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.

1 Introduction

Cells in primary visual cortex (V1) of cat are tuned for the orientation of light/dark borders (5). Understanding the circuity underlying this orientation selectivity remains a central problem in systems neuroscience (reviewed in 3), and serves as a model system for understanding cortical processing.

The orientation tuning bandwidth of simple cells remains roughly constant across contrasts, a phenomenon known as contrast-invariant orientation tuning (10). 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 (11). 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 (4). 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 (9). 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.

2 The model

We study a model shown in cartoon form in fig. 1, previously described (8). 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 × 6.8° of the visual field. The model contains three cortical cell types found in cat V1 layer 4: 1600 excitatory and 225 inhibitory orientation-tuned simple cells with separate ON and OFF subfields, and 225 inhibitory complex cells untuned for orientation with ON-OFF receptive fields (RFs) (4). All cells receive thalamocortical input selected probabilistically, according to Hubel and Wiesel (5) for simple cells, and for complex cells chosen from all LGN cells whose RFs overlap with their RF. 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.

Place Fig. 1 about here

The noise, mimicking membrane potential fluctuations observed *in vivo*, is created by adding fluctuating conductances to the cells (9). The noise conductances are made up of a part constant in time, g_x^{bkgnd}, and a part fluctuating in time, η_x, governed by the Ornstein-Uhlenbeck process (12):

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

where D is the diffusion constant, $dW(t)$ is Gaussian white noise, and the term $-\kappa\eta_x(t)$ represents decay with a characteristic time scale $1/\kappa$. $x = E$ or I indicate excitatory or inhibitory conductance respectively. This gives an update rule (2):

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

where $N(0,1)$ is a Gaussian white noise process with mean zero and unit standard deviation. 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 4 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. 7) we divide the noise conductance into a part common to nearby cells and a part specific to each cell.

3 Results

In simulations using drifting grating stimuli, we can block the inhibition coming from one of the two inhibitory cell types. If we block the complex cell inhibition, leaving only simple-cell inhibition, the model correspond to the original model (11). Here the excitatory simple cells display contrast-invariant orientation tuning, while the inhibitory simple cells have a response component tuned for orientation atop a component untuned for orientation (not shown). If we instead block the simple cell inhibition, leaving only complex-cell inhibition, both types of simple cells will receive inhibition untuned for orientation and will display contrast-invariant orientation tuning (fig. 2a). If both types of inhibition are present, the extra inhibition from the simple inhibitory cells will arrive out of phase with the excitation received by a given cell and so will not affect of the orientation tuning.

Place Fig. 2 about here

Thus given complex-cell inhibition, simple-cell inhibition does not affect orientation tuning. What effects does simple cell inhibition have? To examine this, we examined the effect of the presence or absence of simple-cell inhibition on other V1 response properties. At the preferred orientation and spatial frequency, the antiphase simple-cell inhibition is significant only during temporal phases in which complex-cell inhibition already dominates excitation, and so it has little effect. However, if the spatial frequency of the stimulating drifting grating does not match the spatial frequency preference of the cells (roughly given by the spatial frequency component of the Gabor filter, 0.8 cycles/degree), then simple-cell inhibition can impact responses. It acts to sharpen the spatial frequency tuning of the cells in the network, primarily by suppressing responses to lower spatial frequencies (fig. 2b).

We have previously shown that the model with only simple-cell inhibition can explain the fact that cortical neurons have temporal frequency high-cutoffs at much lower frequencies than LGN cells (6). The explanation relied on two elements: NMDA-receptor-mediated conductances (which we will abbreviate simply as "NMDA") in synapses from LGN to excitatory cells reduced the temporal modulation of the excitatory input at higher frequencies, largely reducing this input to its mean; and the dominant inhibition prevented responses to the mean excitation. Note that this explanation requires that some inhibitory neurons respond to the high temporal frequencies that drive LGN cells. This explanation of temporal frequency tuning also works with complex-cell inhibition, and in this case is even more powerful: because complex-cell inhibition is not modulated at the opposite phase

as the excitation, less demodulation of the excitation is required for the inhibition to dominate the excitation, and so temporal frequency high-cutoffs occur at lower frequencies than in the previous model. The complex cells do not receive inhibition and so respond to higher temporal frequencies that drive LGN cells, while both excitatory and inhibitory simple cells receiving the complex cell inhibition cut off at lower temporal frequencies (fig. 2c).

Anderson *et al.*(1) showed that the individual voltage responses to moving grating stimuli are much more variable than their averaged responses and suggested that, given contrast-invariant orientation tuning of the membrane potential of a cell, this variability can account for the contrast-invariance of the spike tuning. In the full model with physiological levels of noise we find that this is the case, but intracortical inhibition is needed to provide the required contrast-invariance of the voltage tuning (not shown). Furthermore when increasing the correlation of the noisy conductances in the cells in the network from 0% to 100%, both the mean and the fluctuations of the membrane potential decrease (fig. 3), but contrast-invariant voltage and spike tuning persists as long as the fluctuations are not too small (not shown).

Place Fig. 3 about here

4 Discussion

We have shown that feedforward inhibition from complex inhibitory cells that are untuned for orientation, as observed by Hirsch *et al.*(4) in cat V1 layer 4, can combine with feedforward excitation from LGN inputs to explain the sharp, contrast-invariant orientation tuning of layer 4 simple cells. Furthermore, assuming that these inhibitory cells follow the LGN inputs in their temporal frequency tuning, and given NMDA-receptor-mediated conductances in LGN synapses, this can also explain the low-pass temporal frequency tuning of simple cells. Given complex cell inhibition, antiphase simple-cell inhibition acts to somewhat sharpen the spatial-frequency tuning of simple cells, as well as to stabilize the network activity (8). Thus, we propose that the two different inhibitory cell types each have their separate roles in shaping the responses of cortical cells. Physiological noise fluctuations can explain contrast-invariance of the spiking orientation tuning given contrast-invariant orientation tuning of the membrane potential. Correlation between the cells decrease the mean and the fluctuations of membrane potential of the cells.

5 Biosketch

Thomas Lauritzen received his B.Sc. degree in physics in 1996, and his M.Sc. in physics-biophysics in 1998 from the Niels Bohr Institute, Copenhagen, Denmark. He is currently enrolled on the Ph.D. program in biophysics at the University of California, San Francisco.

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Figure legends

Figure 1

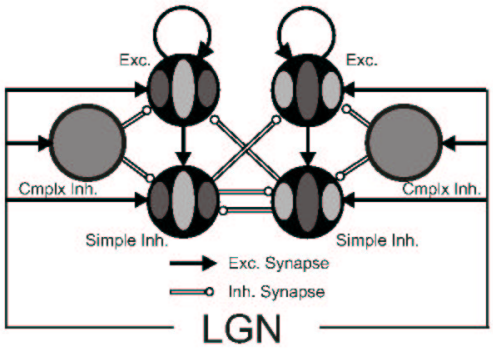
Cartoon of model circuit. Top, two excitatory simple cell receptive fields (RF's); bottom two inhibitory simple cells RF's, with light gray ON subregions and dark gray OFF subregions. Both cell types are tuned for orientation. Middle to the sides, ON-OFF complex cell RF's in medium gray, untuned for orientation. All RF's in cartoon are centered at the same retinotopic point.

Figure 2

Response tuning curves of the network. A: Orientation tuning curves of excitatory and inhibitory simple cells. Given complex cell inhibition, both cell types display contrast-invarience of their orientation tuning. B: Spatial frequency tuning of the excitatory simple cells. The spatial frequency bandwidth given complex cell inhibition (black line) gets sharpened by including simple cell inhibition (gray line). Arrow indicate the tuning of the Gabor filter of the cells. C: Temporal frequency tuning curves of the excitatory simple cells. The temporal frequency tuning show a cut-off around 2 Hz both for complex cell inhibition only (black line) and for simple and complex cell inhibition (gray line).

Figure 3

Noise fluctuations of a model cell at 0% contrast. A and B: Count of fluctuations around the mean potential of a single excitatory cell for 60s of simulated time with 0% correlation (A) and 100% correlation (B) of the noise conductances in the cells in the network. The fluctuations, especially the tail towards the threshold, get smaller with increased correlation. C: Quantification of changes in the membrane potential with increased noise correlation. Both the mean of the membrane potential and the fluctuations (root mean square) decrease with the noise correlation.



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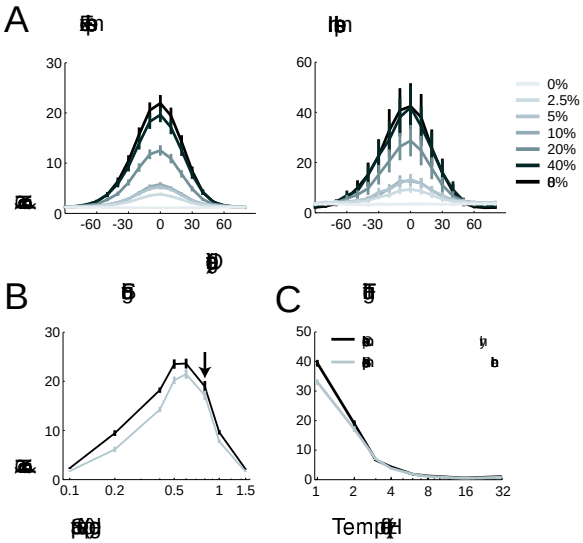

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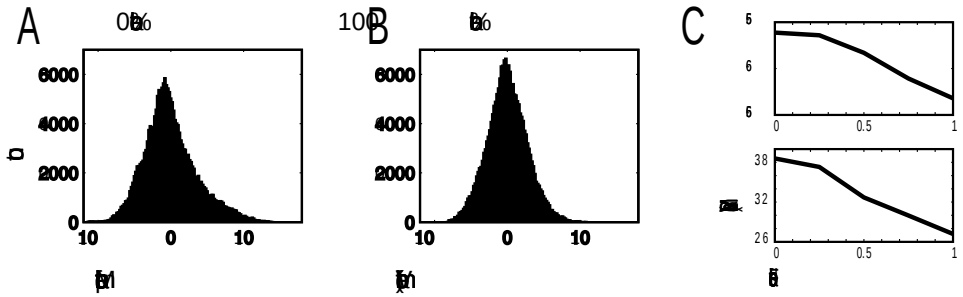
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Figure 3: