New Trends in Computational Modeling: A Neuroid-based Retina Model

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*Abstract***— It is thought that using detailed neuron-models could lead to a better understanding of how the nervous system works. However, neural networks preserve their collective computational properties, regardless of the level of description used for modeling the main building block. In this paper, we built a Neuroid-based retina model. As a result of the implementation, the Neuroid was able to reproduce the essential features of the photoreceptor response to light. Likewise, the retina model responded to specific visual effects such as simultaneous contrast, Mach bands and Hermann grid. All of these suggest that the Neuroid comprises enough functional characteristics, such that we could focus not only on the most relevant computational aspects of nerve cells, but also in the collective capabilities of large-scale neural networks.**

I. INTRODUCTION

The dynamics and computations of single neurons is required to understand how the nervous system works. Based on this argument, numerous neuron-models have been developed at different levels of description, which leads to the establishment of diverse trends in computational modeling. These trends have been considered as mutually exclusive or antagonistic. Nevertheless, from a substantial number of simulations [1], [2], it has been shown that the computational collective properties of networks composed of biophysically accurate neuron-models are preserved in networks based on simplified modeling units. This allows us to focus, not only on the computational aspects of the nerve cells but on the collective capabilities of networks as processing units.

One of the most intricate and fascinating neural networks is the retina. This tissue is located in the back of the eyeball and has a three-layer structure, with the bodies of nerve cells placed in three rows separated by two layers packed with synaptic connections [3]. Essentially, the retina is responsible for receiving and transducing light into electrical signals which are pre-processed before travelling down the optic nerve toward the brain, for further processing and image perception. Collective properties of the retina are mediated by various mechanisms such as: Feed-forward and feedback pathways, convergence and divergence patterns, lateral inhibition, sign-conserving and sign-inverting chemical synapses, and electrical couplings via gap junctions. Several

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attempts have been made for modeling retinal dynamics, some of which were based on discrete Cellular Neural Networks [4], [5], whereas others involve numerous detailed compartmental units [6], [7]. Well-known alternatives like the Integrate-and-Fire (IF) neuron-model have been also used for modeling the retina [8]. But the question arises: which is the best abstraction level for describing physiological neural networks? We believe that if different levels of description are incorporated for the development of new intermediate neuron-models, we could achieve a better understanding of the computational capabilities of neurons and, therefore, the nervous system. In fact, several efforts have been made to develop such neuron-models [9], [10], although their use has been still limited.

In this paper, we implemented a Neuroid-based retinal network to evaluate: 1) the flexibility of the model for representing singular nerve cells, such as photoreceptors, and 2) the computational capabilities of a large-scale network composed of numerous interconnected Neuroids. We focused on simulating the Cone \rightarrow ON-bipolar cell \rightarrow ON-ganglion cell pathway, rather than replicating the retinal architecture in detail.

II. METHODOLOGY

A. Modeling a single photoreceptor

Photoreceptors, like bipolar and horizontal cells, respond with graded potentials. However spike activity has been observed in photoreceptors when their K^+ conductance decreases [11]. On the other hand, although the Neuroid was developed from a mathematical description of the train of action potentials, it also provides an analog graded response. Let $s(t)$ be the stimulus that reaches the trigger zone and depolarizes the membrane to the activation threshold, *umbr*. Then the signal that propagates along the neuron's axon can be modeled as a frequency-modulated impulse train, *y*(*t*), given by (1) as follows

$$
y(t) = \sum_{n=0}^{\infty} \delta\left(t - \frac{n\beta T}{s(t) - u m b r}\right), \text{if } s(t) > u m b r. \tag{1}
$$

In (1), δ denotes a single spike, *T* is the time between two consecutive spikes, and β is the reciprocal of the Frequency-Intensity curve slope. This frequency-modulated impulse train is "demodulated" into an output signal, which resembles the triggering event, $s(t)$, and extends indefinitely after the last spike. To prevent this, and in turn, to adjust the amplitude of the output signal, two other parameters were required: a decay constant, *maxcount*, and a multiplicative factor, *K^r* .

In order to model the response of a single photoreceptor to light, we reversed the condition for pulse generation (see "Implementation" in [10]) so we could rewrite the frequencymodulated impulse train, $y(t)$, as depicted in (2)

$$
y(t) = \sum_{n=0}^{\infty} \delta(t - n[1 + \alpha(s(t) - umbr)T]).
$$
 (2)

It is worth noting that the physiological meaning of *s*(*t*), *T* and *umbr* is preserved in (2). The new proportionality constant is $\alpha = 1/β$. The luminance levels from -6 to 8 Log $cd/m²$ were rescaled to test the model over the actual range of the visual system (0 for total darkness and 1 for the brightest light). Using $T = 2$ ms, $umbr = 0.2144$ (which corresponds to -3 Log cd/m², the cone threshold based on the scheme proposed by [12]), $K_r = 1.1$, *countmax* = 32 ms, we plotted the response to a stimulus of increasing amplitude for $\alpha = 1$, 5, 10 and 20.

B. Reversing the sign of the synapses

Retinal circuits mediating contrast comprise signconserving and sign-inverting synapses [13]. We used the original synaptic coupling block defined in [10] to represent the ionotropic sign-conserving synapse, $s_{sc}(t)$, expressed as

$$
s_{sc}(t) = \sum_{i=1}^{n} w_i x_i.
$$
 (3)

Thus, to model the metabotropic sign-inverting synapse, $s_{si}(t)$, we reformulated (3) the equation $y = 1 - x$, such that it was possible to obtain the maximum output value for null inputs

$$
s_{si}(t) = \sum_{i=1}^{n} w_i (1 - x_i).
$$
 (4)

As a convention, for (3) and (4), x_i and w_i denote the *i*-th input and its synaptic weight, respectively.

C. The Center-Surround organization

Responses of retinal ganglion cells to illumination in one region of the retina are antagonized by illumination in surrounding regions. This explains why objects with specific luminance look brighter on a dark background than on a bright background. The antagonistic surround has been observed in both outer and inner retina, and it is mediated by horizontal [14], [15] and amacrine [16], [17] cells, respectively. To capture the essence of this feature, we expressed the Resulting Intensity of Light (*RIL*) as a linear combination of *n* excitatory-center (*ci*) and *m* inhibitorysurrounding (s_i) inputs:

$$
RIL = n w_c c_i + m w_s s_j. \tag{5}
$$

From (5), for the maximum level of luminance on the receptive field $(c_i = s_i = 1)$, the synaptic weights w_c and w_s can be calculated as a function of the *RIL* and the number of excitatory and inhibitory inputs.

D. Receptive Fields tessellation

Despite the irregularities exhibited by receptive fields of retinal ganglion cells, neural ensembles give a uniform highresolution visual representation. Previous work [18], [19] showed that we can achieve uniform coverage assuming a hexagonal packing of retinal ganglion cells. This is so because receptive fields arranged in hexagonal fashion will be equidistant from each other with no spaces between them, thereby providing an optimal 2D-spatial sampling scheme. Based on that, we implemented a three-layer network of 127 photoreceptors, hexagonally packed $(n = 1; m = 6)$, which would converge to 17 ON-ganglion cells through 35 ONbipolar cells (convergence ratio $= 7:1$), also in hexagonal fashion, as depicted in Fig. 1. We used (5) to calculate the

individual contribution for one single surrounding cell (ws), assuming $c_i = s_i = w_c = 1$ and $RIL = 0.6$ (normalized)

$$
w_s = (0.6 - 1)/6 \approx -0.067. \tag{6}
$$

We used Neuroids for modeling the 127 photoreceptors and the 17 ON-ganglion cells ($T = 2$ ms, $umbr = 0.05$, $\alpha =$ 1.2, $K_r = 2.1$ and *countmax* = 24 ms). All simulations were performed in LabVIEW (version 10.0, running on Acer Aspire One).

III. RESULTS

A. A Neuroid-based Photoreceptor

One single change was performed on the Neuroid's original structure to construct a photoreceptor model. Fig. 2(a) shows the model responses to a stimulus of increasing amplitude, for different values of α. As illustrated in Figs. 2(a) and (b), sensitivity to low stimulus intensities above the threshold increases linearly with this parameter. In addition, for greater values of this parameter, the input-output relation exhibited an "S" shape, suggesting some resemblance to the Michaelis-Menten function, which was plotted with the dashed line in Fig. 2(b).

Figure 1. The architecture of the Neuroid-based retina model: open circles, ON-center cells; filled circles, OFF-surrounding cells; shaded area, overlapping of receptive fields. The center-surround organization comprises one active center surrounded by six inhibitory neighboring cells packed in hexagonal fashion.

B. The Retina Model responds to illumination patterns

We implemented a three-layer Neuroid network to represent the retinal architecture. Whereas the ON-bipolar and ON-ganglion cells layers received graded signals from Neuroids in previous layers, the photoreceptor layer received 2D time varying illumination patterns, as shown in Fig. 3(a) (patterns *i*, *ii*, *iii* and *iv*). The stimulus propagated through the intermediate and bottom layers after 5 s. Thus, there was residual activity in ON-bipolar and ON-ganglion cells, even though the stimulus was no longer present. Results for simultaneous contrast under specific patterns of light stimulation are illustrated in Fig. 3(b) (patterns *i*, *ii*, *iii*, *iv*, *v*, and *vi*). Fig. 3(c) shows the response of the retinal network to the Mach bands.

Figure 2. (a) Responses of the Neuroid-based photoreceptor model to a stimulus of increasing amplitude, as a function of α .(b) The input-output relation compared to the Michaelis-Menten function (dashed line).

IV. DISCUSSION AND CONCLUSION

Several compartmental neuron-models have been

the activation threshold (*umbr*) and the sensitivity of the model (α) , we could represent both rods and cones to extend the retinal network model. As shown in Fig $3(a)$, there was no response of ONganglion cells layer to the illumination pattern *iv*. This is due to no light fell into the active center of any receptive field.

developed to reproduce response properties of photoreceptors to light [6]. Nevertheless, our functional model preserved the essential features of photoreceptor's dynamics. Therefore, by setting the appropriate values for

From Fig 3(b) we can observe how the retinal network adapted to background illumination (simultaneous contrast) and exhibited changes in brightness of specific stimulated areas (illumination patterns from *iii* to *iv* and from *v* to *vi*). Moreover, when illumination pattern was switched from *i* to

Figure 3. Responses of the Neuroid-based retina model to a time varying illumination pattern. (a) If light does not fall into the active center of any receptive field, the model will not respond, as shown for pattern *iv*, where no light is "perceived" by the model. (b) Under direct stimulation on the active centers, the edge of the illuminated area preserves its intensity, even though the stimulation intensity decreases (patterns *i* and *ii*). Patterns *iii*-*iv* and *v*-*vi* show the changes in brightness due to the effect of the background illumination (simultaneous contrast), and pattern ν shows a dark gray spot at the intersection of brighter areas (Hermann grid). (c) The Mach bands effect as edge detection mechanism for a three-value grayscale.

ii, the collective brightness altered response was evoked as a function of the intensity of the stimulus. But we cannot talk about intensity without linking this property to the size of the receptive field. For a coherent light beam, intensity is generally associated with the number of photons that reach the photoreceptors per unit time, but for diffuse light, intensity may be considered as a spatial-temporal property, which may be also described in statistical terms, such that the ganglion cells response across the receptive field may be given by the probability that one photon is absorbed by one photoreceptor.

Even though the Neuroid does not accurately replicate some aspects of actual retinal cells such as the temporal transients of photoreceptive responses, the model comprises enough functional characteristics to provide us with a holistic comprehension of the computational capabilities of neural networks as processing units. The underlying goal consists in contribute to establish a new trend in computational modeling of the nervous system. This novel trend relies on relating different levels of abstraction to develop new intermediate neuron-models [9], [10]. This might encourage researchers to form multi-disciplinary working groups and, thereby, integrate diverse areas of expertise to formulate novel solutions for specific problems. Perhaps only then, we will be able to unravel the mysteries of the mind.

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