AN ARCHITECTURAL MODEL FOR VELOCITY-TUNED SPATIO-TEMPORAL CORTICAL FILTERS

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ABSTRACT

A linear architectural model of cortical simple cells is presented. The model evidences how mutual inhibition, occurring through synaptic coupling functions asymmetrically distributed in space, can be a possible basis for a wide variety of spatio-temporal simple cell response properties, including direction selectivity and velocity tuning. While spatial asymmetries are included explicitly in the structure of the inhibitory interconnections, temporal asymmetries originate from the specific mutual inhibition scheme considered. Extensive simulations supporting the model are reported.

1. Introduction

Recent theoretical and neurophysiological studies (Reid *et al.*, 1991; Hamilton *et al.*, 1989) pointed out that the initial stage of direction selectivity can be related to the linear space-time receptive field structure of simple cells. A large class of simple cells has a very specific space-time behavior in which the spatial phase of the receptive field changes gradually as a function of time. This results in receptive field profiles that are tilted in the space-time domain. To account for the origin of this particular spatio-temporal inseparability, numerous models have been proposed postulating the existence of structural asymmetries of the geniculo-cortical projections both in the temporal and in the spatial domains (for a review, see (Hildreth & Koch, 1987). Among them, feed-forward inhibition along the non-preferred direction, and the combination of lagged and non-lagged geniculate inputs to the cortex have been commonly suggested as the major mechanisms.

In this paper, within a linear field theory framework, we propose and analyse an architectural model for dynamic receptive field formation, based on intracortical interactions occurring through asymmetric mutual inhibition schemes.

2. Materials and Methods

Following a linear neural field approach (Krone *et al.*, 1986), we regard visual cortex as a continuous distribution of neurons and synapses. Accordingly, the geniculo-cortical pathway is modeled by a two-layer network interconnected through feed-forward and feedback connections, both inter- and intra-layers.

Each location on the cortical plane represents a homogeneous population of cells, and connections represent average interactions among populations. Such connections can be modeled by spatial coupling functions which represent the spread of the synaptic influence of a population on its neighbors, as mediated by local axonal and dendritic fields. From an architectural point of view, we assume the superposition of feed-forward (i.e., geniculate) and intracortical contributions which arise from inhibitory pools whose activity is also primed by a geniculate excitatory drive. Geniculate contributions $e_0(x,t)$ are modeled directly by a spatio-temporal convolution of the visual input $s(x,t)$ and a separable kernel $h_0(x,t)$ characterized in the spatial domain by a Gaussian shape with spatial extent σ_0 and, in the temporal domain, by a first-order temporal low-pass filter with time constant τ_0. The output $e_1(x,t)$ of the inhibitory neuron population results from the mutual inhibitory scheme through spatially organized pre- and post-synaptic sites, modeled by the kernels $k_{1a}(x-\xi)$ and $k_{1d}(x-\xi)$, respectively:

$$\tau_1 \frac{\mathrm{d}e_1(x,t)}{\mathrm{d}t} = -e_1(x,t) + \int k_{1d}(x-\xi)[e_0(-\xi,t) - bm(-\xi,t)]\mathrm{d}\xi \tag{1}$$

$$m(x,t) = \int k_{1a}(x-\xi)e_1(-\xi)\mathrm{d}\xi \tag{2}$$

where the function $m(x,t)$ describes the spatio-temporal mutual inhibitory interactions, and b is the inhibition strength. The layer 2 cortical excitation $e_2(x,t)$ is the result of feed-forward contributions collected (k_{2d}) from the inhibitory loop, at axonal synaptic sites, and the geniculate input ($e_0(x,t)$). To focus the attention on the inhibitory loop, in the following we assume a one-to-one mapping from layer 1 to layer 2, i.e., $k_{2d}(x-\xi) = \delta(x-\xi)$, consequently:

$$\tau_2 \frac{\mathrm{d}e_2(x,t)}{\mathrm{d}t} = -e_2(x,t) + e_0(x,t) - bm(x,t) \tag{3}$$

where τ_1 and τ_2 are the time constants associated to layer 1 and layer 2, respectively.

In a previous work (Sabatini, 1996), we evidenced how the steady-state solution of Eqs. (1)-(3) can give rise to highly structured Gabor-like receptive field profiles, when inhibition arises from laterally distributed clusters of cells. In this case, the effective intrinsic kernel $k_1(x)$, defined as $k_1(x-\xi) \stackrel{\mathrm{def}}{=} \int\int k_{1a}(-x',-\xi')k_{1d}(x-x',\xi-\xi')\mathrm{d}x'\mathrm{d}\xi'$, can be modeled as the sum of two Gaussians symmetrically offset with respect to the target cell:

$$k_1(x) = \frac{1}{\sqrt{2\pi}}\left(\frac{w_1}{\sigma_1}\exp[-(x-d_1)^2/2\sigma_1^2] + \frac{w_2}{\sigma_2}\exp[-(x+d_2)^2/2\sigma_2^2]\right). \tag{4}$$

To investigate how spatial asymmetries in the intracortical coupling function lead to non-separable space-time interactions within the resulting discharge field of the simple cells we varied systematically the geometrical parameters (σ, w, d) of the inhibitory kernel to consider three different types of asymmetries: (1) different *spatial spread* of inhibition (i.e., $\sigma_1 \neq \sigma_2$); (2) different *amount* of inhibition ($w_1 \neq w_2$); (3) different *spatial offset* ($d_1 \neq d_2$).

3. Results

Since visual cortex is visuotopically organized, a direct correspondence exists between the spatial organization of intracortical connections and the resulting receptive field topography. Therefore, the dependence of cortical surface activity $e_2(x,t)$ on the visual input $s(x,t)$ can be formulated as $e_2(x,t) = h(x,t) * s(x,t)$, where the symbol $*$ indicates a spatio-temporal convolution, and $h(x,t)$ is the equivalent receptive field interpreted as the spatio-temporal distribution of the signs of all the effects of cortical interactions. To test the efficacy of the various inhibitory schemes, we used a drifting sine wave grating $s(x,t) = C\cos[2\pi(f_x x \pm f_t t)]$ where C is the contrast, f_x and f_t are the spatial and temporal frequency, respectively.

The direction selectivity index[1] (DSI) and the optimal velocity obtained from the various inhibitory kernels were evaluated for different values of τ_1 and b. The optimal velocity is defined as f_t^{opt}/f_x, where f_x is chosen to match the spatial structure of the receptive field, and f_t^{opt} is the frequency which elicits the maximum cell's response. As expected, increasing the parameter b enhances the effects of inhibition, thus resulting in larger DSI and higher optimal velocities. However, for stability reason, b should remain below a theshold value strictly related to the inhibitory kernel considered. Moreover, we observe that, for almost all the cases, the strongest direction selectivity can be obtained when the intracortical time constant τ_1 has values in the range of 10 - 20 ms, i.e., comparable to τ_0 and τ_2. Larger values of τ_1 would result, indeed, in a recrudescence of the velocity low-pass behavior. For each asymmetry, Figs. 1 show the velocity tuning curves and the x-t plots, respectively, for the best cases considered. We have evidenced that appreciable DSI and a true velocity tuning can be obtained when inhibition arises from cortical sites at different distance from the target cell (i.e., ASY-2B, $d_1 \neq d_2$). In such conditions we obtained a DSI as high as 0.66 and an optimal velocity up to $\sim 6^o$/s, as could be inferred also from the spatio-temporal plot which present a marked motion-type (i.e., oriented) non-separability (see Fig. 1ASY-2B). In the architectural

[1] The direction selectivity index is defined as $DSI = (R_p - R_{np})/(R_p + R_{np})$, where R_p is the maximum response amplitude for preferred direction, and R_{np} is the maximum amplitude for non-preferred direction.

model presented in this study, spatial asymmetries were included explicitly in the extension of the inhibitory interconnections, but no explicit asymmetric temporal mechanisms were introduced. It is worth evidencing how temporal asymmetries originate from the specific mutual inhibition scheme considered, which operates, regarding temporal domain, like a quadrature model (Watson & Ahumada, 1985; Adelson & Bergen, 1985). This can be inferred by the examination of the equivalent transfer function $H(\omega_x, \omega_t)$ in the Fourier domain:

$$H(\omega_x, \omega_t) = \frac{H_0(\omega_x, \omega_t)}{1 + j\omega_t\tau_2} \left(\frac{1}{1 + j\omega_t\tau_1 + bK_1(\omega_x)} + j\omega_t\tau_1 \frac{1}{1 + j\omega_t\tau_1 + bK_1(\omega_x)} \right) \tag{5}$$

where upper case letters indicate Fourier transforms, and j is the complex variable. The terms in parentheses in Eq. (5) can be interpreted as the sum of temporal components that are approximately arranged in temporal quadrature. Furthermore, one can observe that a direct monosynaptic influence (e_1) from t he inhibitory neurons of layer 1 to the excitatory cells of layer 2, would result in the cancellation of the quadrature component in Eq. (5) .

Further improvement of the model should take into account also transmission delays between spatially separated interacting cells, theshold non-linearities, and ON-OFF interactions.

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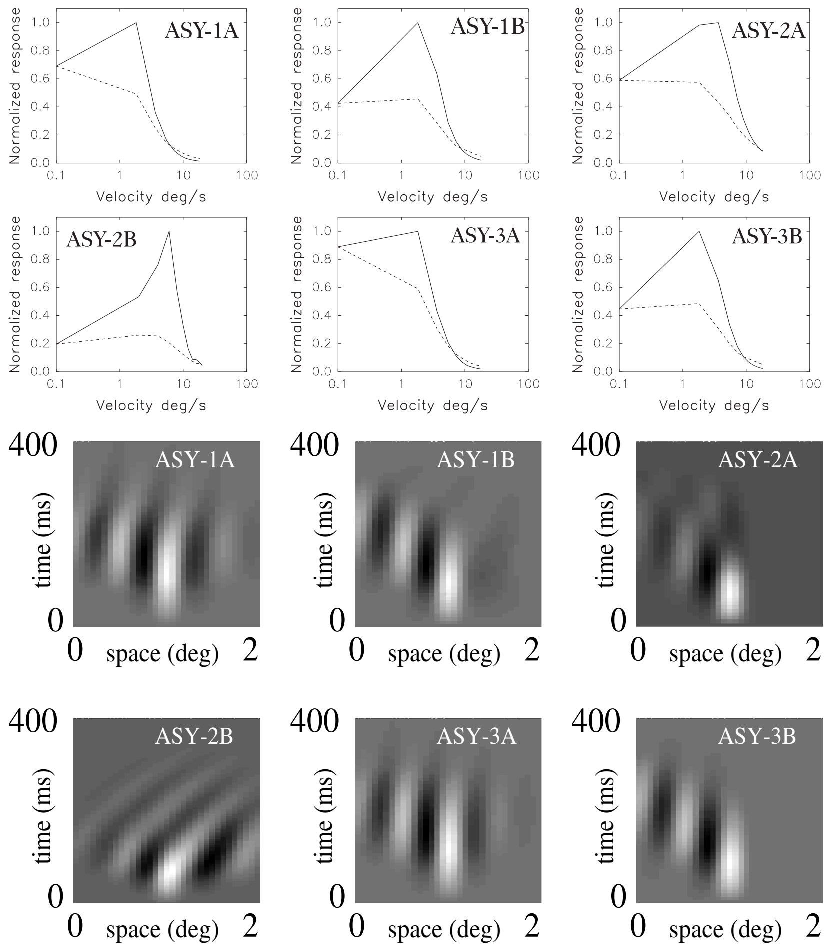

Figure 1: Results from the model related to each asymmetry considered. (Top) velocity tuning curve; (Bottom) spatio-temporal plots. We can evidence a marked velocity tuning for ASY-2B, i.e., when inhibition arises from two differentially offset Gaussians