Dependence of response properties on sparse connectivity in a spiking neuron model of the lateral geniculate nucleus

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Abstract

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 inter-neurons. We show that inter-neuron inhibition and sparse connectivity between LGN cells could be key factors for explaining a number of observed classical and extra-classical response properties in LGN of monkey and cat. Among them are: (i) Weak orientation tuning, (ii) Contrast invariance of spatial frequency tuning in the absence of cortical feedback, (iii) Extra-classical surround suppression, and (iv) Orientation tuning of extra-classical surround suppression. The model also makes two surprising predictions: (a) A possible pinwheel-like spatial organization of orientation preference in the parvo layers of monkey LGN, much like what is seen V1, and (b) 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.

keywords: lateral geniculate nucleus, orientation tuning, surround suppression, spatial summation, model, simulation

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 to 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 that of V1. However, attractive 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 et al., 1986; Shou and Leventhal, 1989; Smith et al., 1990; Xu et al., 2002; Sun et al., 2004), and spatial frequency tuning, which is also somewhat weaker in LGN (mostly low-pass) than in V1 (mostly band-pass) (Kaplan and Shapley, 1982; Hicks et al., 1983; Irvin et al., 1993)

LGN and V1 also share several extra-classical response properties. Among them are surround suppression (length tuning) and contrast dependence of the receptive field size (Levick et al., 1972; Kruger, 1977; Felisberti and Derrington, 1999; Jones et al., 2000; Felisberti and Derrington, 2001; Solomon et al., 2002; Ozeki et al., 2004; Sceniak et al., 2006; Schiller et al., 1976; Dow et al., 1981; Silito et al., 1995; Sceniak et al., 1999; Kapadia et al., 1999; Sceniak et al., 2001; Anderson et al., 2001; Cavanaugh et al., 2002a; Ozeki et al., 2004). Notably, it has been found that extra-classical surround suppression in LGN is comparable in strength to what is observed in V1, while 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 extra-classical surround suppression from LGN to V1 (Ozeki et al., 2004; Webb et al., 2005). Further experimental verification of this and to what extent this in fact takes place, has, as of late, been receiving more attention. In our previous work on extra-classical phenomena we also argued, based on a demonstration of feasibility via simulation, that some extra-classical responses in V1 are partially transferred from LGN (Wielaard and Sajda, 2006b). Whether transfer of extra-classical responses in fact occurs or not, understanding of the classical and extra-classical 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 paper 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 inter-neurons and relay-cells within LGN, while ignoring other inputs such as from cortex and brainstem. 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 as 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 only considers 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 magno 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 extra-classical 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. Via analysis of the neural mechanisms underlying the response properties, we demonstrate that the sparseness of the connectivity as determined by the length scales of inter-geniculate connections, is a key parameter in setting the classical and extra-classical responses of our model LGN.

Methods

Model summary. In this section we provide a brief description of the LGN model. The model is similar in spirit to our recently developed cortical model, see Wielaard and Sajda, 2006b; Wielaard and Sajda, 2006a.

The model consists of a 2 dimensional sheet of LGN cells and a 2 dimensional sheet of retinal ganglion cells. The retinal ganglion cells provide the sole input to our LGN cells, we ignore all other inputs (from cortex, brainstem 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 (inter-neurons) (LeVay and Ferster, 1979; Madarasz et al., 1986; Montero and Zempel, 1986; Montero, 1986). The cells are distributed randomly (and independently) on a square lattice. Experimental data shows 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 (Lee et al., 1983; Reid and Shapley, 1992; Cleland et al., 1971; Cleland and Lee, 1985; 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 inter-neurons) receives input from its corresponding retinal ganglion cell.

We configured the model for the magno and parvo layers of macaque LGN, as well as for the X-cells in layer A of cat LGN, at para-foveal eccentricities ($< 5^\circ$ for macaque, $< 10^\circ$ for cat). For the magno macaque and X cat simulations, retinal ganglion cells are randomly taken to be either ON or OFF with an equal number of both cell types. For the parvo macaque simulations all retinal ganglion cells are taken to be of one type (ON or OFF), consistent with anatomical observations (Schiller and Malpeli, 1978).

Retinal ganglion cell responses are modeled as rectified center-surround difference-of-Gaussian (DOG) spatio-temporal linear filters,

$$g_{j}^{RGC}(t) = g_{j}^{0} \left[ 1 + \gamma_{j} V \right].$$
\[
\int_{-\infty}^{\infty} ds \int d^2 y \, G_j^{RGC}(t-s) \, \mathcal{L}(\|\vec{y}_j - \tilde{y}\|) \, I(\tilde{y},s) \big|_+, \tag{1}
\]

Here \([x]_+ = x\) if \(x \geq 0\) and \([x]_+ = 0\) if \(x \leq 0\), \(\mathcal{L}(r)\) and \(G_j^{RGC}(\tau)\) are the spatial and temporal retinal ganglion cell kernels respectively, \(\tilde{y}_j\) is the receptive field center of the \(j\)th retinal ganglion (LGN) cell, \(I(\tilde{y},s)\) is the visual stimulus. The parameters \(g^0_j\) represent the maintained activity of retinal ganglion cells and the parameters \(\gamma^V_j\) measure their responsiveness to visual stimulation. The \(g^0_j\) are drawn randomly and independently from a uniform distribution between \(20 \text{s}^{-1}\) and \(25 \text{s}^{-1}\), and \(\gamma^V_j = 10 \text{ cd}^{-1}\). For the retinal ganglion cell kernels we used

\[
G_j^{RGC}(\tau) = \begin{cases} 0 & \text{if } \tau \leq \tau^0_j \\ k \tau^5 (e^{-\tau/\tau_1} - c e^{-\tau/\tau_2}) & \text{if } \tau > \tau^0_j \end{cases}
\tag{2}
\]

and

\[
\mathcal{L}(r) = \pm (1-K)^{-1} \left\{ \frac{1}{\pi \sigma^2_c} e^{-(r/\sigma_c)^2} - \frac{K}{\pi \sigma^2_s} e^{-(r/\sigma_s)^2} \right\},
\tag{3}
\]

where \(k\) is a normalization constant, \(\sigma_c\) and \(\sigma_s\) are the center and surround sizes respectively, and \(K\) is the integrated surround-center sensitivity. Note that the spatial kernel is (for a given configuration) not cell specific, other than that it is ON (+) or OFF (−).

Because we consider here only steady state responses (drifting grating stimuli), we ignored the detailed differences between the temporal kernels (impulse response) for macaque magno, parvo and cat X-cells (Benardete and Kaplan, 1999a; Benardete and Kaplan, 1999b; Usrey et al., 1999; Reid and Shapley, 2002). For all configurations we used the time constants \(\tau_1 = 2.5\) ms, \(\tau_2 = 7.5\) ms and \(c = (\tau_1/\tau_2)^6\), where the delay times \(\tau^0_j\) are taken from a uniform distribution between 10 ms and 20 ms.

The temporal kernels are normalized in Fourier space, \(\int_{-\infty}^{\infty} |\hat{G}_j^{RGC}(\omega)| d\omega = 1\), \(\hat{G}_j^{RGC}(\omega) = (2\pi)^{-1} \int_{-\infty}^{\infty} G_j^{RGC}(t) e^{-i\omega t} dt\), and are fully transient, i.e. \(\hat{G}_j^{RGC}(0) = 0\).

For center and surround sizes we used \(\sigma_c = 0.1^\circ, 0.04^\circ, 0.25^\circ\) (centers) and \(\sigma_s = 0.5^\circ, 0.32^\circ, 1.25^\circ\) (surrounds), for the macaque magno, parvo and cat X-cell configurations respectively. The integrated surround-center sensitivity was set to \(K = 0.55\) (Hicks et al., 1983; Derrington and Lennie, 1984; Spear et al., 1984; Shapley, 1990; Croner and Kaplan, 1995; Linsenmeier et al., 1982).

As retino-geniculate mapping we use the identity mapping plus a small scatter,

\[
\tilde{y}_j = \mu^{-1} \cdot \vec{x}_j + \vec{\nu}_j,
\tag{4}
\]

where \(\vec{x}_j\) is the LGN cell’s spatial location, \(\mu\) is a geniculate magnification factor and \(\vec{\nu}_j\) are random vectors, with components drawn randomly and independently from the uniform distribution on \([-a, a]\) where \(a = 0.7 \sigma_c\). The total number of cells was the same for all model configurations and equal to \(N = 4096\). For magno and parvo configurations we used data on cell densities in the LGN layers and in visual space (Conolly and van Essen, 1984; Malpeli et al., 1996) to compute the sizes
(L \times L) and \( \mu \)'s for the different LGN models, yielding \( L_M = 2.4 \text{ mm}, \ \mu_M = 0.75 \text{ mm/deg} \) and \( L_P = 1.6 \text{ mm}, \ \mu_P = 1.23 \text{ mm/deg} \) respectively. For the cat X-cell configuration we used a magnification factor \( \mu_X = 0.3 \text{ mm/deg} \) (Sanderson, 1971a; Sanderson, 1971b) and an average grid spacing for retinal X-cells of \( L_X (\mu N)^{-1} = 0.125^\circ \) (Wässle et al., 1981; Peichl and Wässle, 1983), yielding \( L_X = 2.4 \text{ mm} \).

Dynamic variables of a model LGN cell \( i \) are its membrane potential \( v_i(t) \) and its spike train \( S_i(t) = \sum_k \delta(t-t_{i,k}) \), where \( t \) is time and \( t_{i,k} \) is its \( k \)th spike time. The membrane potential and spike train of each cell obey a set of \( N \) equations of the form

\[
C_i \frac{dv_i}{dt} = -g_{L,i}(v_i-v_L) - g_{E,i}(t, [S]_E, \eta_E)(v_i-v_E) - g_{I,i}(t, [S]_I, \eta_I)(v_i-v_I), \ i = 1, \ldots, N .
\]

These equations are integrated numerically using a second order Runge-Kutta method with time step 0.1 ms. Whenever the membrane potential reaches a fixed threshold level \( v_T \) it is reset to a fixed reset level \( v_R \) and a spike is registered. The equation can be rescaled so that \( v_i(t) \) is dimensionless and \( C_i = 1, \ v_L = 0, \ v_E = 14/3, \ v_I = -2/3, \ v_T = 1, \ v_R = 0, \) and conductances (and currents) have dimension of inverse time.

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

\[
g_{E,i}(t, \eta_E) = \eta_{E,i}(t) + g_{i}^{RGC}(t)
\]

\[
g_{I,i}(t, [S]_I, \eta_I) = \eta_{I,i}(t) + g_{I,i}^{LGN}(t, [S]_I) .
\]

The terms \( \eta_{E,i}(t) \) and \( \eta_{I,i}(t) \) are external stochastic contributions and are given below. The terms \( g_{I,j}^{LGN}(t, [S]_I) \) are the contributions from the (inhibitory) inter-neurons and include only isotropic connections,

\[
g_{I,j}^{LGN}(t, [S]_I) = \\
\int_{-\infty}^{+\infty} ds \sum_{j \in \mathcal{P}(I)} C_{i,j}((||\vec{x}_i - \vec{x}_j||)|G_{I,j}(t-s)S_j(s),
\]

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

$$C_{I,i}(r) = c_{I,P} N_0 \exp\left[-\left(r/\sigma_{\text{eff}}\right)^2\right],$$

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

$$N_0 = \left\{ \sum_{i \in \mathcal{P}(I)} \exp\left[-\left(||\vec{x}_i||/\sigma_{\text{eff}}\right)^2\right] \right\}^{-1}.\tag{9}$$

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

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

$$G_{I,i}(\tau) = \begin{cases} 0 & \text{if } \tau \leq 0 \\ k_i \left(\tau e^{-\tau/a_i}\right)^5 & \text{if } 0 < \tau < \Delta a_i \\ k_i \left(\Delta a_i e^{-\Delta a_i}\right)^5 e^{-\left(\tau - \Delta a_i\right)/b} & \text{if } \tau \geq \Delta a_i \end{cases}.\tag{10}$$

The kernels $G_{I,i}$ have a fast (GABA) component set by $a_i$, chosen from a uniform distribution between 3 ms and 6 ms, and a slow component (Gibson et al., 1999) defined by $b = 10$ ms, while $\Delta = 3/2$. The constants $k_i$ are normalization constants. These kernels imply a spike memory of the order of 50 ms for the inter-neuron inhibition.

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

$$\eta_{\mu,i}(t) = \eta_{\mu,i}^0 \int_{-\infty}^{\infty} G_{\mu,i}^P(t-\tau) S_{\mu,i}^P(\tau) d\tau.\tag{11}$$

Where the kernels $G_{\mu,i}^P$ have the same form as (10), the kernel $G_{E,i}^P$ is as given in Wielaard and Sajda, 2006b, and $S_{\mu,i}^P$ are 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 $\eta_{E,i}^0$ are drawn from a uniform distribution between 1 and 6, and $\eta_{I,i}^0$ are drawn from a uniform distribution between 0 and 10.

We obtained estimates of the effective interaction length scales $\sigma_{\text{eff}}$ for the different configurations from available experimental data (Michael, 1988; Wilson, 1989; Sherman and Friedlander, 1988; Robson, 1993; Bickford et al., 1999). 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 $\sigma_{\text{eff}} = 0.2 \text{ mm}$ (M1) and 0.4 mm (M2), for the parvo models $\sigma_{\text{eff}} = 0.075$ mm (P1) and 0.15 mm (P2), and for the cat X-cell models $\sigma_{\text{eff}} = 0.1 \text{ mm}$ (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 approximately 40% of the relay-neurons, the circuitry is such (by chance) that the 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 ($\sigma_{\text{eff}}$) inter-neuron. Recall we have a 1-1 mapping between ganglion cells and LGN cells, including the inter-neurons. 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, so as 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 $I(\vec{y}, t) = I_0 (1 + \epsilon \cos(\omega t - \vec{k} \cdot \vec{y}))$, and average luminance $I_0$, contrast $\epsilon$, temporal frequency $\omega$, spatial wave vector $\vec{k}$. 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 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. Standard errors in cycle-trial average 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, 7-10 times the average receptive field size. Orientation and direction selectivity are characterized by respectively the orientation index (OI) and direction index (DI),

$$O I = \left| \frac{\int_0^{2\pi} r(\theta) \exp(2i\theta)d\theta}{\int_0^{2\pi} r(\theta)d\theta} \right|.$$  \hspace{1cm} (12)

$$D I = \left| \frac{\int_0^{2\pi} r(\theta) \exp(i\theta)d\theta}{\int_0^{2\pi} r(\theta)d\theta} \right|.$$  \hspace{1cm} (13)

Here $r(\theta)$ is the response and $\theta$ the orientation. Smaller $O I$ ($D I$) indicates a lesser orientation (direction) selectivity. Purely symmetric responses, i.e. $r(\theta + \pi) = r(\theta)$, have $D I = 0$ but can have arbitrary $O I$. Responses independent of orientation (no selectivity) have $O I = D I = 0$. If the response differs from zero for only one orientation (maximum selectivity) then $O I = D I = 1$. If the response differs from zero for only two orientations $\pi$ apart then $O I = 1$ and $D I$ 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 $O I$ were computed, yielding the orientation and
orientation selectivity maps shown in Figures 3 and 4. The gradient \( \vec{\nabla} = (\nabla_x, \nabla_y) \) of the orientation map is defined as

\[
\nabla_x \theta_P(i, j) = \left[ \theta_P(i + 1, j) - \theta_P(i - 1, j) \right] / (\sqrt{2}\pi) \tag{14}
\]

with a similar expression for \( \nabla_y \theta_P(i, j) \).

Classical spatial frequency tuning curves were obtained using large size drifting gratings, 7-10 times the average receptive field size, with fixed orientation. As 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

\[
\beta = \log_2(k_{\text{high}} / k_{\text{low}}) \tag{15}
\]

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 less than \( \sigma_x / 20 \) 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 the grating’s spatial frequency, preferred temporal frequency within 2 Hz of the grating frequency (8Hz) and a maximum response at low contrast that is greater than \( f_b + 5 \) where \( f_b \) 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 \( f(r_A) \) is > 95% of its maximum. We define the surround size \( R \) as the minimum aperture radius > \( r \) for which the suppression \( f_s(r_A) = f_{\text{max}} - f(r_A) \) is > 95% of its maximum. We define the asymptotic response \( f_\infty \) as the average response beyond \( R \). We define the suppression index \( SI \) as the relative surround suppression,

\[
SI = \frac{f_{\text{max}} - f_\infty}{f_{\text{max}} - f_0}, \tag{16}
\]

where \( f_0 \) 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 Wielaard and Sajda, 2006b) the relation between instantaneous firing rate \( \langle S(t) \rangle \) and the cycle-trial averaged excitatory and
inhibitory conductances is given by a rectified weighted difference,
\[ \langle S(t) \rangle = \delta \left[ (V_E - 1) \langle g_E(t) \rangle - (|V_I| + 1) \langle g_I(t) \rangle - \Delta \right]_+, \tag{17} \]
with cell dependent, but stimulus and time independent gain \( \delta > 0 \) and threshold \( \Delta \).

Results

We performed simulations for 6 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 (Methods). The different length scales \( \sigma_{\text{eff}} \) are respectively 0.2 mm, 0.4 mm; 0.075 mm, 0.15 mm; and 0.1 mm, 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 \( \sigma_V = 1/(\rho_V \sigma_c^2) \), where \( \rho_V \) is the retinal ganglion cell density and \( \sigma_c \) is the center size (Methods). For all cases the visual sparsity is \( \sigma_V \sim 1/4 \). Further, the dimensionless receptive field scatter is identical for all configurations and equal to 70% of the center size (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 then 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 paper 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 times 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, while X2 represent a layer A of cat LGN with estimated minimally sparse X-cell connectivity.

In what follows we discuss several classical as well as extra-classical 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 extra-classical response properties, as noted in Wielaard and Sajda, 2006b. We address the difference in behavior between the M, X and P models and as function of the sparseness of connectivity as expressed by the parameter $\sigma$. We demonstrate that inter-neuron inhibition and sparseness of connectivity could be key ingredients in the explanation of classical and extra-classical 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 et al., 1986; Shou and Leventhal, 1989; Smith et al., 1990; Xu et al., 2002; Sun et al., 2004). Tuning curves for several model cells, as well as the distributions of orientation and direction index over all model cells are shown in Figure 1. The spatial frequency of the drifting grating was 2 c/deg, temporal frequency was 8 Hz. These results are for the M1(X2) and M2 configurations of the model (Methods). The 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 the 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 6 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 Figure 1, which shows the mean spike response (F0) of a non-selective 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 Figure 2. Plotted (top) are the tuning curves of cell I for the two levels of connectivity sparsity set by the inhibitory length scales $\sigma_{\text{eff}} = 0.2$ mm 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 conductances are plotted for these 16 orientations. Apart from a trivial phase factor, the excitatory conductance $g_E(t)$ 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 hence rotationally symmetric, apart from a phase factor. Another observation from Figure 2, which we find to hold in general, is that the mean of the inhibitory conductance $g_I(t)$ is nearly insensitive to the grating orientation (<5% change).

The inhibitory conductance itself, however, shows modulations that do depend, both in amplitude and 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 Figure 2. The orientation dependence of these modulations in the inhibitory conductance, and hence 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 Figure 2, we see that at the maximum response ($\theta = \pi/8$) $g_E(t)$ and $g_I(t)$ have anti-phase modulations, while close to the minimum response ($\theta = -\pi/2$) $g_E(t)$ and $g_I(t)$ 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-push around the null-direction. This is illustrated in the lower section of Figure 2.

Figure 2 is just one example of a directionally selective cell in the model. As shown in Figure 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 non-linear model. Such an approximation is obtained when we assume that the inhibitory conductance of a particular cell \(n\) is simply proportional the sum of the ganglion cell inputs into all inter-neurons within a distance \(\sigma_{\text{eff}}\). That is, if \(\mathcal{N}_n\) indicates this set of inter-neurons, we assume that

\[
g_{I,n}(t; \theta) \sim \sum_{m \in \mathcal{N}_n} [B_m + A_m \cos(\omega t + \psi_m(\theta))]_+ ,
\]

(18)

where \(A_m, B_m > 0\). The phase factor \(\psi_m(\theta)\) depends on the temporal delay of the retinal ganglion cell as well as the spatial position of its receptive field, and it has a more or less random behavior for a population of cells. Recall (Methods) that, neglecting noise, we also have

\[
g_{E,n}(t; \theta) \sim [B_n + A_n \cos(\omega t + \psi_n(\theta))]_+ .
\]

(19)

Equations (18) and (19) illustrate several points. First, the amplitude of the waveform resulting from the summation in (18) will in general be larger when the sum contains fewer terms, i.e. for larger sparsity. Second, whatever wave form results from the summation in (18), it will in general have a different dependence on the grating orientation \(\theta\) than (19). Thus we may, in general, expect to find some orientation/direction selectivity in the model. Finally, the resulting wave form \(g_{I,n}(t)\) depends on the set of neighboring inter-neurons \(\mathcal{N}_n\), 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 M1(X2), M2, P1 and P2. Color coded are the preferred angle \(\theta_P\) of the combined response of nearest neighbor cells within a 30 \(\mu\text{m}\) radius. Interestingly, the images show a similar behavior as what is observed in V1 (Blasdel, 1992a; Blasdel, 1992b; Obermayer and Blasdel, 1993), namely regions of steady change in \(\theta_P\), singularities (pinwheel centers), fractures and saddles. Red contours indicate boundaries of regions where \(||\vec{\nabla}\theta_P|| > 0.45\) (fractures, see Methods). Singularities appear as small black patches, which are regions where \(||\vec{\nabla}\theta_P|| > 0.75\). Unlike in our V1 model (Wielaard and Sajda, 2006b), the orientation maps in Figure 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 inter-neuron 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 Figure 3, we observe approximately a doubling of the number of pinwheels when comparing M2 vs. M1(X2) and P2 vs. P1. 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 Figure 3: the configurations M1(X2) and P2, which have about 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 while 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 help of equation (19). Changing cell \( n \) from an ON cell to an OFF cell (or vice versa) simply implies adding an extra phase factor \( \pi \) to it \( \psi_n(\theta) \). Hence 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 Figure 4. In sharp contrast to the orientation map, the spatial distribution of orientation selectivity looks very similar for the M1(X2) and P2 cases. Since 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 Figure 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. Since orientation selectivity is entirely generated by the inter-neuron 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 anti-correlate with regions of fractures.

**Spatial frequency tuning.** In this section we briefly discuss the model’s behavior as function of spatial frequency. Of interest by itself, it is also of relevance with regard to the extra-classical phenomena of surround suppression and receptive field expansion which will be discussed in the following sections.

As described in Methods, we used a center-surround difference-of-Gaussians 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. Hence, 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 Figure 5A.

Because of the interaction (inter-neuron 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 Figure 5A 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 ($\sigma_{\text{eff}} = 0.4$ mm) with respect to M1 ($\sigma_{\text{eff}} = 0.2$ mm). Note that using the results of Figure 5A and B, and of the previous section, we may conclude that, in agreement with experimental observation (Usrey et al., 1999), the center-surround difference-of-Gaussians model will be a reasonable approximation for an LGN cell's receptive field. But with slightly different parameters as those for the corresponding ganglion cell.

The model's distribution of preferred spatial frequencies, shown in Figure 5C, agrees reasonably well with experimental data (Kaplan and Shapley, 1982; Hicks et al., 1983; Irvin et al., 1993). Since 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 inter-neurons. The distribution is seen to be rather insensitive to the sparsity level.

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

Importantly however, as illustrated in Figure 5D, for both levels of sparsity we do not observe a statistically significant change in bandwidth as 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 our V1 model (Wielaard and Sajda, 2004; Wielaard and Sajda, 2006b) as well as in experimental data (Sceniak et al., 2002; Nolt et al., 2004) 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 Figure 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 log scale), and are in good agreement with experimental data from cat (Rodieck and Stone, 1965; Derrington and Fuchs, 1979; 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 2 times) as the M1, M2 cases, but similar bandwidths as 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.

**Extra-classical responses**

**Surround suppression and receptive field expansion.** As pointed out in Wielaard and Sajda, 2006b, a center-surround
difference-of-Gaussians (DOG) model, such as given by Eqs. (1,2,3) for the ganglion cell receptive fields, does not show surround suppression resulting from the classical surround, for drifting gratings with spatial frequencies equal 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 Wielaard and Sajda, 2006b. At significantly higher spatial frequencies than preferred (roughly a factor 5 and larger) such a model shows surround suppression which is unrelated to the classical surround, but caused by resonance between the spatial frequency and the inverse of the center size, see Wielaard and Sajda, 2006b.

In this paper we wish to address truly extra-classical surround suppression. This is achieved by using drifting gratings with spatial frequency about 2 times (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 which for the M configurations yields 1.05 c/deg, while the grating frequency used for the M simulations is 2 c/deg. For the P and X simulations the grating spatial frequencies are 4 c/deg and 1 c/deg respectively (preferred 1.89 c/deg and 0.42 c/deg). At these spatial frequencies the model’s retinal ganglion cells do not show surround suppression. Hence, for the stimuli used in our simulations, the surround suppression in the model is entirely generated by inter-neuron inhibition, as 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 inter-neurons. All LGN cells selected for study had a preferred spatial frequency less than the grating frequency.

The experimental definition of extra-classical 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 extra-classical 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 Wielaard and Sajda, 2006b).

A demonstration that the surround suppression in the model indeed occurs solely by means of the inter-neuron inhibition is given in Figure 6, for a representative M1 model cell. Plotted are the cell’s response as function of aperture size in the upper panel and the corresponding conductances in the lower panel. The drifting grating used had a spatial frequency of 2 c/deg and an 8 Hz temporal frequency. The cell’s preferred spatial frequency is 1.1 c/deg. We see that the excitatory conductance $g_E$ saturates 1 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 hence 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. The fact that the inhibitory conductance saturates more gradually (as function of aperture size) as the excitatory conductance originates from the fact that it is generated by neighboring ($\sigma_{eff}$) inter-neurons which have off-set receptive fields with respect to the receptive field of the cell studied. Hence it takes a larger aperture to saturate the excitatory drive of the relevant inter-neurons 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 inter-neurons that create $g_I$ receive themselves inhibition from inter-neurons which have off-set receptive fields with respect to the receptive fields of that population. Hence it takes again a larger aperture to saturate the excitatory drive to the population of inter-neurons that disinhibit the said cell (i.e. that inhibit the population which inhibits the cell), than to saturate the excitatory drive to the population of inter-neurons 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 Figure 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 7A and C show that we observe only a small change in receptive field size as function of contrast. The fact that we observe only a small change in receptive field size, is in line with recent experimental observations for macaque LGN in 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 Figure 7B and C, we observe a considerable increase in surround size as contrast is lowered. This is again in agreement with experimental data in 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 the classical surround (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 the receptive field size associated with the inhibitory conductance (not shown, but see Wielaard 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 (Solomon et al., 2002; Sceniak et al., 2006) we also observe a decrease of the average surround suppression for lower contrast, as is shown in Figure 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 towards $SI = 0$.

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 2 drifting gratings, each with identical spatial and temporal (8Hz) 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 the one used to analyze surround suppression and receptive field expansion.
In the simulations, one of the gratings (orientation $\theta_C$, phase $\phi_C$) was confined to a centered aperture with radius $1.1 \times$ the sample averaged classical receptive field size. The second grating (orientation $\theta_S$, phase $\phi_S$) was confined to a concentric annulus with inner radius $1.5 \times$ the sample averaged classical receptive field size and outer radius of 3 degrees for M and P simulations, and 7.5 degrees 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 $f_S$ as the difference between the response (mean firing rate, $F_0$) 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 $\theta_C$ alone and not on $\phi_C$ alone, i.e. $f_S = f_S(\theta_C, \theta_C - \theta_S, \phi_C - \phi_S) = f_S(\theta_C, \Delta \theta, \Delta \phi)$. We find that for the model the dependence is predominantly on $\Delta \theta$ and $\Delta \phi$ and only weakly on $\theta_C$ alone. Measures of orientation, direction and phase selectivity when referring to surround suppression are based on $f_S$ (Methods).

We observe a rich diversity in surround orientation tuning for the M1(X2) model. Surround tuning curves ($f_S$ as function of $\Delta \theta$) of three model cells from this configuration are shown in Figure 8. The responses are plotted for fixed $\theta_C$ and 16 surround grating orientations with $\Delta \theta$ ranging from 0 to $15\pi/8$. Cell A is a cell with a directionally selective surround suppression, maximum suppression occurs when surround and center grating have equal drift direction, minimal suppression occurs when they have approximately opposite drift direction. Cell B is a cell with orientation but not direction selective surround suppression, maximum suppression occurs when center and surround grating have approximately the same orientation, minimal suppression occurs when they have approximately orthogonal orientation. The surround suppression of cell C is non-selective for orientation. In the lower sections of Figure 8 the excitatory and inhibitory conductances of cell A and B are plotted for the 16 orientations in the upper section.

The behavior of the inhibitory conductances of cell A and B shown in Figure 8 in fact has two different origins. One of them 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 show below, 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 $g_I(t)$ (Fig. 8, lower sections). 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 $g_E(t)$ of the cell does not noticeably depend of the surround grating orientation. This is again true for all cells in the model as there is no recurrent excitation and excitation arises solely from the retinal ganglion cell’s center-surround inputs, which is in effect set by the central grating for the cell studied (and not by the surround grating).

The effects of the stimulus discontinuity is addressed in detail below. Combined with sparsity in visual space and in connectivity, it primarily creates a trend (non-cell-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 Figure 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 ($\Delta \theta = 0$) $g_I(t)$ has a dominant in-phase modulation with respect to $g_E(t)$, while at the minimum suppression ($\Delta \theta = 6\pi/8$) $g_I(t)$ has a dominant anti-phase modulation. We see that also the amplitude of the modulations plays a role in determining the suppression. Both (amplitude and phase variation) result form sparseness in the connectivity. Further, we see that the mean (F0) 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 $\Delta \phi = 0$, and the mean (F0) of the inhibitory conductance of each cell practically without exception follows this trend. As mentioned, this trend results from the stimulus discontinuity at the aperture-annulus border.

For cell B in Figure 8, we see that unlike for cell A, the variation in the amplitude of the modulations is a major factor in determining the orientation tuning of the surround suppression. Nevertheless, the anti-phase/in-phase (push-pull/push-push) mechanism is still active also for this cell, which can be seen from the conductances at $\Delta \theta = 11\pi/8$ (minimum suppression) and $\Delta \theta = 14\pi/8$ (maximum suppression). The mean (F0) of the inhibitory conductance is rather insensitive to the orientation of the surround grating ($\Delta \theta$) and practically constant for this cell. However, when carefully measured, the mean (F0) of the inhibitory conductance still follows the trend of orientation tuning of the surround suppression for this cell. However the variation in the modulations is dominant and, as we show below, 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 Figure 9A and B. The sample of cells used is the same as this 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 Figure 9A, 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 about a factor of 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 suppression index ($SI$) 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 spks/s on the average) and hence 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 focusses on the origin of the observed trend in surround tuning in the model. In Figure 9C, 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 function of the smallest angle $\Delta\theta_P \in [0, \pi]$ between the wave vector $\mathbf{k}_C$ of the central grating and the wave vector $\mathbf{k}_P$ 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 grating $\Delta\phi = 0$ and $\Delta\phi = \pi$. Characteristic of the distributions is that they are skewed towards $\Delta\theta_P = 0$ for $\Delta\phi = 0$ and towards $\Delta\theta_P = \pi$ 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, while 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 grating drift directions that differ more than $\pi/2$ from the central grating’s drift direction. Conversely, for $\Delta\phi = \pi$ there are few cells for which maximum suppression occurs for surround grating drift directions that differ less than $\pi/2$ 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 Figure 9C, D is shown in Figure 10. Plotted are the trend $\Delta\Sigma$ of the normalized surround tuning for the sample of cells used, i.e. the normalized surround suppression averaged over all cells in the sample as function of orientation or phase, and the square root of its variance $\Delta\sigma^2$ (error bars). More precisely, let $\Delta f_S(x) = (f_S(x) - \langle f_S \rangle_x) / \langle f_S \rangle_x$, where $x$ denotes orientation or phase and $\langle \cdot \rangle_x$ the average over $x$, then

$$\Delta\Sigma(x) = \langle \Delta f_S(x) \rangle_s,$$  \hspace{1cm} (20)

$$\Delta\sigma^2(x) = \left\langle \left[ \Delta f_S(x) - \langle \Delta f_S(x) \rangle_s \right]^2 \right\rangle_s,$$  \hspace{1cm} (21)

where $\langle \cdot \rangle_s$ means the average over cells in the sample. Figure 10A and B show just what we noted earlier based on Figure 9C (D), namely that for center and surround gratings in-phase ($\Delta\phi = 0$), cells tend to have close to maximum suppression when center and surround grating drift in the same direction and close to minimum suppression when they drift in the opposite direction. For center and surround gratings in anti-phase ($\Delta\phi = \pi$), the reverse is true: cells tend to have close to maximum suppression when center and surround grating 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 Figure 10C. We see that, for center and surround gratings drifting in opposite directions ($\Delta\theta = \pi$), cells tend to have close to maximum suppression when center and surround grating are drifting in anti-phase ($\Delta\phi = \pi$) 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 Figures 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 \( \sigma_{\text{eff}} \). Therefore, that part of the surround tuning that is 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 Figures 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 inter-neurons within a range of \( \sigma_{\text{eff}} \) from the cell) contains inter-neurons that have receptive fields that intersect with the stimulus discontinuity. The 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 Figure 11 for the M1 case. It shows that inter-neurons with intersecting receptive fields with the stimulus discontinuity, make up a large part of the environment of an M1(X2) (\( \sigma_{\text{eff}} = 0.2 \) mm) sample cell and a significantly smaller part of the environment of an M2 (\( \sigma_{\text{eff}} = 0.4 \) 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 Figures 9 and 10? To see that indeed it is, one need only to take into account the nonlinearity introduced by the rectification in the ganglion inputs in the model, and treat the inter-neurons subsequently as linear devices generating the inhibition from this input. The reason why this quasi-linear explanation works is because a signature of the trend in the surround suppression is present in the mean (F0) of the inhibitory conductance (rather than in its modulations), as is shown in Figure 12 (left column). Plotted are the trend \( \Delta \Gamma_I \) of the mean (F0) of the inhibitory conductance and the square root of its variance \( \Delta \gamma_I^2 \), i.e. computed from equations (20) and (21) respectively with \( f_S \) replaced by the mean (F0) of the inhibitory conductance. We see that the trends \( \Delta \Gamma_I \) in the mean inhibitory conductance closely resemble the trends \( \Delta \Sigma \) in the suppression. Further, the variance \( \Delta \gamma^2 \) is sufficiently small to assure that for practically all cells in a sample their mean inhibitory conductance carries a signature of this trend, e.g. for M1 with \( \theta_C = -\pi \), \( \Delta \phi = 0 \), 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 the combined excitatory (ganglion cell) input \( \Gamma_{E,i}^0 \) of the inter-neurons that make up the environment of a sample cell \( i \), because the mean of the inhibitory conductance of cell \( i \) is proportional to it. In short, these trends need to be present in

\[
\Gamma_{E,i}^0 = \sum_{j \in \mathcal{P}_i(I)} g_{E,j}^0 ,
\]

where the superscript 0 denotes the F0 components and \( \mathcal{P}_i(I) \) denotes the environment of cell \( i \), i.e. all inter-neurons \( j \) with \( ||\vec{x}_i - \vec{x}_j|| < \sigma_{\text{eff}} \). That this indeed is the case is shown in Figure 12 (right column). Plotted are the trends \( \Delta \Gamma_E \) in \( \Gamma_{E,i}^0 \) (computed from equations (20) and (21) respectively with \( f_S \) replaced by \( \Gamma_{E,i}^0 \)). Clearly the trends \( \Delta \Gamma_I \) and \( \Delta \Gamma_E \) show a reasonable proportionality, and we may conclude that the trends in the surround tuning as function of \( \Delta \theta \) and \( \Delta \phi \) are caused
by the stimulus discontinuity combined with the proper sparsity in the connectivity and in visual space.

So far, our discussion of the results regarding tuning of the surround suppression was based on the model’s M1 configuration. We have shown that sparsity of connectivity is an important factor in the occurrence of orientation tuned suppression in the model. This is also apparent in Figure 10D. It shows that for M2 (σ_{eff} = 0.4 mm), with twice the inhibitory length scale as M1, sparseness has been sufficiently reduced so that practically all orientation tuning of surround suppression has been eliminated. The same conclusion holds for the phase selectivity of the surround suppression in M2 (not shown).

Qualitatively the same trends as discussed for the M1 configuration also hold true for the X1, X2 and P1, P2 configurations. As noted earlier, sparsity for the P2 configuration is about the same as the sparsity for the M1(X2) case, both in visual space and in connectivity. Indeed, for the P2 configuration we find results for orientation tuning of surround suppression which are close to what was presented for the M1(X2) configuration. For the P1 and X1 configurations, the cases with highest sparsity in connectivity, we find an increase of about a factor of 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 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 to 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 which 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 extra-classical surround suppression, i.e. surround suppression at spatial frequencies higher then 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 extra-classical surround suppression observed in the LGN of monkey and cat. Hence, our work demonstrates that the major source of this phenomenon could be feedforward inter-neuron inhibition, that is, inhibition generated by inter-neurons that themselves are driven by retinal input. The surround suppression arises because the neighboring inter-neurons that generate it in any particular cell have slightly off-set receptive fields with respect to the receptive field of that cell, and hence require a larger stimulus to be activated. A validation of such a scenario is available from experimental data taken from cat (Singer and Creutzfeldt, 1970; Dubin and Cleland, 1977). 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 cortico-geniculate feedback projections from V1. However, it seems
hard to argue that the retinal ganglion cell inputs (combined with connectivity between inter-neurons and relay-cells) would not contribute to surround suppression in LGN at all. Retinal ganglion cell inputs are dominant in generating responses in LGN. Hence, 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 via 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 cortico-geniculate feedback are of a weakly modulatory 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; Webb et al., 2002; Sceniak et al., 2006).

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, hence whenever present, inheritance of some surround suppression from retinal ganglion cells by LGN cells seems a reasonable first order approximation. In recent experimental data, extra-classical 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 absence of cortico-thalamic feedback, both magno and parvo LGN cells show a substantial extra-classical 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 extra-classical surround suppression in the parvo LGN cells is stronger than in the magno LGN cells, which is opposite to what is observed for the retinal ganglion cells in Solomon et al., 2006. For what concerns P LGN cells, the experimental data so far thus seems to suggest a significant, if not dominant role for feedforward inter-neuron inhibition in the generation of extra-classical surround suppression.

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

We showed via simulation that retinal input and inter-neurons alone can provide sufficient extra-classical 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 while the inter-neuron inhibition generates strong extra-classical 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 magno LGN cells in monkey, since their connectivity is less sparse. This predicted trend, albeit subtle, can indeed be recovered in experimental data (Shou and Leventhal, 1989; Sun et al., 2004; Smith et al., 1990; 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 is the scale of inhomogeneity of the stimulus compared to 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. Hence 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 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 no experimental data for verification of these findings yet, however, as 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 principal, impossible.

Our simulations also reproduce more subtle properties of extra-classical surround suppression. We observe a decrease of the strength of the suppression with decreasing contrast, similar to what is seen in experiments (Solomon et al., 2002; Sceniak et al., 2006). 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 cortico-geniculate 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 if and how much the extra-classical surround suppression in LGN displays orientation specificity. Little orientation selectivity was observed in marmoset (Solomon et al., 2002), but a considerable orientation selectivity of extra-classical surround suppression was observed in cat LGN (Cudeiro and Sillito, 1996; Naito et al., 2004). Further, the extra-classical 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 focussed on the extra-classical surround, which, by definition, does not elicit a response when stimulated in isolation (but see Solomon et al., 2002; Wielaard and Sajda, 2006b).
We showed via simulation that retinal inputs and inter-neurons alone can provide orientation selectivity of extra-classical 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 extra-classical surround suppression, while it may be entirely absent in monkey magno LGN cells. Thus the difference in sparsity of the connectivity can explain the difference in extra-classical surround tuning observed experimentally in cat and monkey.

We found that extra-classical 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 extra-classical surround orientation tuning in LGN.

A further prediction of our simulations is a bias (trend) in the orientation tuning of extra-classical 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 via similar mechanisms discussed here for the LGN.

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References


Figure captions

Figure 1 Orientation tuning in the M1(X2) and M2 models. (top) Examples of orientation tuning curves for three model cells (I, II, III), with different orientation (OI) and direction (DI) indices. Plotted are average firing rates (F1). Horizontal arrows indicate background firing rate. (bottom) Distributions of orientation index (OI) and direction index (DI). Vertical arrows indicate means, dashed (solid) arrow for unfilled (filled) histograms. The M1(X2) and M2 models have different sparseness of connectivity. Tuning is caused by sparseness of connectivity and is seen to decrease with decreasing sparseness, i.e. for $\sigma_{\text{eff}} = 0.4$ mm. The decrease is significant (Wilcoxon, $P < 0.001$ for OI and $P < 0.000001$ for DI).

Figure 2 Illustration of the mechanism of orientation (direction) tuning for cell I in Figure 1 for the two different degrees of sparseness $\sigma_{\text{eff}} = 0.2$ mm (M1,X2) and $\sigma_{\text{eff}} = 0.4$ mm (M2). Spike responses (F0) are plotted in the top section. Corresponding cycle-trial averaged excitatory and inhibitory conductances for each grating orientation $\theta$ are plotted in the middle section. Orientation (direction) tuning is seen to arise from phase differences in the modulations of the conductances and not from changes in their means. This is illustrated in more detail in the lower section.

Figure 3 Coarse-grained spatial distribution of orientation preference in the model LGN for the M1(X2), M2, P1 and P2 configurations. Color coded is the preferred angle ($\theta_P$) of the combined response of neighboring cells. The 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 clock-wise and $\times$ when counter clock-wise. Note the large number of singularities (pinwheels) for the P2 case. Both cases, magno (M1, M2) as well as parvo (P1, P2), show an increasing singularity density with decreasing sparsity.

Figure 4 Spatial distribution of orientation selectivity in the model. Color coded is the orientation index (OI) of individual cells. Explanation of contours is as in Fig. 4. Note that regions of sharper tuning (blue) anti-correlate with regions of fractures (bounded by red contours).

Figure 5 Summary of spatial frequency tuning properties in the model for the two magno configurations M1 ($\sigma_{\text{eff}} = 0.2$ mm) and M2 ($\sigma_{\text{eff}} = 0.4$ mm). (A,B) Spatial frequency tuning curves (firing rate, first harmonic F1) obtained with a large, high contrast 8 Hz drifting grating of varying spatial frequency for 4 model cells (same cells in both models), and for a retinal ganglion cell (thick dotted curve, drawn on arbitrary scale for clarity). (C) Distribution of the preferred spatial frequency $k_P$. (D) Scatterplot of the spatial frequency bandwidth ($\beta$) at high and low contrast.

Figure 6 Surround suppression via inter-neuron inhibition. Spike responses (F0) are plotted in the top section for high and low contrast. For high contrast, corresponding cycle-trial averaged excitatory and inhibitory conductances for each aperture radius are plotted in the lower section. Surround suppression is seen to arise from the increase in inhibition with increasing aperture.


As explained in the text, the excitatory conductance $g_E(t)$ (i.e. retinal ganglion cell input) is seen to show no surround suppression (lower panel). This is also illustrated by the gray dashed curve in the top panel (maximum of $g_E(t)$ as function of aperture size). The same holds for low contrast (not shown). Surround suppression decreases for low contrast with no notable change in the receptive field size.

**Figure 7** Summary of surround suppression and receptive field expansion in the M1 model. Sample consisted of 78 cells. Arrows indicate means, dashed (solid) arrow for unfilled (filled) histograms. (A) Distribution of receptive field size at high and low contrast. (B) Distribution of surround size at high and low contrast. (C) Distribution of low/high contrast ratio for receptive field and surround sizes. Growth in receptive field size and surround size are statistically significant (Wilcoxon, $P < 0.0001$). (D) Distribution of surround suppression at high and low contrast. Decrease in surround suppression is significant (Wilcoxon, $P < 0.000001$). For the X2 configuration, results for growth ratios and suppression index are identical, distributions of receptive field and surround sizes shift by a factor of 2.5 to the right.

**Figure 8** Orientation tuning of surround suppression in the M1(X2) model. Surround tuning curves ($F_0$) for three model cells are plotted in top panel. Cycle-trial averaged conductances for cell A and B at corresponding surround orientations are plotted in the lower panels. Notice the 3 properties of the inhibitory conductance that depend on the surround orientation: (i) the phase of the dominant modulation, (ii) the amplitude of the modulations, and (iii) the mean ($F_0$) conductance. Notice that the excitatory conductance does not noticeably depend on the surround orientation.

**Figure 9** Distributions of some measures related to orientation tuning of the surround suppression for M1(X2) at high and low contrast. (A) Distributions of orientation index. (B) Distributions of direction index. (C,D) Distributions of surround drift direction of maximum suppression (relative to drift direction of center grating) for in-phase ($\Delta\phi = 0$) and anti-phase ($\Delta\phi = \pi$) center and surround gratings. Observed changes in OI and DI distributions are significant (Wilcoxon, $P < 0.00001$)

**Figure 10** Trends $\Delta \Sigma$ in the surround suppression $\Delta f_S$ for the M1(X2) and M2 models. (A) Trend for M1(X2) in surround orientation tuning for in-phase center and surround gratings. (B) Trend for M1(X2) in surround orientation tuning for anti-phase center and surround gratings. (C) Trend for M1(X2) in surround phase tuning for oppositely drifting center and surround gratings. (D) Trend for M2 in surround orientation tuning for in-phase center and surround gratings.

**Figure 11** Receptive field centers of inter-neurons 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.

**Figure 12** Similarity between trends $\Delta \Gamma_l$ (left) and $\Delta \Gamma_E$ (right) for M1(X2) and M2.
Figure 1: Orientation tuning in the M1(X2) and M2 models. (top)
Figure 2: Illustration of the mechanism of orientation (direc-
Figure 3: Coarse-grained spatial distribution of orientation preference in the model LGN for the M1 (X2), M2, P1 and P2 configurations.
Figure 4: Spatial distribution of orientation selectivity in the

179x127mm (600 x 600 DPI)
Figure 5: Summary of spatial frequency tuning properties in the
Figure 6: Surround suppression via inter-neuron inhibition. Spike
\[ \sigma_{\text{eff}} = 0.2 \text{ mm (M1,X2)} \]

Figure 7: Summary of surround suppression and receptive field ex-
\[ \sigma_{\text{eff}} = 0.2 \text{ mm (M1,X2)} \]

\[ \theta_c = -\pi, \Delta \phi = 0 \]

Figure 8: Orientation tuning of surround suppression in the

178x227mm (600 x 600 DPI)
Figure 9: Distributions of some measures related to orientation
Figure 10: Trends $\Delta \Sigma$ in the surround suppression $\Delta f_S$ for the
Figure 11: Receptive field centers of inter-neurons within interac-

\[ \sigma_{\text{eff}} = 0.2 \text{ mm (M1)} \]

- Relay cell
- ON inter-neuron
- OFF inter-neuron

\[ \sigma_{\text{eff}} = 0.4 \text{ mm (M2)} \]
\[ \sigma_{\text{eff}} = 0.2 \text{ mm (M1,X2)} \]

\[ \sigma_{\text{eff}} = 0.4 \text{ mm (M2)} \]

Figure 12: Similarity between trends \( \Delta \Gamma_I \) (left) and \( \Delta \Gamma_E \) (right)