Mechanisms for Surround Suppression in a Spiking Neuron Model of Macaque Striate Cortex (V1)

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***Abstract*— Surround suppression is a nonlinear extraclassical receptive field response phenomena prevalent in cells in striate cortex (V1). In this paper we present simulation results, from a spiking neuron model of a V1 input layer, which demonstrate two distinct mechanisms for surround suppression. The model consists of a layer of excitatory and inhibitory integrate and fire neurons, representing a small patch of a cortical input layer. The construction of the model is carefully constrained to be agreement with anatomical findings. Ocular dominance and a slight orientation preference is set by specificity in the connections of the cortical simple cells to the LGN axons entering the layer. The LGN neurons themselves have circular symmetric center-surround ON/OFF type receptive fields which are laid out on a 2D lattice of appropriate density in visual space. The length scale of cortical excitation is twice that of inhibition. The cortical connectivity in the model is isotropic, with no orientation or geometric preference (i.e. no long-range horizontal connections). Simulations using stimuli consisting of drifting sinusoidal grating in circular apertures of varying radii demonstrate that surround suppression arises in the model solely from cortical interactions, either directly from cortical inhibition or indirectly from suppression of the recurrent cortical excitation by cortical inhibition. These simulation results suggest that surround suppression is entirely the result of cortical interactions and not a result of feedforward interactions. Further, they suggest that surround suppression is possible with only local cortical interactions and may not require the long-range cortical connections within V1, nor feedback projections from extrastriate areas, as has been proposed.**

***Keywords*— monkey V1, visual cortex, surround suppression, receptive field, texture modulation, spatial integration**

I. Summary

In mammals, the very first stage of cortical visual processing takes place in the striate cortex (area V1). Surround suppression, a non-linear response to drifting gratings having varying radii within a circular aperture, is known to be a prevalent feature of cells throughout V1, including all type of cells in all layers and at all eccentricities [1], [2], [3]. A definitive explanation of the neural mechanisms responsible for surround suppression still remains elusive. Understanding the mechanisms underlying the phenomena is potentially important for developing a theoretical model of early signal integration and neural encoding of visual features in V1.

Thus far, analysis of the experimental findings for surround suppression have led to several alternative hypotheses to its origin. For example, it has been proposed that 1) it arises simply from feedforward LGN input [4], 2) is due to long-range horizontal connections [5], [6], [3], 3) is related to feedback from extrastriate areas [2], [3]. In this paper we identify two possible mechanisms for the phenomena within a computational model we have developed of multiple orientation hypercolumns of V1. Both mechanisms require only local, isotropic cortical connections. In the following we describe the simulation results as well as provide a very brief description of the model. [1]

The model consists of a layer of N conductance based integrate-and-fire point neurons (one compartment), dynamic variables of each neuron are it's membrane potential $V_i(t)$ and it's spike train $\mathcal{S}_i(t) = \sum_k \delta(t - t_{i,k})$, where t is time and $t_{i,k}$ is the kth spike of the ith neuron. Whenever the membrane potential reaches a fixed threshold level, it is reset and a spike is registered. Each membrane potential obeys an equation of the form

$$\begin{aligned} C_i \frac{dV_i(t)}{dt} &= -\lambda_i(V_i(t) - V_L) \\ &\quad -g_{E,i}(t, [\mathcal{S}], \eta_E)(V_i(t) - V_E) \\ &\quad -g_{I,i}(t, [\mathcal{S}], \eta_I)(V_i(t) - V_I) + I_i^{ext}(t) \\ &\quad i = 1, \ldots, N \, . \end{aligned}$$

Where

$$g_{E,i}(t, [\mathcal{S}], \eta_E) = g_i^{LGN}(t) + \eta_{E,i}(t) + g_{E,i}^{cor}(t, [\mathcal{S}]) \, ,$$
$$g_{I,i}(t, [\mathcal{S}], \eta_I) = \eta_{I,i}(t) + g_{I,i}^{cor}(t, [\mathcal{S}]) \, ,$$

Here, C_i is the membrane capacitance, V_L, V_E and V_I are the reversal potentials of leakage, excitation and inhibition respectively, λ_i, $g_{E,i}$ and $g_{I,i}$ are the leakage, excitatory and inhibitory conductances impinging on neuron i. The quantities η_E and η_I are external stochastic terms, $I_i^{ext}(t)$ is an external current and $[\mathcal{S}]$ is shorthand notation for $\mathcal{S}_1(t), \mathcal{S}_2(t), \ldots, \mathcal{S}_N(t)$ to indicate that neuron i is coupled in principle to all neurons in the system. The terms $g_{\cdot,i}^{cor}(t, [\mathcal{S}])$ are the contributions from the cortical excitatory and inhibitory neurons and include only isotropic connections,

$$\begin{aligned} g_{\cdot,i}^{cor}(t, [\mathcal{S}]) &= \int_{-\infty}^{+\infty} ds \sum_{j \in \mathcal{P}(\cdot)} \\ &\quad \mathcal{C}_{i,j}(||\vec{x}_i - \vec{x}_j||) G_{\cdot,j}(t - s) \mathcal{S}_j(s) \, . \end{aligned}$$

LGN axons are taken to be purely excitatory, their contribution enters only into the excitatory conductances of simple cells while the LGN neurons themselves are modeled as rectified center-surround linear spatiotemporal filters, a cortical simple cell, j, being connected to $N_0(j)$ of them,

$$g_j^{LGN}(t) = \sum_{i=1}^{N_0(j)} \Big\{ g_j^0$$

[1] Details of the model to be published elsewhere with some additional background on the model found in [7], [8].

$$+\int_0^t ds \int d^2x G^{LGN}(t-s)A(\vec{x}_j^{\,i}-\vec{x})I(\vec{x},s)\Big\}^+,$$

where $I(\vec{x},s)$ is the stimulus and $\vec{x}_j^{\,i}$ is the receptive field center of the ith LGN cell connected to the jth cortical (simple) cell.

Ocular dominance and a slight orientation preference are imposed in the layer by the LGN input, the anatomy and physiology of which is realistically modeled. LGN receptive field sizes, density of LGN neurons, their axon sizes in V1, retinotopic map and cortical magnification factor are all set to agree with experimentally established data. Similarly for the cortical parameters, such as neuron density, fraction of inhibitory and excitatory cells, fraction of simple and complex cells, axon and dentritic sizes. Finally, synaptic time scales and the 1st order temporal LGN kernel are all set to agree with experimental observations for the macaque monkey.

In figure 1, top panel, we show the extracellular responses (F1 component) of two simple cells displaying surround suppression in response to a monocular 8 Hz drifting grating stimulus in an expanding circular aperture. The aperture radius is plotted horizontally. The grating angle, drift direction, spatial and temporal frequency all set equal to the optimal values for these neurons. Parameters in the model were set to correspond to the input layer $4C\beta$ of macaque at $0-10$ degrees eccentricity. Our model layer represents approximately $16mm^2$ of cortical surface area and contains a little over 16000 neurons.

As seen in figure 1, surround suppression in neuron 1 (black curve) is caused by direct cortical inhibition on the cell, whereas the suppression in neuron 2 (orange curve) is caused by a reduction of the recurrent cortical excitation on the cell. This is made clear in the remaining panels. The middle two panels correspond to neuron 1, the lower two to neuron 2. Each contains 8 subpanels which show cycle averaged quantities for different aperture sizes (aperture size of maximum response is marked by a star). Plotted are the relevant cycle averaged quantities: the total current for voltage clamp at threshold (v=1) (red), the cortical inhibitory conductance (blue), the LGN conductance (black) and the cortical excitatory conductance (green). When we compare the averaged quantities for the optimal aperture (star) with those immediately right of it we see, for neuron 1 (middle two panels): inhibition increases monotonically while cortical excitation also increases monotonically and the LGN excitation remains roughly constant. This implies that the decreased response of the neuron, also illustrated by the suppression of the membrane current at threshold (red), must be entirely due the increase of direct cortical inhibition. In contrast, results for neuron 2 (lower two panels) show that cortical recurrent excitation on the neuron decreases while inhibition and LGN excitation remain constant. This implies that suppression is entirely due to a reduction of the recurrent cortical excitation this neuron experiences.

We conclude that (i) the LGN input does not contribute to the suppression in either of the two mechanisms. This holds true in general: the surround suppression, of course present in any single LGN cell because of their center-surround receptive field, does not directly contribute to the extraclassical surround suppression found in the cortical neurons in our model. The effect is "washed out" by the fact that a small group of LGN neurons feed into a cortical simple cell. (ii) Both mechanisms are a result of local isotropic cortical connections. This suggest that surround suppression is possible with only local cortical interactions and may not require the long-range cortical connections within V1, nor the feedback projections from extrastriate areas.

Lastly, we observe a great diversity of responses in our model. Both mechanisms occur also in complex cells. Further, we also observe cells which display both mechanism in both possible orders of action. Further details will be published elsewhere.

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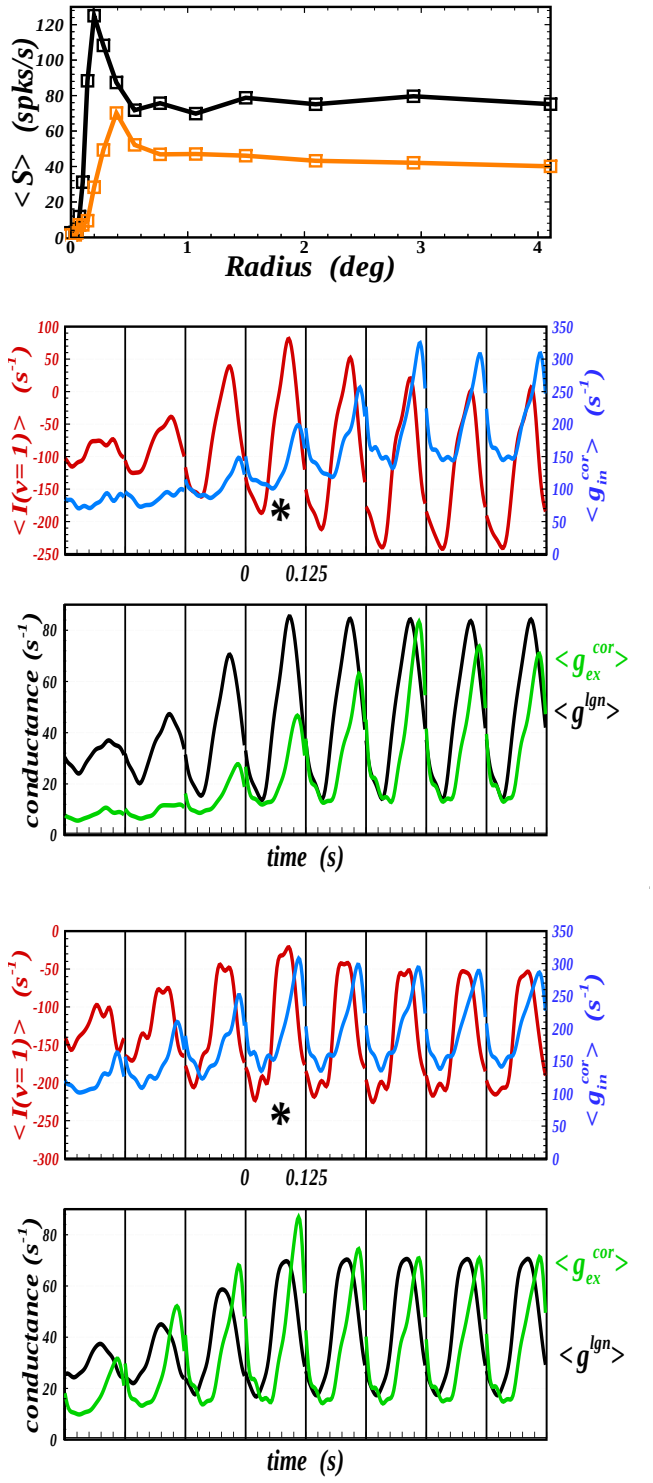

Fig. 1. Two different mechanisms of surround suppression. Top panel: extracellular response (F1 component) of two simple cells as function of the aperture radius. After the response reaches a maximum it is suppressed by direct cortical inhibition in case of neuron 1 (black), or by a reduction in the cortical excitation it receives in case of neuron 2 (orange). The two mechanisms are clarified in the two middle (neuron 1) and two lower panels (neuron 2), each of which consist of 8 subpanels which show cycle averaged quantities (width of 1 subpanel is 1 cycle or 0.125 s) that are obtained for the eight nearest aperture sizes around the aperture size for which the response is maximum, the subpanel belonging to this aperture size is marked by a star. Plotted are the relevant cycle averaged quantities: the total current for voltage clamp at threshold (v=1) (red), the cortical inhibitory conductance (blue), the LGN conductance (black) and the cortical excitatory conductance (green).