BRIGHTNESS ESTIMATION IN A NEURAL NETWORK MODEL WITH PRESYNAPTIC INHIBITION

Veseljka Rebić[1], Mia Šetić[2] and Dražen Domijan[2]
[1]*Department of Psychology, Faculty of Arts and Sciences, University of Zagreb, Ivana Lučića 3, 10000 Zagreb, Croatia*
[2]*Department of Psychology, Faculty of Arts and Sciences, University of Rijeka, Slavka Krautzeka bb, 51000 Rijeka, Croatia*
<verebic@ffzg.hr; mia-setic@ffri.hr; ddomijan@ffri.hr>

Recent psychophysical and neurophysiological investigations showed that visual system encodes luminance and use it to estimate illumination and surface brightness. We proposed a novel neural model for luminance coding based on recurrent inhibition, from the retinal ganglion cells to the axons of the bipolar cells, which modulates the amount of sensory input that ganglion cells receive (Sagdullaev et al., 2006). Extended version of the model, where the amount of presynaptic inhibition is made proportional to the maximum luminance in the visual scene, implements gain control mechanism which adjusts the raw luminance into a measure of brightness of surface. Computer simulations showed that the model scales brightness estimates consistent with the highest-luminance-as-white anchoring rule (Gilchist et al., 2004). Simulations also showed that the model is able to act as a change detector when the presynaptic inhibition temporally lags behind the excitatory input to the ganglion cell.

Traditional assumption about early visual processing is that retinal cells with centre-surround antagonism extract local contrast or luminance discontinuities in the image. Interactions between centre and surround part of the visual field are implemented with lateral inhibition which suppress regions of uniform luminance. Luminance per se is assumed to be uninformative because it depends on the illumination which should be discounted in order to obtain surface reflectance (Neumann, 1996). However, recent theoretical and experimental developments suggest that luminance is somehow encoded in the visual system. For instance, Robilotto and Zaidi (2004) showed that discrimination between surfaces in real-world scenes is best accounted by the model which rests on the luminance encoding. Statistical analyses of the natural images suggest that luminance and contrast provide independent information about environment. Furthermore, contrast provides only relative information which is not sufficient to recover the scale of perceived shades of gray (Gilchrist *et al*., 1999).

Recently, Sagdullaev *et al*. (2006) discovered a specific form of presynaptic inhibition operating on the input to the retinal ganglion cells which enable expansion of the cells' dynamic range. The aim of this work is to explore the computational role of such presynaptic inhibition in encoding luminance and brightness perception.

Method

Neural activity of retinal cells is usually modelled using shunting mechanism. It is a form of non-linear processing which prevents unbounded growth or decline of activity. Unfortunate consequence of this mechanism is that cells lose their ability to scale their response in proportion to the input intensity. Therefore, cells are not able to encode luminance of the input. Here, we propose that presynaptic inhibition from the ganglion cells to their input

pathways from the bipolar cells solves the problem of encoding luminance. Activity of the on-centre off-surround cell, x_i, at spatial position, i, is described with

$$\frac{dx_i}{dt} = -Ax_i + (B - x_i)\left[C_i - w_i y_i\right]^+ - (x_i + D)E_i\,, \tag{1}$$

and presynaptic inhibition from the ganglion cell to its excitatory input, y_i, is given by

$$\frac{dy_i}{dt} = -y_i + x_i\,. \tag{2}$$

Presynaptic inhibition is mediated by the amacrine cells which serve as inhibitory interneurons in the retina. Term, C (S), denotes the central (surround) part of the on-centre cell's receptive field. Parameter A describes passive decay which drives activity towards zero if there is no input; B (D) defines the excitatory (inhibitory) saturation point, that is, an upper (lower) bound for an activity level that can be obtained. Function $[x]^+ = \max(x, 0)$ describes the half-wave rectification. Synaptic weight, w_i, defines the strength of presynaptic inhibition and acts as a form of a gain control mechanism which adjusts cell's sensitivity to the input. Larger values of, w_i, enable greater dynamic range for accurate representation of sensory magnitude. The centre (C) and the surround (S) part of receptive fields are obtained by convolution of spatial filters with input intensity,

$$C = \sum_p C_p I_{i+p} \text{ and } S = \sum_p S_p I_{i+p}\,, \tag{3}$$

where, C_p, and, S_p, are one-dimensional Gaussian spatial filters which model distance dependent input strengths arising from different distances from the target cell. The size of the convolution window is $-P < p < P$.

We considered two extension of the basic model in order to illustrate its usefulness in understanding perception. First, instead of fixed values for synaptic strengths it is possible to make them proportional with the maximum luminance level,

$$w = max(I_i) \text{ for } i = 1, ..., N, \tag{4}$$

which is applied to all model cells and N is the dimension of the network. Computing maximum is a global operation and this could be performed at the cortical level. Such dependence on the maximum luminance enables the model to translate a range of input values to the scale of perceived shades of gray. In other words, the model solves the anchoring problem (Gilchrist et al., 1999).

Another extension concerns the possibility that presynaptic inhibition delivers its influence on the excitatory input with certain temporal delay. This is a plausible assumption because presynaptic signal travel along a longer path and must cross two synapses before it can influence input to the target ganglion cell. In this case, eqn (2) should be replaced with,

$$\frac{dy_i(t)}{dt} = -y_i(t) + x_i(t - \tau), \tag{5}$$

where constant, τ, denotes the delay for inhibitory interneuron to receive input from the ganglion cell at time, *t*. This modification enables cells to behave as change detectors which are useful in modelling properties of the magnocellular pathway and motion perception.

Results and discussion

In order to test the model's behaviour we run a set of computer simulations. Network equations (1) and (2) are numerically solved using the Runge-Kutta method. The parameters were set to: $A=.1$, $B=10$, $D=1$, $P=4$, I_i takes the value from *1* to *10* for all, *i*, w_i were set to *1* except when noted and $\tau=0$ except in Simulation C where $\tau=1$.

Figure 1A presents a basic simulation which demonstrates the network ability to exhibit analogue sensitivity, that is, to maintain activity level proportional to the input magnitude. We varied the strength of presynaptic inhibition, w_i, from 1 (solid line), 2 (dashed line) to 3 (dotted line) in order to show how to increase the range of stimulus values that could be faithfully represented by the network activity. For comparison, fig. 1B shows network response when presynaptic inhibition is removed ($w_i = 0$ for all *i*). Without presynaptic inhibition, the model activity quickly saturates close to the maximum level, *B*. Changes in the decay rate from, $A=.1$ (solid line) to $A=.5$ (dashed line), and $A=1$ (dotted line) does not help much to restore the analogue sensitivity.

Figure 1B shows the network ability to scale its response with the maximum activity level. The same spatial pattern is presented with different amount of background illumination (dim illumination = solid line; medium illumination = dashed line and bright illumination = dotted line). Despite the great variation in the luminance range, the network was still able to maintain accurate relationship between brightness levels. The reason for this is the fact that presynaptic inhibition enables the cell to compute the ratio between input luminance and maximal luminance, that is, the cell will converge to the, $x_i = I_i/w$. In this way, the model implement highest-luminance-as-white anchoring rule. However, this rule is not the only determinant of the perceived shades of gray. Further work is needed in order to simulate a complex pattern of results which include the role of the surface size, articulation, insulation and configuration on the brightness perception (Gilchrist *et al*., 1999).

Figure 1C shows the temporal aspect of the cell's response. In particular, we investigated how the cell responds to a sudden appearance of the stimulus in the receptive field. Amplitude of cell's activity is plotted as a function of time. Input amplitude was set to $I=2$ (solid line) and $I=4$ (dashed line). As can be seen in the top row, the model cell produces activity peak at the beginning of the stimulus presentation. Peak is scaled with the input magnitude as would be expected from the action of presynaptic inhibition. However, if the activity of the inhibitory node is delayed relative to that of the ganglion cell, scaling is temporarily lost (bottom row). In this way, the ganglion cell becomes a change detector which responds vigorously to the sudden appearance of the stimulus irrespective of the stimulus strength. Such property enables the model ganglion cells to contribute to the motion perception and to the detection of temporal structure.

The present model has several advantages over previous computational proposal for how retinal cells encode luminance. Neumann (1996) suggested that luminance might be recovered from the summation of responses of on-centre and off-centre cells. However, such encoding scheme implies dependence of luminance information on the contrast which is not consistent with the findings that luminance and contrast might be independently processed in the visual system. Furthermore, it cannot exhibit change detection unless additional mechanisms are incorporated into the model. Therefore, the model with presynaptic inhibition offers greater flexibility in encoding luminance and translating luminance information into perceived brightness.

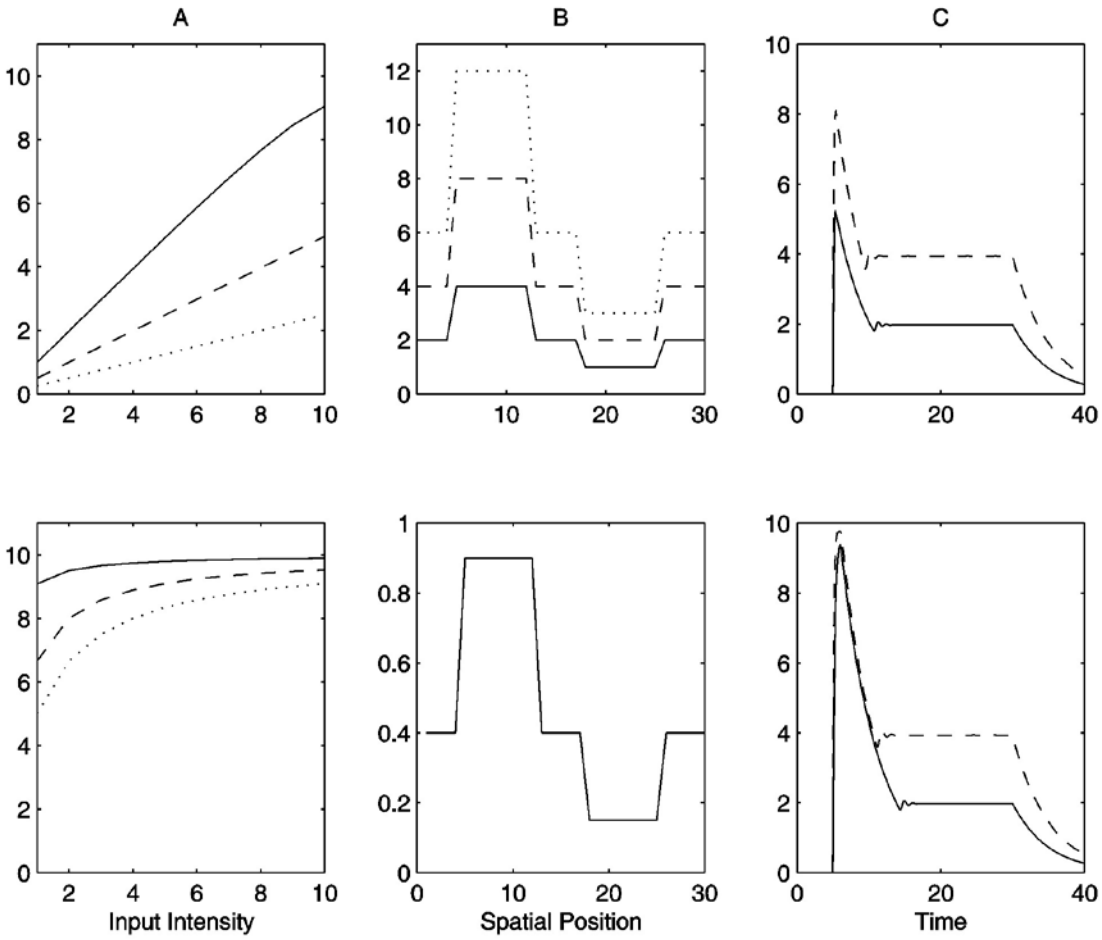

Figure 1. Results of computer simulations. A) Sensitivity to the input magnitude; cell with presynaptic inhibition (top) and cell without presynaptic inhibition (bottom). B) Implementation of the anchoring rule; input with different levels of illumination (top) and the network response (bottom). C) Temporal evolution of the cell's activity without delay (top) and with delay (bottom).

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