst is terminated shortly after due to the build-up of  $I_{ssi}$ , i.e. occurring within a timescale somewhat shorter than  $\tau_{ssi}$ . On the other hand, we observe that the anti-synchrony is due to the strong inhibitory projection, which has a relatively faster timescale  $\tau_{spi}$  and prevents the superposition of bursts. Similar considerations can be made for the other neuron's currents. We can also observe that the different initial traces in the two decoupled neurons (top panel) also reflect some differences in the respective produced



FIG. 15. CPG mutual inhibitory pace-maker. (a) Uncoupled case: N1 and N2 are pace-making bursters with nominal equal but in practice similar frequencies. (b) Coupled case: N1 and N2 settle into a common anti-synchronous bursting pattern. (c) Synaptic currents of N1: In red the self-inhibitory  $I_{ssi}$  with  $\tau_{ssi} = 0.47$  s. In blue the N1 to N2 projected inhibition  $I_{spi}$  with  $\tau_{spi} = 0.1$  s. The green line marks the beginning of an N1 burst. (d) idem for the synaptic currents of N2. For clarity, the number of depicted spikes is reduced by 1/5 of the originally measured amount.

currents. Nevertheless, the two neurons robustly lock to a common and stable pattern of alternated bursts.

It would be interesting, but beyond the scope of the present study, to perform a full exploration of the different emergent states of the two coupled bursters. Nevertheless, we can qualitatively discuss some relevant aspects regarding the relative timescales, which is a new feature.

In the case discussed above, we note that both, the duration of the bursts and of the projected inhibition  $\tau_{spi}$ , are relatively short, leading to the anti-synchrony already discussed. However, one may choose to increase  $\tau_{spi}$ , so that is significantly longer than the bursts. Then the projection from, say N1 to N2, will be delayed concerning the burst in N1. This projected inhibition would make the burst of N2 in anti-synchrony less likely. The result is that the emergent state would be that of synchrony between N1 and N2. This situation is qualitatively similar to that analyzed by van Vreeswijk et al. for the case of two simple spiking neurons coupled by a retarded mutual inhibition [20].

We may finally turn to another, more subtle form of CPG, which shares the same motif (e) in Fig. 7, but is found in a different regime. This CPG is realized with two neurons that exhibit spike-frequency adaptation, i.e. are tonic spiking when uncoupled (cf III C1). This type of CPG model is a classic topic of neuroscience, as it finds a biological realization in the heartbeat neural activity of the leech [33, 34, 51].

The behavior of this mutually-adaptive CPG is shown in Fig. 16. It also shows alternate burst emission but with a different mechanism. We note that for illustrative purposes, we have maintained the synaptic time constants the same as in the previous case. The present mechanism is achieved by mainly driving N1 and N2 to the adaptation regime, reducing the intensity of the self-inhibition (see III C 1).



FIG. 16. CPG mutual inhibitory adaption. (a) Uncoupled case: N1 and N2 are adaptive neurons with tonic spiking. (b) Coupled case: N1 and N2 settle into a common anti-synchronous bursting pattern. (c) The instantaneous frequency rate of N1. The green lines denote the beginning and end of a burst. The horizontal dash-dot line indicates the adapted frequency  $f^*$  when the neurons are decoupled. (d) Total input current  $I_{in}(t)$  to N1. The beginning and end of the burst coincide with the crossing of the excitation threshold  $I_{min}$ . (e) Synaptic currents of N1: In red the self-inhibitory  $I_{ssi}$  with  $\tau_{ssi} = 0.47$  s. In blue the N1 to N2 projected inhibition  $I_{spi}$  with  $\tau_{spi} = 0.1$  s. (f) idem for the synaptic currents of N2. For clarity, the number of depicted spikes is reduced by 1/5 of its original measured number.

As seen in the Fig. 16 the CPG traces show qualitative differences with respect to the previous case. One is that the burst shows some overlap. Also in contrast to the previous case, we can now observe a significant frequency modulation, where f(t) decreases at the onset and the termination of the burst. This modulation is correlated to the total input current  $I_{in}(t)$ , which shows a similar behavior. Note that it crosses the excitability threshold  $I_{min}$  as it produces the bursts.

To appreciate the mechanism, it is essential to look at the synaptic currents generated by each neuron. They are shown in the bottom panels (e) and (f) of Fig. 16. As before, in red is the self-inhibition  $I_{ssi}$ , and in blue is the projected inhibition to the other neuron  $I_{spi}$ . The green dotted lines are a guide to indicate the beginning and end of the burst of N1. The analysis for N2 would be the same.

We focus on the start of the burst of N1. We observe that its self-inhibition  $I_{ssi}$  is decreasing towards

its minimum [red line in panel (e)], similarly as in the CPG case discussed before. However, unlike that case, now N2 is firing and intensively projecting its inhibition  $I_{spi}$  on N1 [blue line in panel (f)]. In consequence, the reason why N1 starts to fire is only due to the decrease of its self-inhibition. We may say that N1 recovers and escapes from the grip of N2. This is one of the four typical CPG mechanisms and is indeed called "intrinsic escape" [23, 52].

We may also look at the end of the burst phase. There we observe that both,  $I_{ssi}$  and the received projection  $I_{spi}$  are on the rise, so they both contribute to inhibiting N1. Moreover, the projection of N2 is *necessary* to increase the inhibition of N1 and render it quiescent. This is because N1 is now an adaptation neuron and not a pacemaker as in the previous case.

This last observation is relevant because it explains an evident qualitative difference between the patterns of the two modes of CPG that we considered. By comparing the spiking traces of Figs. 15 and 16, we observe that in the first case, the bursts do not overlap in time, while in the second they do. The reason is that in the first case, the ability to burst is an intrinsic feature of each neuron, while the inhibitory projection solely creates an anti-correlation between them and lengthens the period. In contrast, in the second case, the neurons do not have the pace-making ability to quiet themselves. Thus the overlap of activity is a *necessity*. This qualitative difference is a robust feature that may help in the classification and understanding of CPGs in biological systems. For instance, the heartbeat of the leech is a classic CPG animal model and is a clear example of overlapping bursts [51]. On the other hand, the CPG of the stomatogastric ganglion of crustaceans, which is another paradigmatic animal model, is a clear example of pace-makers with anti-correlations [32].

# V. EXTENSION TO OXIDE MEMRISTORS AND TO VLSI

In this work, we have introduced a circuit implementation of a general purpose, theoretical based, neuro-synaptic model using solely conventional electronic components. This has the evident advantage that the methodology can be easily reproduced at a very affordable cost and wide availability.

Nevertheless, our methodology also opens an exciting perspective for researchers in material science working on memristive systems. In fact, our circuit can be adopted as a *template* for testing memristive devices and building small to mid-scale functional networks. More specifically, there are two places where memristors may find their place. The first one is in the neuron stage. There, the memristor device (thyristor-plus-resistor) can be replaced by a *volatile* oxide memristor, such as VO<sub>2</sub>, V<sub>2</sub>O<sub>3</sub>, V<sub>3</sub>O<sub>5</sub>, NdNiO<sub>3</sub>, SmNiO<sub>3</sub>, NbO<sub>2</sub>, etc [26, 53–57].

The second place where, in this case, a non-volatile,

oxide memristive can replace a conventional component in our NS template would be the synaptic resistance  $R_W$ . This resistance controls the intensity of the synaptic coupling  $W_{ij}$  between two neuron units *i* and *j*, in the language of artificial intelligence systems. Non-volatile oxide memristors such as TiO<sub>2</sub>, Ta<sub>2</sub>O<sub>5</sub>, HfO<sub>2</sub>, etc, are popular choices for synaptic cross-bars neuronal networks [26, 58]. The plasticity in the resistance modulation of oxide memristors may further endow the network with learning ability [59].

Another interesting direction to extend our work would be to adopt the NS design as a model to implement a very large scale integrated (VLSI) chip. This would enable the implementation of massive numbers of neuro-synaptic units, or alternatively independent neuron and synaptic units. In fact, the porting of the neuron unit has already been reported [17], where the function of the thyristor was emulated by two transistors, hence keeping the simplicity of the model. On the other hand, the synaptic stage requires current mirrors, which are standard to VLSI and have already been implemented for a singlestage of leaky-integration [12, 60]. Hence, a two-stage integration to implement a bio-mimetic  $\alpha$ -type synaptic current should present no significant impediment.

While a distinctive feature of our implementation is its simplicity, we should also mention another important characteristic, relevant to VLSI. Contrary to most current systems, our neuron unit is not based on the subthreshold concept [9], therefore one may expect that its VLSI implementation should not be so severely affected by the device matching problem, which remains an unsolved issue [11, 15].

#### VI. CONCLUSIONS

Here, we have introduced a novel neuro-synaptic device, which may be a platform to implement general spiking neural networks with dynamical bio-mimetic synapses. Our unit may seem like just one more proposal in an already crowded field. We would like to claim that it is potentially a disruptive new paradigm.

A lot of overlap and confusion exists in the field of hardware-based neuromorphic systems. So we may first distinguish our approach by enumerating what our system is not: (i) it is not based on electronic hardware working in the sub-threshold regime, as CMOS chips are. Thus, it may be less affected by the variability mismatch issue; (ii) it is not based on digital hardware, which is not well adapted for the continuous non-linear dynamics of excitability characteristic of spiking systems; (iii) it is not based on oxide electronic systems that are still under research in material science, and are also affected by the mismatch issue; (iv) it is not based on software simulations on neuromorphic chips, such as FPGAs, which present the issue of matching biological timescales [34].

We may also enumerate what our system is: (i) it is

a general spiking neuro-synaptic model from textbooks, that is defined by a set of differential equations that we implemented in hardware; (ii) it is implemented with elemental electronic circuitry, which provides a physical embodiment of the differential equations; (iii) it is implemented in a clear, direct, and quantitative manner, where every model parameter has a hardware correlate enabling full tunability; (iv) its electronic components

are widely available and economical, so our system can be immediately duplicated and the data reproduced; (v) it provides a direct hardware implementation with continuously tuned timescales, so it is ready to be deployed for applications in robotics, medical devices, control systems, etc.

#### VII. ACKNOWLEDGMENTS

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#### VIII. APPENDIX

Here we provide details on the circuit implementations of our NS unit. We shall describe each block in detail. We also provide the values of all components used in the different figures at the end, in Table III.

While the circuits can be easily implemented in breadboards, we also supply the printed circuit board (PCB) designs along with a list of components in github: https://github.com/lps-ssn/neurosynaptic

In Fig. 17 we reproduce the schematic circuit of Fig. 3, where we have now split the circuit into slightly different modular blocks. The convenience of this should be evident, as the two green blocks are identical. Moreover, with this partition, the system is fully modular. For instance, one may construct a network of neurons that communicate  $\delta(t)$  spikes without any delay by combining the red neuron blocks. Alternatively, one may just add one green block at the output of the pre-neuron to implement  $\exp(-t/\tau_s)$  synaptic current coupling. One may also add two green blocks, as in the schematic circuit, to implement the  $\alpha(t/\tau_s)$  synaptic coupling. Another important feature, as we shall show below is that the last green block has two further options, one may implement an excitatory or an inhibitory synaptic current, both of either  $\exp(-t)$  or  $\alpha(t)$  type.

#### A. Neuron Block

The excitatory part of the neuron circuit has been implemented in [28], here we add the synaptic coupling. The circuit is illustrated in Fig. 18. The voltage spikes are produced on the "axon hillock" resistor  $R_a$ . That voltage spike needs to be transformed into a strong



FIG. 17. Details of the neuro-synaptic unit circuit blocks. The neuron block is in the red box, while the blocks of two synaptic stages are in the green boxes.

current impulse so one upstream neuron is able to excite, i.e. to drive spikes on a downstream neuron. Moreover the intensity of the current needs to be modulated so as to realize the synaptic weights  $W_{ij}$  between the two neurons *i* and *j*. The components to replicate the circuit are listed in Table I below.

This is implemented through a simple solution, adopting a conventional current source configuration taken from the data sheet of the ALD1105PBL chip (has 2 matched pairs of CMOS transistors).

The output synaptic current is  $\delta(t)$ -like, and is implemented by a rectangular pulse of current with an intensity controlled by  $R_W$ . In the range of interest  $I_{out} \approx 200 \mu$ A, i.e well within the excitatory current interval, it can be approximated by  $I_{out} \approx 4V/R_W$ . An important point to make is that  $R_W$  can be replaced by a non-volatile memristor in the implementation of learning neuronal networks.

The duration of the rectangular pulse is controlled by the condition  $V_{spike} > \Omega \approx 0.7$  V i.e. when the spike overcomes a the synaptic threshold  $\Omega$  (cf Eq.11 in the main text).

We can estimate some typical values to see that our neuron circuit can easily match bio-compatible timescales. The duration of a spike is easily estimated as it is directly given by the discharge timescale of  $C_m$  on the axon hillock resistor  $R_a$ . For typical values  $C_m = 1\mu$ F and  $R_a = 1$  k $\Omega$  we obtain  $\tau_a = 1$  ms, which is a typical biological timescale.

We adopted the thyristor STMicro P0118MA, which has a trigger current  $I_{GT} \approx 2\mu$ A. From this value and the leaky integration time constant  $\tau_m$  we can obtain an approximate expression for the firing rate for a constant input current  $I_0$ . The capacitor  $C_m$  charges approximately linearly with  $V_C(t) \approx (I_0/C_m)t$ , hence the gate current follows the simple expression  $I_G(t) \approx$  $V_C/R_m = (I_0/R_m C_m)t = I_0(t/\tau_m)$ . The condition

Component	Mfr	Mfr. #	Amount
Thyristor	STMicro	P0118MA	1
Capacitor	Nova	CCC-52	1
Resistor	Nova	CBR-11	2
MOSFET	ALD, Inc.	ALD1105PBL	1
Trimmer Resistor	Bourns	3296P-1-203LF	1

TABLE I. List of Materials for the Neuron plus  $I_{\delta}$  block.



FIG. 18. Schematic of the Neuron plus  $I_{\delta}$  current block. Q1, Q3 and Q4 are CMOS transistors from the ALD1105PBL chip. The memristor that implements the voltage-gated conductance channel of the axon hillock is implemented with a thyristor (STMicro P0118MA) with  $R_m$  connected between the anode and the gate (blue box). The action potential spike voltage  $V_{ap}$  results from the fast discharge of  $C_m$  on  $R_a$  when the memristor is in the lo-R state.

to fire a spike is that the gate current overcomes its switching threshold  $I_G > I_{GT}$ . Hence,  $I_G \approx 2\mu A = I_0 ISI/\tau_m$ , where ISI is the interspike interval. Thus, the firing rate results  $f(I_0) \approx (I_0/2 \ \mu A)/\tau_m$ . For typical values  $I_0 = 60\mu A$ ,  $R_m = 1 \ M\Omega$  and  $C_m = 1\mu F$ , we get  $f \approx 30 \text{Hz}$ , which is bio-compatible.

# B. Synaptic Block

The detailed circuit that implements the green blocks of the schematic model of Fig. 3 is shown in Fig. 19. The components are listed in the Table II below. The circuit implements the leaky-integration of the input with a timescale given by  $\tau_s = R_s C_s$ .

This circuit equation is

$$dq/dt = -q/\tau_s + i_{in} \tag{29}$$

hence,

$$dV_C/dt = -V_C/\tau_s + i_{in}/C_s \tag{30}$$

and,

$$\tau_s dI_R/dt = -I_R + i_{in} \tag{31}$$

which is exactly the form of Eqs.2, 3, 11 and 12 of the main text.

Note that if  $i_{in}(t) = I_0\delta(t)$ , then  $I_R(t) \propto \exp(-t/\tau_s)$ and if  $i_{in}(t) = \exp(-t/\tau_s)$ , then  $I_R(t) \propto \alpha(-t/\tau_s)$ . As discussed in the main text.

The key feature that one needs to achieve with the circuit to implement the system of equations, is to capture the feature of successive stage current integration. Therefore, one needs to reproduce or mirror the output current on one block as the input for the next block. As was explained in the main text this is achieved with a standard solution, namely a current mirror. We implemented this using again the ALD1105PBL chip.



**Current Mirror - Excitatory** 

FIG. 19. The excitatory synaptic current circuit block. Q1, Q2, Q3, and Q4 are CMOS transistors from the ALD1105PBL chip.

Component	Mfr	Mfr. #	Amount
MOSFET	ALD, Inc.	ALD1105PBL	1
Capacitor	Nova	CCC-52	1
Resistor	Nova	CBR-11	1

TABLE II. List of Materials for excitatory and inhibitory synaptic current circuit block.

We may note that there is a price to pay for adopting this simple solution. The  $R_s$  is not connected directly to the ground, as the equations above require, but to the gate of the NMOS pair Q1-Q2 in Fig. 19. This introduces an approximation since the approximated expression for the output current becomes  $I_{Rs} \approx (V_C - 0.7V)/R_s$ .

Nevertheless, as shown in the synaptic current traces shown in Fig. 3, the two stages are well approximated by the exponential and the alpha functional forms, which in practice demonstrates that the circuit generates the appropriate waveforms. Finally, notice that the output current is positive or excitatory (i.e. outgoing), hence this block implements an excitatory synaptic current. The excitatory synaptic circuit can be easily modified to implement the inhibitory synaptic current case. It is sufficient to take out half of the current mirror, so the output becomes a "sink to ground" of a current of magnitude identical to its input. Hence, this would be a negative or inhibitory current, for instance, if connected to a neuron it would decrease the charge of the membrane  $C_m$ . The circuit implementation is shown in Fig. 20.



FIG. 20. The last synaptic current circuit block can be inhibitory or excitatory. For the excitatory case Q1, Q2, Q3, and Q4 are CMOS transistors from the ALD1105PBL chip.

For the inhibitory case Q3 and Q4 are not used.

With these synaptic blocks, we can realize the four model synaptic currents that we discussed in the text, as follows. For the AMPA, i.e. excitatory exponential-type, adopt one block (19). For the NMDA, i.e. excitatory alpha-type, adopt two identical blocks (19). For the GABA<sub>a</sub>, i.e. inhibitory exponential-type, adopt one block (20). For the GABA<sub>b</sub>, i.e. inhibitory alpha-type, adopt two blocks, the first stage (19) and the second stage (20).

# C. Model Parameters

In the table below, we list all the theoretical model parameters and their counterparts in the hardware model.

Figure No.	Theoretical model	Hardware model	Timogeolog
Figure No.	parameters	counterparts	Timescales
	$C_m$	$0.1 \ \mu F$	$\tau_m \approx 68 \ ms$
Fig. 2	$R_m$	$680 \ k\Omega$	
	$R_a$	$2 \ k\Omega$	$\tau_a \approx 200 \ \mu s$
	$C_m, R_m, R_a$	same as Fig.2	
<b>D</b> : 4	$R_W$	$10 \ k\Omega$	
Fig.4	$C_s$	22 nF	$\tau_s = 1.8 \ ms$
	$R_s$	$82 \ k\Omega$	U
	$C_m, R_m, R_a$	same as Fig.2	
	$R_{W}$	$10 \ k\Omega$	
Fig.5	$C_{s}$ for $(a)$	$47 \ \mu F$	(a) $\tau_s = 4.7 \ s$
8.0	$C_{s}$ for (b) and (c)	$1 \mu F$	(b, c) $\tau_s = 100 \ ms$
	$R_{s}$	$100 k\Omega$	(,)
	$C_m R_m R_a$	same as Fig 2	
	$B_{W}$	$5 k\Omega$	
Fig.6	C-	$47 \mu F$	$\tau_{-} = 470 \ ms$
	R R	$100 k\Omega$	18 110 1100
	C R R	same as Fig 2	
Fig.9	$B_{m}, R_{m}, R_{a}$	same as Fig.2	
	$n_W, C_s, n_s$	same as Fig.o	
Fig.10	$D$ $C_m, \pi_m, \pi_a$	same as $FIg.2$	
	$R_W, C_s, R_s$	same as Fig.8	- 00.4
	$C_m$	33 nF	$ au_m \approx 22.4 \ ms$
	$R_m$	680 k12	0.0
D: 11	$R_a$	$2 k\Omega$	$ au_a \approx 66 \ \mu s$
Fig.11	$C_s$	$10 \ \mu F$	$\tau_s = 1 \ s$
	$R_s$	$100 \ k\Omega$	
	$R_W$	$2.2 \ k\Omega$	
	$I_0$	92.8 $\mu A$	
	$C_m, R_m, R_a$	same as Fig.11	
Fig.12	$C_s, R_s, R_W$	same as Fig.11	
	$I_0$	$92.8 \ \mu A$	
	$C_m, R_m, R_a$	same as Fig.11	
Fig.13	$C_s, R_s, R_W$	same as Fig.11	
	$I_0$	$53.2 \ \mu A$	
	$C_m, R_m, R_a$	same as Fig.11	
Fig 14	self-synapse $C_s, R_s$	same as Fig.11	$\tau_{ssi} = 1 \ s$
F 1g. 14	self-synapse $R_W$	$0.27 \ k\Omega$	
	projection $C_s, R_s$	$10 \ nF$ , $100 \ k\Omega$	$\tau_{spi} = 1 \ ms$
	projection $R_W$	$1.5 \ k\Omega$	
	$R_W$	$4.7 \ \Omega$	
	N1 $I_0$	$62.0 \ \mu A$	
	N2 $I_0$	$46.5 \ \mu A$	
	$C_m, R_m, R_a$	same as Fig.11	
Eig 15	self-synapse $C_s, R_s$	$4.7 \ \mu F, 100 \ k\Omega$	$ au_{ssi} = 470 \ ms$
г ig.15	self-synapse $R_W$	$100 \ \Omega$	
	projection $C_s, R_s$	$1 \ \mu F, \ 100 \ k\Omega$	$\tau_{spi} = 100 \ ms$
	projection $R_W$	$0.4 \ \Omega$	-
	N1 $I_0$	78.8 $\mu A$	
	N2 $I_0$	$70.6 \ \mu A$	
	$C_m, R_m, R_a$	same as Fig.11	
D: 10	self-synapse $C_s$ , $R_s$	$4.7 \ \mu F, 100 \ k\Omega$	
Fig.16	self-synapse $R_W$	100 Ω	
	projection $C_s$ , $R_s$	$1 \ \mu F, \ 100 \ k\Omega$	
	projection $R_W$	0.4 Ω	
	N1 I0	$57.3 \ \mu A$	
	N2 $I_0$	52.4 $\mu A$	
1	· · · · · · · · · · · · · · · · · · ·	, <i>,</i>	

TABLE III. Parameters for figures

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