Dependence of Response Properties on Sparse Connectivity in a Spiking Neuron Model of the Lateral Geniculate Nucleus

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Wielaaard J, Sajda P. Dependence of response properties on sparse connectivity in a spiking neuron model of the lateral geniculate nucleus. J Neurophysiol 98: 3292–3308, 2007. First published October 3, 2007; doi:10.1152/jn.00654.2007. We present a large-scale anatomically constrained spiking neuron model of the lateral geniculate nucleus (LGN), which operates solely with retinal input, relay cells, and interneurons. We show that interneuron inhibition and sparse connectivity between LGN cells could be key factors for explaining a number of observed classical and extraclassical response properties in LGN of monkey and cat. Among them are 1) weak orientation tuning, 2) contrast invariance of spatial frequency tuning in the absence of cortical feedback, 3) extraclassical surround suppression, and 4) orientation tuning of extraclassical surround suppression. The model also makes two surprising predictions: 1) a possible pinwheel-like spatial organization of orientation preference in the parvo layers of monkey LGN, much like what is seen in V1, and 2) a stimulus-induced trend (bias) in the orientation and phase preference of surround suppression, originating from the stimulus discontinuity between center and surround gratings rather than from specific circuitry.

INTRODUCTION

The lateral geniculate nucleus (LGN) constitutes an important processing stage in the early visual pathway and is the major source of afferent sensory input into the primary visual cortex (V1). Compared with V1, however, surprisingly little effort has gone into developing anatomically and physiologically constrained models of the LGN. One possible reason is that the LGN is perceived to be “less interesting” because its anatomy and its responses to visual stimulation are in many respects simpler than those of V1. However, the attraction for modeling studies is that several visual response properties are shared by LGN and V1, with some of them appearing in less pronounced form in LGN than in V1. Given the dazzling complexity of V1, the simpler anatomy of the LGN and its shared response properties with V1 make it an interesting object for modelers to explore in terms of identifying mechanisms underlying early vision.

Among the visual responses shared by LGN and V1 are classical orientation tuning, which is substantially weaker in LGN than in V1 (Shou and Leventhal 1989; Shou et al. 1986; Smith et al. 1990; Sun et al. 2004; Xu et al. 2002), and spatial frequency tuning, which is also somewhat weaker in LGN (mostly low-pass) than in V1 (mostly band-pass) (Hicks et al. 1983; Irvin et al. 1993; Kaplan and Shapley 1982).

LGN and V1 also share several extraclassical response properties. Among them are surround suppression (length tuning) and contrast dependence of the receptive field (RF) size (Anderson et al. 2001; Cavanaugh et al. 2002a; Dow et al. 1981; Felisberti and Derrington 1999, 2001; Jones et al. 2000; Kappadia et al. 1999; Kruger 1977; Levick et al. 1972; Ozeki et al. 2004; Sceniak et al. 1999, 2001, 2006; Schiller et al. 1976; Silito et al. 1995; Solomon et al. 2002). Notably, it has been found that extraclassical surround suppression in LGN is comparable in strength to what is observed in V1, whereas receptive field expansion for low contrast is somewhat less in LGN than in V1.

There is some experimental evidence for at least a partial transfer of extraclassical surround suppression from LGN to V1 (Ozeki et al. 2004; Webb et al. 2005). As of late, further experimental verification of this and to what extent this in fact takes place have been receiving more attention. In our previous work on extraclassical phenomena we also argued, based on a demonstration of feasibility by simulation, that some extraclassical responses in V1 are partially transferred from LGN (Wielaard and Sajda 2006b). Irrespective of whether transfer of extraclassical responses in fact occurs, understanding of the classical and extraclassical responses in LGN is necessary in its own right. In addition, it will help in understanding of the mechanisms governing these responses in V1.

In this study we present a large-scale spiking neuron model of the LGN. We explore what can be achieved in terms of response properties by modeling only the retinal input and neural connectivity between interneurons and relay cells within LGN, while ignoring other inputs such as those from cortex and brain stem. One might argue that this approximation is a rather drastic one. Particularly, in synaptic terms, cortical feedback is well known to be substantial (e.g., Sherman and Guillery 1996; van Horn et al. 2000). However, it is equally well known that retinal ganglion cells are dominant in driving responses in LGN (e.g., Reid and Shapley 1992; Usrey et al. 1999). A model like ours, neglecting feedback of any kind, is thus not an unreasonable approximation. These issues are addressed in further detail in the DISCUSSION section.

There are several motivations for studying an LGN model that considers only the feedforward pathway, rather than a model that attempts to address relative contributions from feedforward and feedback connections. One of course is clarity and transparency. Moreover, given our current knowledge of LGN and cortex, addressing these issues on a purely theoretical
basis seems simply not yet feasible. Meaningful answers must necessarily be derived from the outcome of carefully designed experiments.

The parameters of our model are further anatomically and physiologically constrained by relevant data for the magnocellular and parvo cellular layers of macaque and the X-cell network of layer A in cat. We demonstrate that in this way we are able to obtain a variety of classical as well as extraclassical responses, in good agreement with experimental data. Also, we are able to explain some characteristic differences observed in experimental data taken from monkey and cat. By analysis of the neural mechanisms underlying the response properties, we demonstrate that the sparseness of the connectivity, as determined by the length scales of intergeniculate connections, is a key parameter in setting the classical and extraclassical responses of our model LGN.

METHODS

Model summary

We provide a brief description of the LGN model. The model is similar in spirit to our recently developed cortical model (see Wiesel and Sajda 2006a,b).

The model consists of a two-dimensional (2D) sheet of LGN cells and a 2D sheet of retinal ganglion cells. The retinal ganglion cells provide the sole input to our LGN cells; we ignore all other inputs (from cortex, brain stem, etc.). The model includes recurrent inhibitory connections among the LGN cells; it does not include recurrent connections among the retinal ganglion cells.

Following experimental data, the LGN cell population in the model is made up of 75% excitatory cells (relay cells) and 25% inhibitory cells randomly (and independently) on a square lattice. Experimental data is made up of 75% excitatory cells (relay cells) and 25% inhibitory connections among the LGN cells; it does not include recurrent (from cortex, brain stem, etc.). The model includes recurrent inhibitory connections among the LGN cells.

We configured the model for the magnocellular and parvo layers of macaque LGN, as well as for the X-cells in layer A of cat LGN, at parafoveal eccentricities (<5° for macaque, <10° for cat). For the magnocellular and X cells, excitatory connections are randomly taken to be either ON or OFF with an equal number of both types. For the macaque parvocellular simulations all retinal ganglion cells are randomly taken to be one to one for simplicity, i.e., each LGN cell (including interneurons) receives input from its corresponding retinal ganglion cell.

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Following experimental data, the LGN cell population in the model is made up of 75% excitatory cells (relay cells) and 25% inhibitory cells randomly (and independently) on a square lattice. Experimental data show that, in both cat and monkey, the receptive field of an LGN cell closely resembles the receptive field of just one dominant retinal ganglion cell or that of several retinal ganglion cells with strongly overlapping receptive fields (Cleland and Lee 1985; Cleland et al. 1971; Lee et al. 1983; Reid and Shapley 1992; Usrey et al. 1999). Therefore—and also given the 2D nature of the model—the connectivity between retinal ganglion cells and LGN cells is taken to be one to one for simplicity, i.e., each LGN cell (including interneurons) receives input from its corresponding retinal ganglion cell.

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The quantities $g_{E,i}(t, \eta_i)$ and $g_{I,i}(t, [\mathcal{F}], \eta_i)$ are the excitatory and inhibitory conductances of cell $i$. The notation $\eta_{E,i}$ stands for external noise, and $[\mathcal{F}]$ stands for the spike trains of all (inhibitory) interneurons connected to cell $i$. We assume noise, interactions with interneurons, and retinal ganglion cell input act additively in contributing to the total conductance of a cell

$$g_{E,i}(t, \eta_i) = \eta_{E,i}(t) + g_{E,i}^{neu}(t)$$

$$g_{I,i}(t, [\mathcal{F}], \eta_i) = \eta_{I,i}(t) + g_{I,i}^{syn}(t, [\mathcal{F}])$$

(6)

The terms $\eta_{E,i}(t)$ and $\eta_{I,i}(t)$ are external stochastic contributions and are subsequently given. The terms $g_{E,i}^{neu}(t, [\mathcal{F}])$ are the contributions from the (inhibitory) interneurons and include only isotropic connections

$$g_{E,i}^{neu}(t, [\mathcal{F}]) = \int_{-\infty}^{\infty} ds \sum_{j=0} \delta_{ij} (\|\vec{x}_i - \vec{x}_j\|) g_{I,j}(t - \tau) \delta_\tau(s)$$

(7)

where $\mathcal{P}(I)$ denotes the population of interneurons. The functions $G_{ij}(\tau)$ describe the synaptic dynamics of the interneuron synapses and the functions $\epsilon_{ij}(r)$ describe the strength and spatial range of the interneuron interaction with cell $i$. We assume the availability of postsynaptic sites $N_o$ on a cell (dendrites) to decay exponentially as a function of distance with length scale $D$, i.e., $N_o \approx \exp(-r/D^2)$, and make a similar assumption for the presynaptic sites $N_p$ (axons of interneurons), $N_p \sim \exp(-r/A^2)$. The spatial coupling strength (assuming individual synapses have equal strength) between two cells then decays exponentially with length scale $\sigma_{eff} = D^2 + A^2$ and can be written as

$$\epsilon_{ij}(r) = c_{i,j} N_o \exp(-r/\sigma_{eff}^2)$$

(8)

where $r_{ij} = \|\vec{x}_i - \vec{x}_j\|$, and with the normalization constant

$$N_o = \left\{ \sum_{r=0} \exp(-r/\sigma_{eff}^2) \right\}^{-1}$$

(9)

In this way, the parameters $c_{i,j}$ are interaction strengths that define the density and length scale invariant contribution of the interneuron population $\mathcal{P}(I)$ to the conductance of an interneuron itself ($P = I$) or a relay cell ($P = E$). Their numerical values are $c_{i,j} = c_{i,i} = 2$. The change in membrane potential of cell $i$ due to $\mathcal{P}(P)$ due to a single spike of interneuron $j \in \mathcal{P}(I)$ is proportional to $c_{i,j} g_{I,i}(\sigma_{eff}^{-2})^{-1} \exp(-r_{ij}/\sigma_{eff}^2)$, where $\eta_i$ is the cell density of the interneuron population.

The synaptic temporal kernels $G_{ij}(\tau)$ are normalized to unity, $\int_0^\infty G_{ij}(\tau) d\tau = 1$, and are of the form

$$G_{ij}(\tau) = \begin{cases} 0 & \text{if } \tau \leq 0 \\ k(2\tau)^{-\alpha/2} & \text{if } 0 < \tau < \Delta_i \\ k(\Delta_i\alpha\tau)^{-\alpha/2} e^{-(\tau/\Delta_i)^\alpha} & \text{if } \tau \geq \Delta_i \end{cases}$$

(10)

The kernels $G_{ij}$ have a fast $\gamma$-aminobutyric acid (GABA) component set by $\alpha$, chosen from a uniform distribution between 3 and 6 ms, and a slow component (Gibson et al. 1999) defined by $B = 10$ ms, whereas $\Delta = 3/2$. The constants $k$ are normalization constants. These kernels imply a spike memory of the order of 50 ms for the interneuron inhibition.

The external stochastic terms $\eta_{\mu,i}(t)$ in Eq. 6 are given by

$$\eta_{\mu,i}(t) = \eta_{\mu,i}^0 \int_{-\infty}^t G_{E,i}(t - \tau) \delta_{\tau}(\tau) d\tau$$

(11)

where the kernels $G_{E,i}^\mu$ have the same form as Eq. 10, the kernel $G_{E,i}^\mu$ is as given in Wieland and Sajda (2006b), and $\delta_{\tau}$ is the Poisson spike trains [mean firing rates 100 spikes/s ($\mu = E$) and 125 spikes/s ($\mu = I$)] belonging to neuron $i$ (different ones for each cell). The noise strengths $\eta_{\mu,i}^0$ are drawn from a uniform distribution between 0 and 1, and $\eta_{\mu,i}^0$ are drawn from a uniform distribution between 0 and 0.1.

We obtained estimates of the effective interaction length scales $\sigma_{eff}$ for the different configurations from available experimental data (Bickford et al. 1999; Michael 1988; Robson 1993; Sherman and Friedlander 1988; Wilson 1989). Simulations were performed for two different length scales (min–max estimates) for each configuration. The different models are referred to as M1, M2; P1, P2; and X1, X2 for magno, parvo, and cat configurations, respectively. The effective length scales used in the different models are: for the magno models $\sigma_{eff} = 0.2$ mm (M1) and 0.4 mm (M2); for the parvo models $\sigma_{eff} = 0.075$ mm (P1) and 0.15 mm (P2); and for the cat X-cell models $\sigma_{eff} = 0.1$ mm (X1) and 0.2 mm (X2).

We did not include triadic circuitry (see, e.g., Sherman and Guillery 1996) explicitly in the model, i.e., in a synaptic fashion. However, with the model’s circuitry as set, triadic interactions occur entirely spontaneously and are numerous in the model. For about 40% of the relay neurons, the circuitry is such (by chance) that the receptive field (RF) of its retinal ganglion cell overlaps for >93% with the RF of at least one retinal ganglion cell (of the same sign, on or off) belonging to a nearby ($\sigma_{eff}$) interneuron. Recall we have a one to one mapping between ganglion cells and LGN cells, including the interneurons. From a perspective of the visual input, such a relay cell will thus receive triadic interactions in the sense that it will be excited as well as inhibited by the same local visual stimulation. Another motivation for not including triads explicitly on the synaptic level (i.e., triadic synapses) is that our aim in this study is to address sparsity in the connectivity. To this end, it is desirable to keep the circuitry as isotropic as possible without contradicting anatomical data, to properly address the effects of sparsity rather than of specific circuitry.

**Stimuli and data collection**

All experiments were performed with drifting grating stimuli, with luminance given by $I_i(t) = I_{0,i}[1 + \epsilon \cos(\omega t - k \cdot \vec{x})]$, and average luminance $I_{0,i}$ contrast $\epsilon$, temporal frequency $\omega$, and spatial wave vector $k$. We used a temporal frequency of 8 Hz in all simulations, which is close to the averaged preferred temporal frequencies of the model configurations. Unless varied as part of the experiment, the spatial frequency of all gratings was kept fixed and equal to 2, 4, and 1 c/deg for the M, P, and X configurations, respectively. Each stimulus was presented for 3 s and preceded by a 1-s blank stimulus. The procedure was repeated five times with different initial conditions and noise realizations. SES in cycle-trial–averaged responses and conductances are negligible. Experiments were performed at “high” contrast, $\epsilon = 1$, and “low” contrast, $\epsilon = 0.3$.

Classical orientation tuning curves were obtained using large size drifting gratings, seven- to tenfold the average receptive field size. Orientation and direction selectivity are characterized by respectively the orientation index (OI) and direction index (DI)

$$OI = \int_0^{2\pi} \{ \int_0^{2\pi} r(\theta) \exp(2i\phi) d\phi \}^{1/2} r(\theta) d\theta$$

$$DI = \int_0^{2\pi} \{ \int_0^{2\pi} r(\theta) \exp(2i\phi) d\phi \}^{1/2} r(\theta) d\theta$$

(12)

(13)

Here $r(\theta)$ is the response and $\theta$ is the orientation. Smaller $OI$ (DI) indicates a lesser orientation (direction) selectivity. Purely symmetric responses [i.e., $r(\theta + \pi) = r(\theta)$] have $DI = 0$ but can have arbitrary $OI$ values. Responses independent of orientation (no selectivity) have $OI = DI = 0$. If the response differs from zero for only one orientation (maximum selectivity) then $OI = DI = 1$. If the response differs from zero for only two orientations $\pi$ apart then $OI = 1$ and $DI$ is arbitrary.

Spatially averaged responses are obtained by averaging responses of the cells in 0.06-mm patches. For such spatially averaged responses the preferred orientation $\theta_p$ and the orientation index $OI$ were com-
puted, yielding the orientation and orientation selectivity maps shown in Figs. 3 and 4. The gradient \( \nabla \theta = (\nabla_{\varphi}, \nabla_{\gamma}) \) of the orientation map is defined as

\[
\nabla \theta(i, j) = [\theta(i + 1, j) - \theta(i - 1, j)](\sqrt{2}\pi) \quad (14)
\]

with a similar expression for \( \nabla \phi(i, j) \).

Classical spatial frequency tuning curves were obtained using large size drifting gratings, seven- to tenfold the average receptive field size, with fixed orientation. Because the LGN cells show only weak orientation tuning, spatial frequency tuning curves generally differed little at the preferred and orthogonal orientations. The spatial frequency bandwidth is given by

\[
\beta = \log\left(k_{\text{high}}/k_{\text{low}}\right) \quad (15)
\]

where \( k_{\text{high}} \) is the spatial frequency greater than preferred, at half the maximum response, and \( k_{\text{low}} \) is the spatial frequency less than preferred, at half the maximum response. For low-pass cells, i.e., cells that do not have a \( k_{\text{low}} \), we set \( k_{\text{low}} \) equal to the smallest spatial frequency used. In this way practically all cells with bandwidths \( >4 \) are low-pass cells.

For the analysis of surround suppression and contrast-dependent receptive field size the drifting grating was confined to a circular aperture of varying radius \( r_A \). Other parameters of the grating were kept fixed. Simulations were performed for about 25–35 different aperture centers \( \sigma_{\text{eff}} \) apart and confined to the central region (larger than \( \sigma_{\text{eff}} \) removed from the boundary) of the models. Samples for analysis consist of cells with receptive field centers \(<\sigma/20 \) away from the aperture center. (We assumed an LGN cell’s receptive field center to coincide with the corresponding retinal ganglion cell’s receptive field center.) Selected cells have preferred spatial frequency less than \( 1/2 \) Hz of the grating frequency (5 Hz), and a maximum response at low contrast that is \( >1(f_{\text{dc}} + 5) \), where \( f_{\text{dc}} \) is the mean blank response in spikes/s. In this way we collect about 70–80 cells in a sample, with approximately uniformly distributed preferred angles.

Surround suppression is characterized by comparing the neuron’s maximum firing rate to its steady firing rate for large apertures. We define the receptive field size \( r \) as the minimum aperture radius for which the response \( f(r) \) is \( >95\% \) of its maximum. We define the surround size \( R \) as the minimum aperture radius \( >r \) for which the suppression \( f(x) = f_{\text{max}} - f(x) \) is \( >95\% \) of its maximum. We define the asymptotic response \( f_0 \) as the average response beyond \( R \). We define the suppression index \( SI \) as the relative surround suppression

\[
SI = \frac{f_{\text{max}} - f_{\text{dc}}}{f_{\text{max}} - f_{\text{0}}} \quad (16)
\]

where \( f_{\text{dc}} \) is the response to a blank stimulus. The suppression index \( SI \) is similar to the one used in Solomon et al. (2002).

Neural mechanisms in the model are analyzed based on spike responses and conductances. To a good approximation (see Wieland and Sajda 2006b) the relation between instantaneous firing rate \( \langle f(t) \rangle \) and the cycle-trial-averaged excitatory and inhibitory conductances is given by a rectified weighted difference

\[
\langle f(t) \rangle = \delta[(V_k - 1)(g_E(t)) - (V_c + 1)(g_I(t)) - \Delta]. \quad (17)
\]

with cell-dependent, but stimulus- and time-independent, gain \( \delta > 0 \) and threshold \( \Delta \).

RESULTS

We performed simulations for six different model configurations corresponding to the magno (M) and parvo (P) layers of macaque and X-cells in layer A of cat LGN, with two different characteristic length scales each. The different model configurations are referred to as M1, M2; P1, P2; and X1, X2, respectively (see METHODS). The different length scales \( \sigma_{\text{eff}} \) are, respectively, 0.2, 0.4; 0.075, 0.15; and 0.1, 0.2 mm, and were taken from experimental data and represent min–max estimates. The different model configurations differ in the sparseness of their connectivity. This sparsity can be expressed by the dimensionless parameter \( \sigma = 1(\rho/\sigma_{\text{eff}}^2) \), where \( \rho \) is the cell density and with larger \( \sigma \) indicating sparser connectivity. For the M1, M2; P1, P2; and X1, X2 configurations the parameter \( \sigma \) has the value 1/28, 1/114; 1/9, 1/36; and 1/7, 1/28, respectively. The M2 case thus has the least sparse connectivity; the P1 and X1 cases have the most sparse connectivity; and the M1, P2, and X2 cases have intermediate, about equally sparse connectivity.

We note that the visual sparsity is about equal for M, X, and P configurations. Visual sparsity can be expressed by the dimensionless parameter \( \sigma_{\text{sparsity}} = 1/\sigma_{\text{eff}}^2 \). For all cases the visual sparsity is \( \sigma_{\text{sparsity}} = 1/4 \). Further, the dimensionless receptive field scatter is identical for all configurations and equal to 70% of the center size (see METHODS).

Differences between the different cases other than sparseness of the connectivity have deliberately been kept minimal to enhance transparency in the interpretation of the results. In fact, other than sparseness of connectivity, the only relevant difference between M, X, and P configurations is the fact that the M, X cases contain a 1:1 mixture of ON and OFF cells, whereas the P cases contain only one type, either ON or OFF. The most notable compromise made in this respect is that the retinal ganglion cell temporal kernels (impulse response) are identical for all cases. We note that this is acceptable only because we limit ourselves in this study to stationary responses to drifting grating stimuli.

From this modeling perspective, we note also that the M1 and X2 cases are in fact identical up to a trivial scaling factor in the visual field, i.e., the visual length scale of X2 is simply a factor 2.5-fold the visual length scale of M1. Thus covered by essentially the same simulation, the M1 and X2 cases do, however, represent approximations of quite different realities. M1 represents a macaque LGN magno layer with estimated maximally sparse connectivity, whereas X2 represents a layer A of cat LGN with estimated minimally sparse X-cell connectivity.

In what follows we discuss several classical as well as extraclassical response properties observed in the LGN models and identify their neural mechanisms. The discussion of classical response properties is useful in its own right. It also serves to add context and meaning to the discussion of extraclassical response properties, as noted in Wieland and Sajda (2006b). We address the difference in behavior between the M, X, and P models and the function of sparseness of connectivity as expressed by the parameter \( \sigma \). We demonstrate that interneuron inhibition and sparseness of connectivity could be key ingredients in the explanation of classical and extraclassical response phenomena in monkey and cat LGN and why the phenomena quantitatively differ in these animals.

Classical responses

ORIENTATION TUNING. Cells in the model LGN show weak orientation and direction selectivity for large drifting grating
stimuli, in agreement with experimental data (Shou and Leventhal 1989; Shou et al. 1986; Smith et al. 1990; Sun et al. 2004; Xu et al. 2002). Tuning curves for several model cells and the distributions of orientation and direction index over all model cells are shown in Fig. 1. The spatial frequency of the drifting grating was 2 c/deg and temporal frequency was 8 Hz. These results are for the M1(X2) and M2 configurations of the model (see METHODS). Results for the P1, P2, and X1 configurations are qualitatively similar in that we observe about equal orientation selectivity and direction selectivity for P2 as for M1(X2) and, on average, roughly a doubling of these properties for P1 and X1. We thus observe a consistent increase in orientation and direction selectivity for increasing sparsity in the six model configurations.

We also observe a substantial diversity of orientation and direction selectivity in the model, similar to what is seen experimentally. This is illustrated in Fig. 1, which shows the mean spike response (F0) of a nonselective cell (cell III), a cell selective for orientation but not direction (cell II), and a directionally selective cell (cell I).

That the orientation and direction selectivity observed indeed originates in the sparseness of the connectivity is illustrated in Fig. 2, in which plots (top) are shown for the tuning curves of cell I for the two levels of connectivity sparsity set by the inhibitory length scales $\sigma_{\text{eff}}$ values of 0.2 and 0.4 mm. The responses are plotted for 16 grating orientations from $-\pi$ to $7\pi/8$. In the lower section the cell’s excitatory and inhibitory conductivities are plotted for these 16 orientations. Apart from a trivial phase factor, the excitatory conductance $g_{\text{E}}(t)$ of the cell is independent of the grating orientation. This is true for all cells in the model because there is no recurrent excitation. Excitation arises solely from the retinal ganglion cell inputs, which are given by a center–surround convolution with the stimulus and thus rotationally symmetric, apart from a phase factor. Another observation from Fig. 2, which we find to hold in general, is that the mean of the inhibitory conductance $g_{\text{I}}(t)$ is nearly insensitive to the grating orientation (<5% change).

The inhibitory conductance itself, however, shows modulations that do depend, both in amplitude and in phase, on the grating orientation. Clearly, the amplitude of these modulations must decrease with decreasing sparsity, i.e., with increasing $\sigma_{\text{eff}}$, and this is apparent in Fig. 2. The orientation dependence of these modulations in the inhibitory conductance, and thus the sparse connectivity, creates the orientation/direction selectivity we observe in our model. Comparing the responses at grating orientations $\theta = \pi/8$ and $\theta = -\pi/2$ for the cell in Fig. 2, we see that at the maximum response (at $\theta = \pi/8$) $g_{\text{I}}(t)$ and $g_{\text{E}}(t)$ have antiphase modulations, whereas close to the minimum response (at $\theta = -\pi/2$) $g_{\text{E}}(t)$ and $g_{\text{I}}(t)$ have in-phase modulations. Thus underlying the direction selectivity of this cell is a change in its excitatory–inhibitory synaptic drive from push–pull around the preferred direction ($\theta = \pi/8$) to push–pull around the null direction. This is illustrated in the bottom section of Fig. 2.

Figure 2 is just one example of a directionally selective cell in the model. As shown in Fig. 1, we also observe a considerable diversity in orientation and direction selectivity in the model. This diversity can be intuitively understood by using a simple linear approximation to the full nonlinear model. Such an approximation is obtained when we assume that the inhibitory conductance of a particular cell $n$ is simply proportional to the sum of the ganglion cell inputs into all interneurons within a distance $\sigma_{\text{eff}}$. That is, if $N_n$ indicates this set of interneurons, we assume that
where $A_n$, $B_n > 0$. The phase factor $\psi_n(\theta)$ depends on the temporal position of its receptive field, and it has a more or less random behavior for a population of cells. Recall (see METHODS) that, neglecting noise, we also have

\[ g_{\text{ext}}(t; \theta) \sim \sum_{n \in \mathcal{N}_n} [B_n + A_n \cos(\omega t + \psi_n(\theta))], \quad (18) \]

Equations 18 and 19 illustrate several points. First, the amplitude of the waveform resulting from the summation in Eq. 18 will in general be larger when the sum contains fewer terms, i.e., for larger sparsity. Second, whatever waveform results from the summation in Eq. 18, it will in general have a different dependence on the grating orientation $\theta$ than that in Eq. 19. Thus we may in general expect to find some orientation/direction selectivity in the model. Finally, the resulting waveform $g_{\text{ext}}(t)$ depends on the set of neighboring interneurons $\mathcal{N}_n$, which is a different set for each cell $n$, so we may also expect diversity in the orientation/direction selectivity.

Next we turn to the spatial organization of orientation tuning in the model. Figure 3 shows the coarse-grained spatial organization of the preferred orientation of the model configurations $M_1(X_2)$, $M_2$, $P_1$, and $P_2$. The preferred angles $\theta_p$ of the combined response of nearest-neighbor cells within a 30-μm radius are color coded. Interestingly, the images show a behavior similar to that observed in V1 (Blasdel 1992a,b; Obermayer and Blasdel 1993), that is, regions of steady change in $\theta_p$, singularities (pinwheel centers), fractures, and saddles. Red contours indicate boundaries of regions where $\|\nabla \theta_p\| > 0.45$ (fractures; see METHODS). Singularities appear as small black patches, which are regions where $\|\nabla \theta_p\| > 0.75$. Unlike in our V1 model (Wielaard and Sajda 2006b), the orientation maps in Fig. 3, however, appear entirely spontaneous—no particular spatial structure is present in the input. The orientation maps in the LGN model arise from spontaneous dynamic organization (self-organization) of the interneuron inhibition. This is also apparent from the observed trends in the orientation maps, with the maps becoming more organized (more pinwheels) for longer-range inhibition. As can be seen in Fig. 3, we observe approximately a doubling of the number of pinwheels when comparing $M_2$ versus $M_1(X_2)$ and $P_2$ versus $P_1$. In contrast with what we found for orientation/direction selectivity, it is not primarily sparsity that controls the spatial structure of the orientation map. Randomness in the input starkly hinders the self-organization. Orientation maps are better organized when less randomness is present in the input. This is evident from Fig. 3: the configurations $M_1(X_2)$ and $P_2$, which have about

![Sparsity](image-url)

**FIG. 3.** Coarse-grained spatial distribution of orientation preference in the model lateral geniculate nucleus (LGN) for the $M_1(X_2)$, $M_2$, $P_1$, and $P_2$ configurations. Color coded is the preferred angle ($\theta_p$) of the combined response of neighboring cells. Images show regions of steady change in $\theta_p$, singularities, fractures, and saddles. Regions of fractures and singularities are bounded by red contours. Singularities appear as small black patches. Less fractured singularities are indicated by + when clockwise and x when counterclockwise. Note the large number of singularities (pinwheels) for the $P_2$ case. Both cases, magno ($M_1$, $M_2$) as well as parvo ($P_1$, $P_2$), show an increasing singularity density with decreasing sparsity.
equal sparsity, differ greatly in their orientation map, with P2 showing a much better organization. Recall that the primary difference between the two cases is that M1 contains a balanced mixture of on and off cells, whereas P2 contains cells of only one type, which means that the M1(X2) case has a more random input than P2, and this can be seen with the help of Eq. 19. Changing cell n from an on cell to an off cell (or vice versa) simply implies adding an extra phase factor π to it ψn(θ). Thus doing this for half of the cells will broaden the distribution of $\psi_n(\theta)$ over the cell population.

Finally, we discuss the spatial distribution of orientation selectivity in the model. Plots of the spatial organization of the orientation index (OI) for the M1(X2) and P2 cases are shown in Fig. 4. In sharp contrast to the orientation map, the spatial distribution of orientation selectivity looks very similar for the M1(X2) and P2 cases. Because M1(X2) and P2 have about equal sparsity, this is in line with our earlier observation that orientation selectivity in the model depends primarily on the sparseness of the connectivity. Unlike what is observed in V1 (Blasdel 1992b), we do not observe a positive correlation between regions of sharper tuning (blue) and regions of fractures (enclosed by red contours). Rather, on the contrary, as can be seen in Fig. 4, we observe a negative correlation between the two. The explanation (not shown) is that fractures in the orientation map occur where inhibition is relatively less organized, i.e., relatively weak. Regions of smoothly varying orientation preference occur where inhibition is relatively well organized, i.e., relatively strong. Because orientation selectivity is entirely generated by the interneuron inhibition, and stronger inhibition generates higher selective cells, regions of better tuned cells will be located in regions of smoothly varying orientation preference, i.e., will anticorrelate with regions of fractures.

**Spatial Frequency Tuning.** In this section we briefly discuss the model’s behavior as a function of spatial frequency. Of interest by itself, it is also of relevance with regard to the extraclassical phenomena of surround suppression and receptive field expansion, discussed in the following sections.

As described in Methods, we used a center–surround DOG model for the ganglion cell receptive fields. In the model, all ganglion cell receptive fields are identical in their spatial structure, up to a translation. Thus all ganglion cells have identically shaped spatial frequency tuning curves, differing only by a normalization factor (Eq. 1), and the same is true for the feedforward excitatory inputs in our LGN cells. The shape of this tuning curve is illustrated symbolically by the thick dotted curve in Fig. 5A.

Because of the interaction (interneuron inhibition) in the model, the receptive field of an LGN cell will in general differ somewhat from the corresponding ganglion cell receptive field. Shown in Fig. 5, A and B are representative spatial frequency tuning curves for the M1 and M2 models. We see that, as for orientation tuning, the inhibitory interaction generates only a weak sharpening of the tuning, which predominantly occurs at the low-frequency section.

In contrast with orientation tuning, however, where we found a strong dependence of selectivity on sparseness, the spatial frequency selectivity is only weakly dependent on sparseness, and decreases only slightly for M2 (σ_{eff} = 0.4 mm) with respect to M1 (σ_{eff} = 0.2 mm). Note that using the results of Fig. 5, A and B, and of the previous section, we may conclude that, in agreement with experimental observation (Ursley et al. 1999), the center–surround DOG model will be a reasonable approximation for an LGN cell’s receptive field, but with parameters slightly different from those for the corresponding ganglion cell.

The model’s distribution of preferred spatial frequencies, shown in Fig. 5C, agrees reasonably well with experimental data (Hicks et al. 1983; Irvin et al. 1993; Kaplan and Shapley 1982). Because all excitatory inputs have identically shaped spatial frequency tuning curves (thick dotted curve in Fig. 5A), with preferred frequency of 1.05 c/deg, the diversity observed in the spatial frequency tuning is entirely due to the inhibitory interaction resulting from the interneurons. The distribution is seen to be rather insensitive to the sparsity level.

Figure 5D shows a scatterplot of the spatial frequency bandwidth B (see Methods) for high- and low-contrast stimuli. The diversity in bandwidths in the model also shows good agreement with experimental data (Hicks et al. 1983; Irvin et al. 1993; Kaplan and Shapley 1982). Note that the small decrease in sharpening for decreasing sparsity is also apparent in this plot because the open circles (M2, σ_{eff} = 0.4 mm) are shifted slightly away from the origin with respect to the filled circles (M1, σ_{eff} = 0.2 mm). Also note the predominantly low-pass nature of the cell’s spatial frequency tuning, in agreement with experimental observation.

Importantly, however, as illustrated in Fig. 5D, for both levels of sparsity we do not observe a statistically significant change in bandwidth as a function of contrast. A narrowing in bandwidth for lower contrast can be interpreted as the spatial frequency domain equivalent of contrast-dependent receptive field expansion. In both our V1 model (Wieland and Sajda 2004, 2006b) and in experimental data (Nolt et al. 2004; Sceniak et al. 2002) a decrease in spatial frequency tuning bandwidth is observed to accompany low-contrast receptive field expansion. Contrast-dependent receptive field expansion has also been observed in LGN (Solomon et al. 2002); however, recent experimental observations (Sceniak et al. 2006) show that, in the absence of feedback from V1, LGN cells show little or no contrast-dependent receptive field expansion. The results shown in Fig. 5D are thus consistent with these experimental data.

All results discussed in this section are for the M1 and M2 configuration. Spatial frequency tuning curves for the X2 case
are of course identical to those for the M1 but shifted 1/2.5 c/deg to the left (on a log scale) and are in good agreement with experimental data from cat (Derrington and Fuchs 1979; Ro diceck and Stone 1965; So and Shapley 1979). Qualitatively the comparison between X1 and X2 is similar to what we discussed here for M1 and M2. Cells in the P1, P2 configurations have higher preferred spatial frequencies (about twofold) than those in the M1, M2 cases, but bandwidths similar to those in the M1, M2 cases. Again, qualitatively, the relative comparison between P1 and P2 is similar to what is discussed for the M1 and M2 cases.

Extraclassical responses

SURROUND SUPPRESSION AND RECEPTIVE FIELD EXPANSION. As pointed out in Wieliaard and Sajda (2006b), a center–surround DOG model, such as given by Eqs. 1–3 for the ganglion cell receptive fields, does not show surround suppression resulting from the classical surround, for drifting gratings with spatial frequencies equal to or greater than the preferred spatial frequency. At lower spatial frequencies such a model does show surround suppression caused by the classical surround, and we referred to this as classical surround suppression (see Wieliaard and Sajda 2006b). At significantly higher spatial frequencies than preferred (roughly a factor ≥5) such a model shows surround suppression that is unrelated to the classical surround, but caused by resonance between the spatial frequency and the inverse of the center size (see Wieliaard and Sajda 2006b).

Here we seek to address truly extraclassical surround suppression. This is achieved by using drifting gratings with spatial frequency about twofold (rounded to whole numbers) larger than the preferred spatial frequency of the DOG retinal ganglion cell model used. For example, the preferred spatial frequency for the retinal ganglion cells follows from a simple formula that for the M configurations yields 1.05 c/deg, whereas the grating frequency used for the M simulations is 2 c/deg. For the P and X simulations the grating spatial frequencies are 4 and 1 c/deg, respectively (preferred 1.89 and 0.42 c/deg). At these spatial frequencies the model’s retinal ganglion cells do not show surround suppression. Thus for the stimuli used in our simulations, the surround suppression in the model is entirely generated by interneuron inhibition because there is no other source available by which it could occur. Note that the preferred spatial frequency of LGN cells differs in general from that of the ganglion cells and this difference is a result of the interaction with interneurons. All LGN cells selected for study had a preferred spatial frequency less than the grating frequency.

The experimental definition of extraclassical surround suppression requires that it is observable only when stimulation occurs simultaneously in the central part of the receptive field, the so-called classical receptive field. Stimulation of the extraclassical surround alone without stimulation of the classical receptive field yields no response. We note that the model’s surround suppression is consistent with this definition (not shown, but see Wieliaard and Sajda 2006b).

A demonstration that the surround suppression in the model indeed occurs solely by means of the interneuron inhibition is given in Fig. 6, for a representative M1 model cell. Plotted are the cell’s response as a function of aperture size in the top panel and the corresponding conductances in the bottom panel. The drifting grating used had a spatial frequency of 2 c/deg and an 8-Hz temporal frequency. The cell’s preferred spatial fre-
frequency is 1.1 c/deg. We see that the excitatory conductance $g_E$ saturates one aperture after the aperture of maximum response (receptive field size). The excitatory conductance arises solely from the ganglion cell input (and noise; see METHODS) and thus shows no surround suppression after saturation. The inhibitory conductance $g_I$ is seen to continue its increase well after the aperture of maximum response, and this is causing the surround suppression in the cell’s response. That the inhibitory conductance saturates more gradually (as a function of aperture size) than the excitatory conductance originates from the fact that it is generated by neighboring ($\sigma_{eff}$) interneurons that have offset receptive fields with respect to the receptive field of the cell studied. Thus it takes a larger aperture to saturate the excitatory drive of the relevant interneurons than to saturate the excitatory drive of the cell studied.

In fact, we see that after saturation the inhibitory conductance $g_I$ displays a slight surround suppression itself. For this particular cell it has no noticeable effect on the cell’s response. The surround suppression of $g_I$ is generated in similar fashion as surround suppression in the cell’s response: the population of interneurons that create $g_I$ themselves receive inhibition from interneurons that have offset receptive fields with respect to the receptive fields of that population. Thus again it takes a larger aperture to saturate the excitatory drive to the population of interneurons that disinhibit the said cell (i.e., that inhibit the population that inhibits the cell) than to saturate the excitatory drive to the population of interneurons directly inhibiting the cell.

A summary of our results for surround suppression and contrast-dependent receptive field expansion for the M1 case is provided in Fig. 7. This sample consisted of 78 cells. The drifting grating used had spatial and temporal frequencies of 2 c/deg and 8 Hz. Information on how we selected the cells in the sample, contrast levels, definitions of receptive field size, surround size, and suppression index $SI$ are given in METHODS. Briefly, the receptive field size is the smallest aperture of maximum response, the surround size is the smallest aperture of maximum suppression, and the suppression index is the relative suppression with respect to the maximum response.

Figure 7, A and C shows that we observe only a small change in receptive field size as a function of contrast. That we observe only a small change in receptive field size is in line with recent experimental observations for macaque LGN in the absence of cortical feedback (Sceniak et al. 2006). It also is consistent with our observation made earlier regarding the contrast invariance of the model’s spatial frequency tuning (Fig. 5D). However, as can be seen in Fig. 7, B and C, we observe a considerable increase in surround size as contrast is
lowered. Again this is in agreement with experimental data in the absence of cortical feedback (Sceniak et al. 2006). Also note that, although the surround size shows a growth for decreasing contrast, it remains of approximately the same magnitude as that of the classical surround (see METHODS). This agrees with what was recently observed in cat (Bonin et al. 2005) and previously in primates (Solomon et al. 2002). Finally, we find that the growth of the surround size is accompanied by a growth in the spatial summation extent of inhibition, i.e., a growth in the receptive field size associated with the inhibitory conductance (not shown, but see Wielaad and Sajda 2006b). Note that, in the model, the spatial summation extent of excitation is fixed, i.e., is independent of contrast.

In agreement with experimental data (Sceniak et al. 2006; Solomon et al. 2002) we also observe a decrease of the average surround suppression for lower contrast, as shown in Fig. 7D. Note also that the shape of the SI distribution is centered around its mean, which agrees with the experimental observations, and differs from the shape of the suppression index distribution in V1, which is skewed toward SI = 0.

Results discussed are for the M1 configuration. The other configurations yield qualitatively similar results.

**ORIENTATION TUNING OF SUPPRESSION.** We studied the orientation and phase selectivity of surround suppression in the model using an aperture–annulus configuration of two drifting gratings, each with identical spatial and temporal (8 Hz) frequency and contrast. The spatial frequencies of the gratings are again set to about twice the preferred frequency of the retinal ganglion cells for each configuration, as explained earlier. The cell sample used for the analysis in this section is identical to that used to analyze surround suppression and receptive field expansion.

In the simulations, one of the gratings (orientation θC, phase φC) was confined to a centered aperture with radius 1.1-fold the sample averaged classical receptive field size. The second grating (orientation θE, phase φE) was confined to a concentric annulus with inner radius 1.5-fold the sample averaged classical receptive field size and outer radius of 3° for M and P simulations and 7.5° for X simulations. Parameters of the central grating were kept fixed; orientation and phase of the annulus grating were varied. We defined the surround suppression fS as the difference between the response (mean firing rate, F0) when the central grating was presented alone and the response when it was simultaneously presented with the annulus grating. In general the surround suppression depends on the orientation and the phase difference between the center and surround gratings and on θC alone and not on φC alone, i.e.,

\[ fS = fS(\thetaC, \thetaE - \thetaC, \phiE - \phiC) = fS(\thetaC, \Delta\theta, \Delta\phi). \]

We find that for the model the dependence is predominantly on Δθ and Δϕ and only weakly on θC alone. Measures of orientation, direction, and phase selectivity when referring to surround suppression are based on fS (see METHODS).

We observe a rich diversity in surround orientation tuning for the M1(X2) model. Surround tuning curves (fS as a function of Δθ) of three model cells from this configuration are shown in Fig. 8. The responses are plotted for fixed θC and 16 surround grating orientations with Δθ ranging from 0 to 15π/8. Cell A is characterized by a directionally selective surround suppression; maximum suppression occurs when surround and center grating have equal drift direction and minimal suppression occurs when they have approximately opposite drift direction. Cell B is characterized by orientation but not direction selective surround suppression; maximum suppression occurs when center and surround grating have approximately the same orientation and minimal suppression occurs when they have approximately orthogonal orientation. The surround suppression of cell C is nonselective for orientation. In the bottom sections of Fig. 8 the excitatory and inhibitory conductances of cells A and B are plotted for the 16 orientations in the top section.

The behavior of the inhibitory conductances of cells A and B shown in Fig. 8 in fact has two different origins: one is the sparseness of the connectivity, similar to what we observed for classical orientation/direction selectivity; the other, somewhat surprisingly, involves the stimulus itself and is, as we subsequently show, the discontinuity of the stimulus across the aperture–annulus border.

Sparseness of the connectivity primarily causes temporal modulations in the cycle-trial–averaged inhibitory conductance gI(t) (Fig. 8, bottom). These modulations depend, both in amplitude and phase, on the grating orientation. They are cell specific, as explained earlier, and are the major cause of the diversity in the orientation tuning of surround suppression in the model. Note in this respect that the excitatory conductance gE(t) of the cell does not noticeably depend on the surround grating orientation; again, this is true for all cells in the model because there is no recurrent excitation and excitation arises solely from the retinal ganglion cell’s center–surround inputs.
in effect set by the central grating for the cell studied (and not by the surround grating).

The effect of stimulus discontinuity is subsequently addressed in detail. Combined with sparsity in visual space and in connectivity, it primarily creates a trend (noncell specific) in the dependence of the mean \( F_0 \) inhibitory conductances on the relative orientation \( \Delta \theta \) and relative phase \( \Delta \phi \) of center and surround grating.

Returning to Fig. 8 and comparing the responses of cell A at grating orientations \( \Delta \theta = 0 \) and \( \Delta \theta = 6\pi/8 \), we see that around the maximum surround suppression \( \Delta \theta = 0 \) \( g_E(t) \) has a dominant in-phase modulation with respect to \( g_I(t) \), whereas at the minimum suppression \( \Delta \theta = 6\pi/8 \) \( g_I(t) \) has a dominant antiphase modulation. We also see that the amplitude of the modulations plays a role in determining the suppression. Both amplitude and phase variation result from sparseness in the connectivity. Further, we see that the mean \( F_0 \) of the inhibitory conductance contributes to the surround suppression as well, i.e., it has a maximum around \( \Delta \theta = 0 \) and a minimum around \( \Delta \theta = 6\pi/8 \). In fact, as we will see, the surround tuning curve of cell A itself is close to the trend of orientation tuning of the surround suppression in the model. For cell B in Fig. 8, we see that, unlike for cell A, variation in the amplitude of the modulations is a major factor in determining the orientation tuning of the surround suppression for this cell. However, when carefully measured, the mean \( F_0 \) of the inhibitory conductance still follows the trend of orientation tuning of the surround suppression for this cell. However, variation in the modulations is dominant and, as we subsequently show, the surround tuning curve of this cell does not follow the trend of orientation tuning of the surround suppression in the model.

Distributions of orientation and direction indices (OI and DI) for the surround suppression in the M1(X2) model are shown in Fig. 9, A and B. The sample of cells used is the same as that used for the analysis of surround suppression in the previous section (Fig. 7). At high contrast we observe only weak orientation tuning of surround suppression; the average degree of orientation/direction selectivity is about equal to that of the classical orientation/direction selectivity discussed earlier (Fig. 1).

However, as can be seen in Fig. 9, A and B, we observe a large increase in both orientation and direction selectivity of the suppression for low contrast. The average OI and DI increase by a factor of about 2 at low contrast. This is quite unlike the classical orientation/direction selectivity in the model, which we find to decrease for low contrast (average \( OI = 0.06 \) and \( DI = 0.05 \) for low contrast, not shown, but compare Fig. 1).

Note that mere divisive changes (independent of orientation) of the response with contrast leave both OI and SI invariant. Similarly, mere divisive changes (independent of size) of the response (minus blank) leave the suppression index SI invariant. The increase in the average OI and DI of the surround suppression for low contrast is thus in line with the decrease of the average SI with decreasing contrast and is in fact caused by an iceberg effect (not shown). Interestingly, in the model we do not observe an iceberg effect for classical orientation tuning; rather, the responses settle on the background (blank) responses (which are substantial, 10
spikes/s on the average) and thus the average OI and SI decrease for low contrast. Thus neither classical orientation tuning nor orientation tuning of surround suppression is contrast invariant in our model.

The remainder of this section focuses on the origin of the observed trend in surround tuning in the model. In Fig. 9, C and D we plotted the distribution of the preferred direction of the surround tuning (direction of maximum suppression) relative to the drift direction of the central grating, i.e., as a function of the smallest angle $\Delta \theta_p \in [0, \pi]$ between the wave vector $k_p$ of the central grating and the wave vector $k_s$ of the surround grating at the preferred direction of the suppression. These results are for fixed $\theta_c = -\pi$ but are not significantly different at other orientations of the central grating (not shown). Results are shown for high and low contrast and for two phase differences between center and surround gratings $\Delta \phi = 0$ and $\Delta \phi = \pi$. A characteristic of the distributions is that they are skewed toward $\Delta \theta_p = 0$ for $\Delta \phi = 0$ and toward $\Delta \theta_p = \pi$ for $\Delta \phi = \pi$, with somewhat larger variance at low contrast. Thus for $\Delta \phi = 0$ maximum suppression occurs predominantly when center and surround gratings are drifting in approximately the same direction, whereas for $\Delta \phi = \pi$ maximum suppression occurs predominantly when center and surround gratings are drifting in approximately opposite directions. Further, for $\Delta \phi = 0$ there are few cells for which maximum suppression occurs for surround drift directions that differ $>\pi/2$ from the central grating’s drift direction. Conversely, for $\Delta \phi = \pi$ there are few cells for which maximum suppression occurs for surround drift directions that differ $<\pi/2$ from the central grating’s drift direction. Clearly, there is a strongly stimulus dependent aspect (the relative phase $\Delta \phi$) to the surround tuning observed in the model.

A different way to present the findings of Fig. 9, C and D is shown in Fig. 10. The trends $\Delta \Sigma$ of the normalized surround tuning for the sample of cells used are plotted, i.e., the normalized surround suppression averaged over all cells in the sample as a function of orientation or phase, as well as the square root of its variance $\Delta \sigma^2$. More precisely, let $\Delta f(x) = \langle f_s(x) \rangle \langle f_s(x)\rangle$, where $x$ denotes orientation or phase and $\langle \rangle_x$ the average over $x$, then

$$\Delta \Sigma(x) = \langle \Delta f(x) \rangle,$$

$$\Delta \sigma^2(x) = \langle [\Delta f(x) - \langle \Delta f(x) \rangle]^2 \rangle,$$

where $\langle \rangle$ means the average over cells in the sample. Figure 10, A and B shows just what we noted earlier based on Fig. 9, C and D: for center and surround gratings in phase ($\Delta \phi = 0$), cells tend to have close to maximum suppression when center and surround gratings drift in the same direction and close to minimum suppression when they drift in the opposite direction. For center and surround gratings in antiphase ($\Delta \phi = \pi$), the reverse is true: cells tend to have close to maximum suppression when center and surround gratings drift in the opposite direction and close to minimum suppression when they drift in the same direction. We obtain similar results for the phase dependence for fixed drift directions of center and surround gratings. An example is provided in Fig. 10C. We see for center and surround gratings drifting in opposite directions ($\Delta \theta = \pi$) that cells tend to have close to maximum suppression when center and surround gratings are drifting in antiphase ($\Delta \phi = \pi$) and close to minimum suppression when they are drifting in phase ($\Delta \phi = 0$).

What is the origin of the observed trends in the orientation tuning of surround suppression shown in Figs. 9 and 10? As explained in METHODS, the samples used for analysis contain cells from vastly different spatial locations, covering a spatial range multiple times the effective interaction range $\sigma_{eff}$. Therefore that part of the surround tuning caused by modulations in the conductance as a result of sparsity in the connectivity.
cannot leave any bias in the preferred direction of the suppression when averaged over all cells in a sample. Sparsity in the connectivity thus cannot by itself explain the observed trends in Figs. 9 and 10. Clearly, we need a more specific property that all cells in the sample have in common during the simulations and that depends on the relative orientation ($\Delta \theta$) and phase ($\Delta \phi$) of the center and surround gratings. An obvious candidate is the stimulus geometry, particularly the stimulus discontinuity around the aperture–annulus boundary. This is an identical condition shared by all cells in the sample. Further, the environment of each sample cell (all interneurons within a range of $\sigma_{\text{eff}}$ from the cell) contains interneurons that have receptive fields that intersect with the stimulus discontinuity. Contributions from such cells to the total inhibitory input of a sample cell inevitably depend on $\Delta \theta$ and $\Delta \phi$. This idea is illustrated in Fig. 11 for the M1 case. It shows that interneurons with intersecting receptive fields with the stimulus discontinuity make up a large part of the environment of an M1(X2) ($\sigma_{\text{eff}} = 0.2 \text{ mm}$) sample cell and a significantly smaller part of the environment of an M2 ($\sigma_{\text{eff}} = 0.4 \text{ mm}$) sample cell. The idea is thus consistent with the observation that for M2 any trend in the tuning of surround suppression diminishes with respect to M1 (Fig. 10D).

Is the dependence on $\Delta \theta$ and $\Delta \phi$ generated by the stimulus discontinuity specific enough, when averaged over sample cells, to explain the trends in Figs. 9 and 10? To see that indeed it is, one need only take into account the nonlinearity introduced by the rectification in the ganglion inputs in the model and subsequently treat the interneurons as linear devices generating the inhibition from this input. The reason that this quasi-linear explanation works is because a signature of the inhibitory conductance (rather than in its modulations), as the trend in the surround suppression is present in the mean (F0) of the inhibitory conductance closely resemble the trends $\Gamma_{\text{in}}, \Gamma_{\text{out}}$ of the inhibitory conductance and the square root of its variance, respectively, with $\sigma_{\text{eff}}$ replaced by the mean (F0) of the inhibitory conductance. We see that the trends $\Delta \Gamma_{\text{in}}, \Delta \Gamma_{\text{out}}$ in the mean inhibitory conductance closely resemble the trends $\Delta \Sigma$ in the suppression. Further, the variance $\Delta \Sigma^2$ is sufficiently small to ensure that for practically all cells in a sample their mean inhibitory conductance carries a signature of this trend; e.g., for M1 with $\theta_s = -\pi, \Delta \phi = 0$, there is a very low probability to find a cell for which the mean inhibitory conductance does not have a maximum around $\theta_s = -\pi$ and a minimum around $\theta_s = 0$. Following the quasi-linear approximation, these trends are explained when they are also present in the mean (F0) of $\sigma_{\text{eff}}$

FIG. 11. Receptive field centers of interneurons within interaction range $\sigma_{\text{eff}}$ of a relay cell for M1 (left) and M2 (right) configurations. Aperture and inner edge of annulus are indicated by the dashed circles.
highest sparsity in connectivity, we find an increase of factor of about 2 in the average orientation (OI) and direction (DI) indices of the surround suppression. Thus as for orientation selectivity, selectivity of surround suppression in the model consistently increases as a function of increasing sparseness of connectivity. The increased selectivity for the P1 and X1 cases results in the surround tuning being somewhat less insensitive to the orientation of the central grating (as compared with M1, M2, X2, and P2). Further, for P1 and X1 we find that for some combinations of orientations and phases of the central and surround gratings the surround can be facilitatory, whereas for the other configurations (M1, M2, X2, and P2) the effect of the surround is exclusively suppressive for all orientations and phases.

**Discussion**

We presented a large-scale model of the LGN that operates solely with retinal input and inhibitory interactions between LGN cells. The model was further constrained by experimental data for monkey and cat. We explored a number of response properties that are attainable with this model and compared them with experimental data. The main focus of our analysis has been on extraclassical surround suppression, i.e., surround suppression at spatial frequencies higher than the preferred. We analyzed the dependence of the model’s response properties on the sparsity of its connectivity.

The model easily produces the amount of extraclassical surround suppression observed in the LGN of monkey and cat. Thus our work demonstrates that the major source of this phenomenon could be feedforward interneuron inhibition, that is, inhibition generated by interneurons that themselves are driven by retinal input. The surround suppression arises because the neighboring interneurons that generate it in any particular cell have slightly offset receptive fields with respect to the receptive field of that cell, thus requiring a larger stimulus to be activated. Validation of such a scenario is available from experimental data taken from cat (Dubin and Cleland 1977; Singer and Creutzfeldt 1970). However, as far as we know, no systematic experimental studies have been published yet for primates.

Ours is just a possible scenario for the origin of surround suppression in LGN. Possible other sources, not present in the model, are the input from retinal ganglion cells and the corticogeniculate feedback projections from V1. However, it seems hard to argue that the retinal ganglion cell inputs (combined with connectivity between interneurons and relay cells) would not contribute at all to surround suppression in LGN. Retinal ganglion cell inputs are dominant in generating responses in LGN. Thus based on our simulations one may reasonably expect them to be responsible for at least some part of the surround suppression.

The anatomy provides plenty of means for generation of surround suppression by feedback from V1 (see, e.g., Sherman and Guillery 1996; van Horn et al. 2000). However, the general impression so far seems to be that the actual effects of corticogeniculate feedback are of a weakly modular nature (Crick and Koch 1998). Available experimental data regarding surround suppression in LGN with and without V1 feedback indeed reveal only marginal effects of the feedback projections on the strength of the suppression (Cudeiro and Sillito 1996; Sceniak et al. 2006; Webb et al. 2002).

To our knowledge, no experimental studies have yet been published that explicitly verify inheritance of LGN surround suppression from retinal ganglion cells’ inputs. However, each LGN cell receives dominant excitatory input from just one or a few retinal ganglion cells; thus whenever present, inheritance of some surround suppression from retinal ganglion cells by LGN cells seems a reasonable first-order approximation. In recent experimental data, extraclassical surround suppression was found to be present in magno retinal ganglion cells of macaque and, to a far lesser extent, in parvo retinal ganglion cells (Solomon et al. 2006). Another recent observation is that, in the absence of corticothalamic feedback, both magno and parvo LGN cells show a substantial extraclassical surround suppression, the strength of which in fact exceeds the strength of the surround suppression observed in V1 (Sceniak et al. 2006). Curiously, for these data extraclassical surround suppression in the parvo LGN cells is stronger than that in the magno LGN cells, which is opposite to what is observed for the retinal ganglion cells in Solomon et al. (2006). With respect to parvo LGN cells, the experimental data so far thus seem to suggest a significant, if not dominant, role for feedforward interneuron inhibition in the generation of extraclassical surround suppression.

Some caution needs to be observed in interpreting our work if a large part of the LGN surround suppression would be inherited from retinal ganglion cells. No such inheritance takes place in the model as presented. By construction, retinal ganglion cells in the model do not exhibit extraclassical surround suppression. However, including some surround suppression (25%) in our model retinal ganglion cells by lowering the spatial frequency of the stimulus (not shown, but see also Wieland and Sajda 2006b for details) we indeed find an increase of the average amount of suppression for the LGN cells, but qualitatively our conclusions remain the same.

We showed by simulation that retinal input and interneurons alone can provide sufficient extraclassical surround suppression in LGN. We added context and meaning to this main result by showing that the model at the same time maintains reasonable classical response properties as well. We showed that although the interneuron inhibition generates strong extraclassical surround suppression, that same inhibition generates only weak orientation tuning and maintains low-pass spatial frequency selectivity, characteristic of both monkey and cat LGN.

The weak orientation tuning in the model has its origin in the sparsity of the connectivity. We showed that orientation selectivity increases for increasing sparsity. According to our simulations, X-LGN cells in cat and parvo LGN cells in monkey must show better orientation selectivity, overall, than that of magno LGN cells in monkey because their connectivity is less sparse. This predicted trend, albeit subtle, can indeed be recovered in experimental data (Shou and Leventhal 1989; Smith et al. 1990; Sun et al. 2004; Xu et al. 2002). Another trend present in these cited studies is a slight decrease of orientation selectivity with decreasing spatial frequency in both monkey and cat. We observe the same trend in the model (not shown) and, given that orientation selectivity is caused by sparsity, the explanation in fact is straightforward. Key to this observation is the scale of inhomogeneity of the stimulus.
compared with the visual spatial scale set by the inhibitory interaction range. Clearly, when these are of the same order of magnitude, sparseness of the connectivity will be more effective in generating orientation selectivity than when the first is much larger than the latter—thus the decrease of orientation selectivity for lower spatial frequencies.

Predictions of our simulations regarding LGN orientation tuning are that it is not contrast invariant and that orientation preference may be spatially organized in a pinwheel structure. A decrease of orientation selectivity with decreasing contrast is observed in both our cat and monkey simulations. The effect is not related to an iceberg effect, but rather is a result of the relatively high maintained activity of LGN cells in the absence of visual stimulation. As far as we know, systematic studies of contrast dependence of orientation tuning in LGN are unavailable. The model predicts that the spatial pinwheel-like organization of orientation preference is most profound in the monkey parvo layers, and may be much like what is seen in V1. Monkey magno layer and cat X-cell layer A simulations show a significantly less particular spatial organization of orientation preference. There are as yet no experimental data for verification of these findings; however, because the LGN parvo and A layers are close to the outer surface area, collection of optical imaging data may prove challenging but is not, in principle, impossible.

Our simulations also reproduce more subtle properties of extraclassical surround suppression. We observe a decrease of the strength of the suppression with decreasing contrast, similar to what is seen in experiments (Sceniak et al. 2006; Solomon et al. 2002). Further, we observe no notable change in the receptive field size but a substantial change in the surround size when contrast is lowered, which is in agreement with recent observations made for macaque LGN in the absence of corticogeniculate feedback (Sceniak et al. 2006). The absence of contrast-dependent growth of the classical receptive field is confirmed by the contrast invariance of spatial frequency tuning in the model. These observations are qualitatively similar in our monkey and cat simulations.

It is not yet well established whether and how much the extraclassical surround suppression in LGN displays orientation specificity. Little orientation selectivity was observed in marmoset (Solomon et al. 2002), but a considerable orientation selectivity of extraclassical surround suppression was observed in cat LGN (Cudeiro and Sillito 1996; Naito et al. 2004). Further, the extraclassical surround in marmoset was found to lack homogeneity (Webb et al. 2005). Stimulation of the surround alone, at low spatial frequencies, was found to show orientation selectivity comparable to classical orientation selectivity in cat LGN (Sun et al. 2004). Similar results were obtained for cat retinal ganglion cells (Shou et al. 2000). However, stimulation of the surround alone, at low spatial frequencies to elicit a response, is somewhat different from our approach to the problem. In this work we focused on the extraclassical surround that, by definition, does not elicit a response when stimulated in isolation (but see Solomon et al. 2002; Wieland and Sajda 2006b).

We showed by simulation that retinal inputs and interneurons alone can provide orientation selectivity of extraclassical surround suppression. Its origin lies, as for classical orientation tuning, in the sparsity of the connectivity and we showed that it similarly increases for increasing sparsity. Based on our sparsity estimates of the LGN connectivity in monkey and cat, our simulations predict that cat X cells and monkey parvo cells show a weak to more profound orientation selectivity of extraclassical surround suppression, whereas it may be entirely absent in monkey magno LGN cells. Thus the difference in sparsity of the connectivity can explain the difference in extraclassical surround tuning observed experimentally in cat and monkey.

We found that extraclassical surround tuning is not contrast invariant, which, unlike for the classical orientation tuning in the model, is due to an iceberg effect. To our knowledge there are no data yet available that address contrast dependence of extraclassical surround orientation tuning in LGN.

A further prediction of our simulations is a bias (trend) in the orientation tuning of extraclassical surround suppression in LGN, which we have found to originate in the stimulus discontinuity at the aperture–annulus border. This trend depends on the relative orientation and phase of aperture and annulus grating stimuli, but is insensitive to the aperture grating’s orientation itself. For example, when aperture and annulus gratings are in phase, maximum suppression tends to occur when the two gratings have identical drift directions, less suppression for orthogonal drift directions, and minimum suppression tends to occur for opposite drift directions. This particular trend has been partially observed in cat LGN (Cudeiro and Sillito 1996; Naito et al. 2004). Although it was shown to be less profound in the absence of cortical feedback (Cudeiro and Sillito 1996), evaluation of the data in terms of our parameter $\Delta \Sigma$ gives an estimated change in $\Delta \Sigma$ of 0.15 between identical and orthogonal drift directions, in the absence of cortical feedback, which agrees well with what we find in our simulations. This particular trend has also been modeled based on cortical feedback alone (Hayot and Tranchina 2001). Curiously, it has also been observed in macaque V1 (Cavanaugh et al. 2002b), which leads us to speculate that it could be partially transferred from LGN to V1 and/or could be generated within V1 itself by similar mechanisms discussed here for the LGN.

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