The contributions of inhibition and noise to responses in V1.

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Abstract

We include a recently described class of inhibitory cells, complex cells untuned for orientation, as well as simple cells in a model of V1 to study their contribution in shaping simple cell responses. Untuned complex cell inhibition can suffice to explain contrast-invariant orientation-tuning and low-pass temporal frequency tuning of cortical simple cells. Given this complex cell inhibition, antiphase ("push-pull") inhibition from tuned simple inhibitory neurons acts to sharpen spatial frequency tuning and increase the stability of cortical activity. Intracortical inhibition is needed to achieve contrast-invariance of the voltage tuning, which is converted by physiological noise levels into contrast-invariant spike tuning.

Summary

Cells in primary visual cortex (V1) of cat are tuned for the orientation of light/dark borders (Hubel and Wiesel 1962). Understanding the circuity underlying this orientation selectivity remains a central problem in systems neuroscience (reviewed in Ferster and Miller 2000), and serves as a model system for understanding cortical processing. Hubel and Wiesel (1962) proposed that the orientation selectivity of simple cells is shaped by an oriented arrangement of input from cells in the lateral geniculate nucleus (LGN) that are not tuned for orientation: ON-center LGN cells project to the ON-subfields of layer 4 simple cells, and OFF-center LGN cells project to their OFF subfields. Such an input pattern has been verified experimentally (Alonso et al. 2001; Reid and Alonso 1995).

However, the pattern of feedforward excitation alone is not sufficient to explain simple cell response properties. The orientation tuning bandwidth remains roughly constant across contrasts, a phenomenon known as contrast-invariant orientation tuning (Skottun et al. 1987). We have previously shown that a simple correlation-based model circuit of simple cells in cat V1 layer 4, in which inhibitory simple cells provide "push-pull" or antiphase inhibition, can account for the contrast invariance of orientation tuning, provided that the inhibition dominates the excitation (Troyer et al. 1998). A prediction was that layer 4 inhibitory simple cells, though tuned for orientation, would have a contrast-dependent response to non-preferred orientations. Recently, Hirsch et al. reported that inhibitory neurons in cat V1 layer 4 show two types of receptive fields (RFs): simple RFs tuned for orientation, without responses to non-preferred orientations; and complex RFs with mixed ON/OFF responses, lacking in orientation tuning (Hirsch et al. 2000). This suggests that complex inhibitory neurons may provide the broadly tuned inhibition we had attributed to simple cells.

We have explored this hypothesis by including both types of inhibitory cells in our layer 4 model (fig 1.). Complex cell inhibition can account for contrast-invariant orientation tuning of both excitatory and inhibitory simple cells. However, this is not as robust as antiphase inhibition – somewhat more careful tuning of parameters is required. Given such complex cell inhibition, antiphase inhibition from simple cells has only weak additional effects on

excitatory cell responses, in particular suppressing responses at lower spatial frequencies.

Recent results from the Ferster group reveal that the combination of voltage noise and contrast-invariant tuning of the voltage yields contrast-invariant spike responses. We have investigated how the tuning of the membrane potential and spike rate arises in the presence of varying rates of antiphase (push-pull) inhibition and physiological noise in a single cell model (Palmer and Miller 2003). We found that balanced or dominant feedforward intracortical inhibition is necessary for voltage tuning to become contrast-invariant, and that as expected voltage noise converts this voltage tuning into contrast-invariant spiking tuning.

Here we include the *in vivo* noise levels in our network model. We verify the results from the single-cell studies, and show further that the resulting contrast-invariance of orientation tuning is stable even for large correlations in the membrane potential fluctuations between individual cells.

Details of the model

We study a model shown in cartoon form in fig. 1, previously described (Lauritzen and Miller 2003). Here we present the basics of the model along with present additions.

The input to our model comes from 7200 LGN X-cells arranged in four overlying 30×30 sheets of ON cells and four similar sheets of OFF cells, covering $6.8 \times 6.8^\circ$ of the visual field.

The model contains three cortical cell types found in cat V1 layer 4: 1600 orientation-tuned excitatory and 225 inhibitory simple cells with separate ON and OFF subfields, and 225 inhibitory complex cells untuned for orientation with ON-OFF receptive fields (RFs) (Hirsch et al. 2000). All cells receive thalamocortical input selected probabilistically, the simple cell inputs chosen from ON-center inputs overlying ON-subregions and OFF-center inputs overlying OFF subregions (Alonso et al. 2001; Hubel and Wiesel 1962; Reid and Alonso 1995), and the complex cell inputs chosen from all LGN cells whose RFs overlap. Cortical connectivity is also determined probabilistically, with the most probable connections illustrated in the cartoon in fig. 1. The simple cells form synapses primarily to other cells with similar preferred orientation, according to the correlation of their RFs: Excitatory

synapses are formed to cells with correlated RFs, and inhibitory synapses are formed to cells with anti-correlated RFs. The complex cells project randomly with equal (10%) probability to all simple cells within 150 μm, and, for simplicity, do not receive any cortical input. The full model contains simple cells preferring all orientations and spatial phases.

The noise, mimicking membrane potential fluctuations observed *in vivo*, is created by adding fluctuating conductances to the cells (Palmer and Miller 2003). The noise conductances are made up of a part constant in time, g_x^{bkgnd}, with $x = E$ or I for excitatory or inhibitory conductance and a part fluctuating in time, η_x, governed by the Ornstein-Uhlenbeck process (Uhlenbeck and Ornstein 1930):

$$d\eta(t) = -\kappa\eta(t)dt + \sqrt{D}dW(t),$$

where D is the diffusion constant, $dW(t)$ is Gaussian white noise, and the term $-\kappa\eta(t)$ represents decay with a characteristic time scale $1/\kappa$. This gives an update rule (Destexhe et al. 2001; Gillespie 1996):

$$\eta(t+\delta) = [e^{-\kappa\delta t}\eta(t) + \sqrt{\frac{D}{2\kappa}(1 - e^{2\delta t\kappa})}N(0,1)]^+,$$

where $N(0,1)$ is a Gaussian white noise process with mean zero and unit standard deviation, and $[...]^+$ denotes that we rectify the conductances at zero. We define two diffusion terms, as well as two background conductances, one excitatory and one inhibitory. Noise parameters are chosen to achieve voltage fluctuations as measured intracellularly by David Ferster's group, with a standard deviation around 5 mV. The noise input to inhibitory cells is scaled accordingly to get the same noise profile for inhibitory simple cells as for the excitatory cells.

Since the membrane potential fluctuations in nearby cells are correlated (e.g. Lampl et al. 1999) we divide the noise conductance into a part common to nearby cells and a part specific to each cell.

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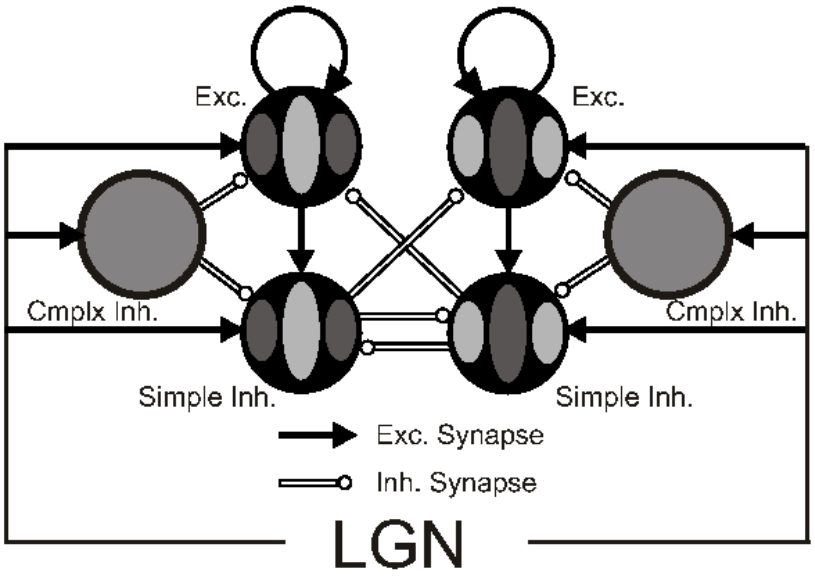

Figure 1:

Cartoon of model circuit. Top, two excitatory simple cell receptive fields (RF's); bottom two inhibitory simple cells RF's, with light gray ON subregions and dark gray OFF subregions. Both cell types are tuned for orientation. Middle to the sides, ON-OFF complex cell RF's in medium gray, untuned for orientation. All RF's in cartoon are centered at the same retinotopic point. Complex cells inhibit all types of simple cells; simple cell connectivity is correlation-based (push-pull), so that excitatory simple cells tend to connect to other simple cells with whom they are well correlated, and inhibitory simple cells tend to connect to other simple cells with whom they are strongly anticorrelated. The actual model contains cells of all preferred orientations and spatial phases and multiple retinotopic positions. Connections are assigned probabilistically with the most probable connections as shown in the cartoon.