Functional Periodic Intracortical Couplings Induced by Structured Lateral Inhibition in a Linear Cortical Network

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The spatial organization of cortical axon and dendritic fields could be an interesting structural paradigm to obtain a functional specificity without postulating highly specific feedforward connections. In this article, we investigate the functional implications of recurrent intracortical inhibition when it occurs through clustered medium-range interconnection schemes (Wörgötter & Koch, 1991; Somogyi, 1989; Kritzer, Cowey, & Somogyi, 1992). Moreover, the interaction between the inhibitory schemes and visual orientation maps is explored. Assuming linearity, we show that clustered inhibitory mechanisms can trigger a propagation process that allows the development of extra (i.e., induced) interactions among the cortical sites involved in the recurrent loops. In addition, we point out how these interactions functionally modify the response of cortical simple cells and yield to highly structured Gabor-like receptive fields. This study should be considered not as a realistic biological model of the primary visual cortex but as an attempt to explain possible computational principles related to intracortical connectivity and to the underlying single-cell properties.

1 The Model

Assuming an “averaged model” of the visual cortex (Amari, 1977; Krone, Mallot, Palm, & Schüz, 1986; Chernjavsky & Moody, 1990), the cortical surface \( x = (x_1, x_2) \) can be viewed as a two-dimensional homogeneous distribution of cortical elements (nodes) characterized by specific orientation selectivities \( \theta(x) \). In this context, a node represents a neural population, and interconnections represent functionally the overlap of axonal and dendritic clouds. Under linear steady-state conditions, the distribution of cortical ex-
cition $e(x)$ can be modeled as:

$$e(x) = e_0(x) - b \int k_{fb}(x; \xi)e(\xi)d\xi$$

$$= e_0(x) - b \int k_{ff}(x; \xi; b)e_0(\xi)d\xi,$$

(1.1)

where $e_0(x)$ is the feedforward distribution of excitation, $b$ is the strength of the inhibition ($b > 0$), and $k_{fb}(x; \xi)$ is the intracortical coupling function. The feedforward resolvent kernel of the integral equation $k_{ff}(x; \xi; b)$ (Winter, 1990) represents an interaction among cortical sites, telling how total afferent drive at one site affects activity at a second site. Equation 1.1 can thus be interpreted as a two-dimensional iterative filter that detects specific characteristics present in the input pattern of excitation $e_0(x)$; the spatial extension on which these characteristics are detected is possibly larger than that of $k_{fb}(x; \cdot)$. This occurs both directly, by physical local interactions, and indirectly, through the propagation property of iterative filters, which asserts that the output values of the filter can be recurrently affected by a larger neighbor region on the cortical plane. In this way, one can speak of "induced" functional couplings not directly related to the presence of the corresponding specific wirings.

In the case of a translation-invariant kernel ($k_{fb}(x; \xi) = k_{fb}(x - \xi)$), the exact solution of equation 1.1 can be straightforwardly obtained by Fourier transform techniques, and the resulting cortical activity patterns tend to have spatial frequency corresponding to the peak of the function $(1 + bK_{fb}(f))^{-1}$, where uppercase letters refer to Fourier transforms and $f = (f_1, f_2)$ is the spatial frequency vector. In particular, when inhibition arises from clusters of cells spatially distributed at a distance $\pm d$ from the target cell (see Fig. 1a), the spectrum of the feedforward resolvent kernel, $K_{ff} = K_{fb}(1 + bK_{fb})^{-1}$, shrinks, and the energy peak shifts approximately at $f^* = (\pm 1/2d, 0)$ (see Fig. 1b). This result can be motivated by the following expansion:

$$[1 + bK_{fb}(f)]^{-1} = 1 - bK_{fb}(f) + b^2K_{fb}^2(f) - b^3K_{fb}^3(f) + \cdots$$

$$= 1 - bK_{ff}(f).$$

(1.2)

Since $K_{fb}$ is a low-pass filter, its powers in equation 1.2 lead to a decrease in bandwidth and so to an increasing width of the resolvent coupling function. Specifically, if we consider $k_{fb}(x - \xi)$ as the sum of two laterally offset gaussians ($g(x_1 \pm d, x_2; \sigma^2)$), the powers of its spectrum result, in the space domain, in a sum of shifted gaussians with alternating signs, which contribute to the coupling with decreasing strength:

$$k_{ff}(x - \xi) = \sum_{l=1}^{\infty} (-1)^{l-1}k_l(x - \xi)$$

(1.3)
Figure 1: (a) Schematic representation of the recurrent inhibitory kernel $(k_{fb})$, with $\theta = \pi/2$, $\phi_0 = \theta + \pi/2$, $\Delta\varphi = \pi/8$. The thick vertical line in the center represents the orientation of the target cell, which receives inhibition from the cells in the shaded area. The lateral spread of interconnections can be characterized by the angular bandwidth $\alpha$ related to the half-peak magnitude extension of the kernel. (b) The normalized cross-sections, along the direction $\phi_0$, of $|K_{fb}(f)|$ (dashed line) and of $|K_{ff}(f)|$ for $b = 0.5$ (thick continuous line). Increasing the value of $b$ (see the insets), one can observe a major redistribution of the spectrum energy around the frequency $f_1 = \pm 1/2d$. 
where

\[ k_l(x_1 - \xi_1, x_2 - \xi_2) = \sum_{m=0}^{l-1} \left( \frac{l}{m} \right) T^l g \left( x_1 - \xi_1 - (2m - l)d, x_2 - \xi_2; l \sigma_k^2 \right) . \] (1.4)

This expression well evidences the regular distribution of the additionally induced couplings and their potential impact on the spatial structure of simple cell-receptive fields. It is worth noting that the geometrical organization of inhibitory couplings has a key role: whereas recurrent inhibitory couplings among clustered populations of neurons induce a regular spatial alternation of excitatory and inhibitory interactions, this behavior does not show up when inhibitory connections are uniformly distributed on the cortical plane.

The functional effects of clustered recurrent inhibition are even more interesting when the structure of the interconnection schemes depends on the orientation selectivity of the target cell. In the following, we focus on inhibitory interactions occurring along the axis orthogonal to the receptive field orientation (Gilbert & Wiesel, 1989; Kisvárday et al., 1994) (see Figs. 2a–d). Accordingly, we consider \( k_{fb} \) as:

\[ k_{fb}(x; \xi) = g_{\psi_0}(x; \xi) + g_{\psi_0 + \Delta \phi}(x; \xi) + g_{\psi_0 - \Delta \phi}(x; \xi) , \] (1.5)

where \( g_{\phi} \) represents a couple of translated \((\pm d)\) two-dimensional gaussian functions (with spatial spread \( \sigma_k \)), rotated through an angle \( \psi(x) \) about the location of the target cell. Specifically, we choose \( \psi_0(x) = \theta(x) + \pi/2 \) and \( \Delta \phi \) in the range \([0, \sigma_k (2 \ln 2)^{1/2}/d]\), the upper limit giving a maximally flat function \( k_{fb} \).

Figures 2e–h show different resultant interaction fields for various orientation maps. Observe that smooth periodic variations in the orientation preferences are decisive to sustain the propagation mechanism and thereby to prime the periodicity of the induced couplings (see Fig. 2e). Random arrangements of orientations, in contrast, as well as high gradients in the orientation map where inhibition is present, result in a loss of coherence and hence in the attenuation or even the suppression of the contributions of recurrence (see Figs. 2f–h). For all these maps, we observe that although the recurrent kernel \( k_{fb} \) has a uniform positive sign, the resultant interaction fields \( k_{ff}'s \) show areas with couplings that are negative in sign. The characteristics of these resultant interaction fields (i.e., the size, the shape, and the sign of interaction) have a complex dependence on the pattern of recurrent connections.

2 Effects on the Cortical Receptive Field Profiles

Since the striate cortex is visuotopically organized, one might expect a relationship between the morphology of lateral connections and the receptive...
Figure 2: The white blobs in the top quartet figure show the feedback inhibitory kernels \( k_{fb} \) referred to the central cell of four different distributions of orientation selective cells. The orientation fields in (a–c) correspond to portions of a single orientation map (derived after Niebur and Wörgötter’s model in Niebur & Wörgötter, 1994) and represent different typical situations, such as an iso-orientation domain (a), a pinwheel discontinuity (b), and a fracture discontinuity (c). The map in (d) represents a random arrangement of orientations. The middle quartet figure (e–h) shows the contour plots of the resulting feedforward kernels after recursion still referred to the central cell for the corresponding orientation fields. The plots point out the topology of the inhibitory-excitatory effects of the total afferent drive, on the activity of the central cell: gray lines represent positive values of \( k_{ff} \) (i.e., inhibitory contributions), whereas black lines represent negative values of \( k_{ff} \) (i.e., excitatory contributions). The bottom quartet (i–l) shows the corresponding receptive fields, \( h(\cdot, x') \), obtained by a numerical evaluation of equation 2.3. The parameters used in all the simulations are \( \sigma_t = 0.0625 \), \( \Delta \varphi = \pi/12 \), \( d = 0.25 \), \( \sigma_n = 0.1 \), and \( p = 3 \). The inhibition strength \( b \) is 0.5.

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field topography (Mallot, von Seelen, & Giannakopoulos, 1990; Schwarz & Bolz, 1991). To model the effect of recurrent inhibition on the linear simple cell receptive fields, we assume the superposition of contributions from the lateral geniculate nucleus (LGN) and intracortical inhibitory interactions. Specifically, functional projections of LGN afferent neurons provide a weak bias in orientation to cortical cells, and the final profiles of the receptive fields are molded by the inhibitory processes inside the cortex. From a computational point of view, assuming the linearity of simple cells (De Angelis, Ohzawa, & Freeman, 1993), the initial excitation $e_0(x)$ is given by

$$e_0(x) = \int h_0(x; x') i(x') dx',$$

where $i(x)$ is the input signal and $h_0(x; x')$ is the initial receptive field profile due only to thalamic feedforward connections. The initial receptive field profile is modeled as an oriented gaussian function:

$$h_0(x; x') = \frac{1}{2\pi \sigma^2} \exp \left[ -\frac{x_1^2}{2\sigma_1^2} - \frac{x_2^2}{2\sigma_2^2} \right],$$

where $p$ refers to the aspect ratio of the field, and $\sigma_h$ specifies the receptive field size. Taking into account intracortical contributions, the resulting excitation can be expressed as

$$e(x) = \int h(x; x') i(x') dx'. $$

From equation 2.1, we can derive the expression for the resulting receptive field after inhibition:

$$h(x; x') = h_0(x; x') - b \int k_{ff}(x; \xi; b) h_0(\xi; x') d\xi. $$

By a numerical evaluation of equation 2.3, we obtained the resultant receptive field profiles for the four orientation fields considered (see Figs. 2i–l). We note that horizontal inhibition may well be the substrate for a variety of influences observed between the receptive field center and its surround, including the refining of the orientation and spatial frequency tuning of simple cells, and the emergence of the Gabor-like receptive field profiles (Sabatini, 1996). Equation 2.3 indicates how the receptive fields, interpreted as retinotopic distributions of the activity on the cortical plane, reflect the whole spatial pattern of couplings and not only the direct neuroanatomical connectivity (von Seelen, Mallot, & Giannakopoulos, 1987). Therefore, asymmetry and anisotropy of lateral intracortical couplings manifest themselves in their visuotopic representations, influencing the receptive fields of the cells exposed to the interconnection patterns (Martin & Whitteridge, 1984; Gilbert & Wiesel, 1989).

**References**


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