Supplementary Note

Network architecture. The network we use in our simulations contains three stages: retina, LGN and V1. The retina consists of uncoupled analog units that are driven by the image; the output of each unit is an analog firing rate. The retina feeds into the LGN, which consists of a layer of uncoupled, spiking, excitatory neurons. And finally, the LGN feeds into V1, which also consists of spiking neurons, this time coupled through lateral connections. Unlike the LGN, V1 includes inhibitory as well as excitatory neurons. We describe each of these three layers below.

Retina. The retina, which is modeled after Somers et al. (1995)[1], contains two layers. One layer consists of ON center-surround cells and the other of OFF center-surround cells. Each layer contains 441 cells arranged in a 21 by 21 array, and the spacing between cells, expressed in degrees of visual angle, is 0.2º.

The firing rate of a cell at location (x,y) is determined by the firing rates of the associated center and surround subfields. Specifically,

$$\begin{aligned} r_{\mathrm{ON}}(x,y,t) &= \left[r_{\mathrm{baseline}} + r_{\mathrm{center}}(x,y,t) - r_{\mathrm{surround}}(x,y,t-\delta) \right]^{+} \\ r_{\mathrm{OFF}}(x,y,t) &= \left[r_{\mathrm{baseline}} - r_{\mathrm{center}}(x,y,t) + r_{\mathrm{surround}}(x,y,t-\delta) \right]^{+}, \end{aligned} \tag{1}$$

where $[\cdot]^{+}=\max(\cdot,0)$ denotes rectification and δ is a 3 ms delay between center and surround responses. The center and surround retinal subfield responses are generated by convolving the image with a spatio-temporal receptive field. Letting $\alpha=\{\text{center, surround}\}$, the subfield responses are given by

$$r_{\alpha}(x,y,t) = \int_{-\infty}^{+\infty}\int_{-\infty}^{+\infty}\int_{-\infty}^{t} F_{\alpha}(x-x',y-y')\,G_{\alpha}(t-t')\,I(x',y',t')\,dx'dy'dt'. \tag{2}$$

The center and surround receptive fields $F_{\alpha}(x,y)$ are modeled as circularly symmetric Gaussians,

$$F_{\alpha}(x,y) = \frac{K_{\alpha}}{2\pi\sigma_{\alpha}^{2}} e^{-\frac{x^2+y^2}{2\sigma_{\alpha}^{2}}}, \tag{3}$$

and the temporal impulse response function, $G_{\alpha}(t)$, is modeled as a decaying exponential,

$$G_\alpha(t)=\frac{1}{\tau_\alpha}e^{-\frac{t}{\tau_\alpha}}. \tag{4}$$

We used σ_{center} =0.176º, σ_{surround}=0.53º, K_{center}=17, K_{surround}=16, τ_{center}=10 ms, τ_{surround}=20 ms, and r_{baseline}=15 spk/s.

The stimulus is a rectangular stationary bar of width w, length l and contrast c. The bar appears at time t=0 and forms an angle θ with respect to the x-axis. Without loss of generality, we center the bar at the origin, (0, 0). To compute $r_\alpha(x,y,t)$ we first compute the response when the bar is parallel to the y-axis (θ=0), then rotate into a new frame. Denoting the response to a bar at orientation θ as $r_\alpha(x,y,t|\theta)$, we have, via equation (2),

$$r_\alpha(x,y,t\,|\,\theta=0)=g(c)K_\alpha\left(\int_{-w/2}^{w/2}\frac{1}{\sqrt{2\pi\sigma_\alpha^2}}e^{-\frac{(x-x')^2}{2\sigma_\alpha^2}}dx'\right)\left(\int_{-l/2}^{l/2}\frac{1}{\sqrt{2\pi\sigma_\alpha^2}}e^{-\frac{(y-y')^2}{2\sigma_\alpha^2}}dy'\right)\left(\int_0^t\frac{1}{\tau_\alpha}e^{-\frac{(t-t')}{\tau_\alpha}}dt'\right), \tag{5}$$

where $g(c)$ is the effective intensity of the stimulus at contrast c. Using $\operatorname{erfc}(x)=\int_0^x\frac{2}{\sqrt{\pi}}e^{-x'^2}dx'$, this can be rewritten as

$$r_\alpha(x,y,t\,|\,\theta=0)=g(c)\frac{K_\alpha}{4}\left(\operatorname{erfc}\left(\frac{x+\frac{w}{2}}{\sqrt{2}\sigma_\alpha}\right)-\operatorname{erfc}\left(\frac{x-\frac{w}{2}}{\sqrt{2}\sigma_\alpha}\right)\right)\left(\operatorname{erfc}\left(\frac{y+\frac{l}{2}}{\sqrt{2}\sigma_\alpha}\right)-\operatorname{erfc}\left(\frac{y-\frac{l}{2}}{\sqrt{2}\sigma_\alpha}\right)\right)\left(1\text{-}e^{-\frac{t}{\tau_\alpha}}\right). \tag{6}$$

Tilting the bar by θ with respect to the x-axis is equivalent to a coordinate rotation of -θ. Thus,

$$r_\alpha(x,y,t\,|\,\theta)=r_\alpha(x\cos\theta+y\sin\theta,-x\sin\theta+y\cos\theta,t\,|\,0). \tag{7}$$

In all simulations, the bar's dimensions in degrees of visual angle were w=1º, and l=4º. The effective intensity $g(c)$ was defined to be

$$g(c)=\beta\left[\log_{10}(c)\right]^+, \tag{8}$$

with β=3. This expression was chosen to account for the contrast dependence of LGN responses (see below).

LGN. Following Somers et al. (1995), we assume a one-to-one correspondence between retinal ganglion cells and LGN cells, so that the response of each ganglion cell is uniquely passed on to one LGN cell of the same center polarity. The firing rate of an LGN cell at location (x,y) in response to a bar at orientation θ is either $r_{\mathrm{ON}}(x,y,t-\delta_{syn}|\theta)$ or $r_{\mathrm{OFF}}(x,y,t-\delta_{syn}|\theta)$, depending on whether the LGN is ON or OFF. Here δ_{syn} is the synaptic delay between retinal and LGN cells; it is drawn from a Gaussian distribution with mean 3 ms and standard deviation 1 ms.

Given the parameters listed above, the peak response of the LGN cells (that is, the LGN cell with the largest firing rate) versus contrast, denoted R_{LGN}, is well fit by

$$R_{\mathrm{LGN}}(c) = r_{baseline} + 25\left[\log_{10}(c)\right]^{+}, \tag{9}$$

where, as above, $r_{baseline}$=15 spk/s is the spontaneous firing rate. This relation is consistent with both Somers et al. (1995) and with experimental data[2,3].

Note that this LGN model is simplified in several ways. It does not account for the mild orientation bias that has been reported in LGN responses[4], for the precise firing statistics and bursting in LGN[5,6], or for is the strong temporal correlations that have been observed in LGN responses[7]. These properties are likely to influence the information available in the input to the cortex. However, it is unlikely that the fraction of this information that is transmitted to the cortical stage will depend critically on these assumptions.

V1: feedforward connectivity. The cortical simple cell receptive field structure is established by segregation of ON and OFF LGN inputs into 3 main subfields (OFF-ON-OFF). Thalamocortical connections are defined in a 2 stage process:

1. First, we model the receptive field of each cortical cell with respect to the LGN using a Gabor function, $G(x,y,\theta)$, defined by

$$G(x,y,\theta) = e^{-\left(\frac{x^2}{2\sigma_x^2}+\frac{y^2}{2\sigma_y^2}\right)} \cos(2\pi k\theta). \quad (10)$$

The parameters σ_x and σ_y determine the size of the receptive fields. The anisotropy of the receptive fields is controlled by the parameter $\gamma = \sigma_y^2 / \sigma_x^2$. The parameter k determines the preferred spatial frequency of the receptive fields and was fixed at 0.5 cycles/deg. The receptive fields of all cortical cells are centered at the same position in space; they differ only by their orientation, θ. Positive regions of the Gabor function correspond to ON subfields; negative regions correspond to OFF subfields.

2. The LGN afferents of each cortical cell were randomly chosen within the subfield boundaries, with ON-subfields yielding connections from ON center LGN cells and OFF subfields yielding connections from OFF-center LGN cells. The probability of a connection from an ON-center LGN cell (resp. OFF-center) at position (x,y) to a cortical cell with preferred orientation θ was denoted $P_+(x,y,\theta)$ (resp. $P_-(x,y,\theta)$). The connections probabilities, $P_\pm(x,y,\theta)$, have the form

$$P_\pm(x,y,\theta) = \frac{\left[\pm G(x,y,\theta)\right]^+}{\int dxdy\left[\pm G(x,y,\theta)\right]^+}. \quad (11)$$

Each excitatory cortical cell received 24 LGN ON afferents and 24 OFF afferents; each inhibitory cell received 16 of each. These connections were drawn without replacement from the probability distribution given in equation (11). The strength of a connection, once one is made, is set to $|G(x,y,\theta)|$.

Two sets of parameters were used: (i) In the sharpening model, the parameters of the Gabor function were such that the initial receptive field structure is weakly anisotropic and the inputs are broadly selective to the orientation of the stimulus (σ_x=0.70, σ_y=0.47 $\Rightarrow$ γ=0.44). (ii) In the no-sharpening model on the contrary, the parameters of the Gabor function were such that the subfields are very elongated and the input to the cortex is highly selective to orientation (σ_x=1.09, σ_y=2.45 $\Rightarrow$ γ=5).

V1: Neurons. The V1 layer contains 1008 excitatory neurons and 252 inhibitory neurons. Excitatory neurons are modeled as regular spiking conductance-based integrate-and-fire neurons, while inhibitory neurons are modeled as conductance-based fast-spiking neurons. The neuron model and parameters were taken from Somers et al. (1995). Each cortical neuron is modeled as a single voltage compartment in which the membrane potential is given by

$$C_m \frac{dV_i(t)}{dt} = -\sum_j g_{ij}\left(t-\tau_{ij}\right)\left(V_i(t)-E_{\text{EXCIT}}\right) - \sum_j g_{ij}\left(t-\tau_{ij}\right)\left(V_i(t)-E_{\text{INHIB}}\right) - g_{\text{LEAK}}\left(V_i(t)-E_{\text{LEAK}}\right) - g_{\text{AHP}}(t)\left(V_i(t)-E_{\text{AHP}}\right). \quad (12)$$

The sum over j does not include all presynaptic cells; instead, the presynaptic cells are drawn probabilistically according to a scheme described below. The parameter τ_{ij} is a delay, and $g_{ij}(t)$, the synaptic conductance generated at post-synaptic cell i by the spiking of pre-synaptic cell j, is given by an alpha-function,

$$g_{ij}(t) = \bar{g}_{ij} \sum_l \left[t - t_j^l\right]^+ \left(\frac{e}{\tau_{\text{peak}}}\right) \exp\left(-\frac{t-t_j^l}{\tau_{\text{peak}}}\right). \quad (13)$$

Here t_j^l is the time of l^{th} spike from presynaptic cell j. When the membrane potential exceeds the spike threshold (-55 mV), a spike is emitted, the spike threshold is elevated mimicking a relative refractory period (see Somers et al. (1995) for details), and a K^+ mediated after-hyperpolarization (AHP) conductance was activated. The AHP conductance, $g_{\text{AHP}}(t)$, obeys the same equation as (13) except that the prefactor is $\bar{g}_{\text{AHP}}$ and the sum is over the index i (the cell's own spikes) rather than over j (presynaptic spikes). The values of the peak synaptic conductances, $\bar{g}_{ij}$, are given below. Conductance changes reached their maximal values at τ_{peak}, which was 1 ms for excitatory synapses, 2 ms for inhibitory synapses, and 2 ms for after-hyperpolarization. The small values of τ_{peak} means that we are effectively modeling AMPA and $GABA_A$ synapses; NMDA and $GABA_B$ were not included in this model.

The neuron parameters were as follows. For the reversal potentials we used E_{EXCIT}=0 mV, E_{INHIB}=-70 mV, E_{AHP}=-90 mV, and E_{LEAK}=-65 mV. The membrane capacitance, leakage conductance and after-hyperpolarization conductance of regular spiking (excitatory) neurons were given by C_m= 0.5 nF, g_{LEAK}=25 nS and $\bar{g}_{\text{AHP}}$=40 nS. Fast spiking (inhibitory) neurons had

parameter values of C_m= 0.2 nF, g_{LEAK}=20 nS and $\bar{g}_{AHP}$ =20 nS. See Somers et al. (1995) for more details on the parameters and choice of parameter values.

The neurons were organized into 252 orientation columns, spanning the length of the cortical patch. Each column consists of 4 excitatory neurons and 1 inhibitory neuron. Preferred orientations vary monotonically across columns, with neighboring columns differing by 0.71º (=180º/252) in orientation.

V1: Connectivity. We implemented two models: sharpening and no-sharpening. In the sharpening model, the pattern of the connections between neurons versus the difference in their preferred orientations is chosen so that the connection strengths form, on average, a "Mexican hat" function[1,8]. Specifically, the probability of a connection between two cells is a Gaussian function of the difference in their preferred orientations. The width (standard deviation) of this Gaussian is 7.5º for excitatory projections and 60º for inhibitory ones (note that the numbers refer to degrees in the orientation domain, not visual angle). All cells (both excitatory and inhibitory) receive input from 40 excitatory V1 cells and 30 inhibitory V1 cells. The synaptic conductances are fixed and identical for all connections of the same type: $\bar{g}_{E\to E}$ =1.1 nS, $\bar{g}_{I\to E}$=1.5 nS, $\bar{g}_{E\to I}$= 1.5 nS and $\bar{g}_{I\to I}$=1 nS.

In the no-sharpening model, the only cortical connections that are active are inhibitory to excitatory. This model thus implements a pure "feedforward inhibition" [9]. For simplicity, we assumed that inhibition has no orientation specificity, so it comes equally from cells of all preferred orientations. All excitatory cells receive 30 inhibitory inputs drawn from a uniform distribution, and the peak conductance is $\bar{g}_{I\to E}$ = 6.5 nS.

Note that because the sharpening model leads to an amplification of the thalamic inputs, while the no-sharpening model suppresses them, the strength of the thalamocortical projections differs in the two cases. In the sharpening model, $\bar{g}_{LGN\to E}$ = 5.5 nS; $\bar{g}_{LGN\to I}$=6 nS. In the no-sharpening model, $\bar{g}_{LGN\to E}$ = 20.5 nS ; $\bar{g}_{LGN\to I}$=14.8 nS.

Each synapse has a randomly chosen synaptic delay, which represents the total soma-to-soma time delay for spike evoked PSPs. The delays are drawn from a zero-bounded Gaussian distribution with mean τ_d and standard deviation σ_d. Following Somers et al. (1995), all cortical synapses (both excitatory and inhibitory) have τ_d=3 ms and σ_d=1 ms; LGN to excitatory synapses have τ_d= 10 ms and σ_d=7 ms, and LGN to inhibitory synapses have τ_d=5 ms and σ_d=3 ms.

No external noise was injected at the cortical stage. The variability of the cortical responses was due to the fact that (i) the LGN spikes were drawn from a Poisson distribution (with a seed that was varied from trial to trial) and (ii) at the cortical stage, inhibitory and excitatory inputs were approximately balanced[10].

Parametric study. We implemented one additional sharpening model (S2) and two additional no-sharpening models (NS2 and NS3; see Fig. 5),.

The sharpening and no-sharpening models presented above (S, NS) make different assumptions about the strengths of the thalamocortical inputs. These are weak in the S model (and amplified by the cortex), whereas they are strong in the NS model (and suppressed by the cortex). The new sharpening model (S2) was constructed to explore the dependency of our results on this difference. In S2, like in NS, cortical suppression dominates and the cortex no longer functions as an amplifier. The synaptic conductances are: $\bar{g}_{E\to E}$ = 1.7 nS, $\bar{g}_{I\to E}$ = 3 nS, $\bar{g}_{E\to I}$ = 1.1 nS and $\bar{g}_{I\to I}$ = 0.5 nS.; $\bar{g}_{LGN\to E}$ = 11 nS ; $\bar{g}_{LGN\to I}$ = 12.8 nS.

In the no-sharpening model presented above (NS), the only cortical connections that are active are the projections from inhibitory to excitatory cells (I→E). This architecture was chosen because it represented the simplest implementation of the no-sharpening scheme. The two new no-sharpening models, NS2 and NS3, were constructed to examine the robustness of our results with respect to the connectivity pattern and number of cortical connections. In these new networks, the full set of cortical connections is present (E→E, E→I, I→E, I→I) and the number of connections is chosen to be identical to that used in the sharpening model (40 excitatory and 30 inhibitory synapses onto each cortical cell). NS2 is comparable to S2 in terms of cortical connectivity and strength of thalamocortical projections (but here the input orientation curve is narrow instead of broad, the other small connectivity differences between the two networks are due to the necessity of matching the output tuning curves and variabilities). The intracortical connectivity has a Mexican hat shape: the probability of an excitatory connection between two neurons separated by preferred orientation φ is Gaussian in φ with a width of 20º; the probability of an inhibitory connection between two neurons separated by preferred orientation φ is Gaussian in φ with a width of 60º. The synaptic conductances are: $\bar{g}_{E\to E}$ = 2.5 nS, $\bar{g}_{I\to E}$ = 3.9 nS, $\bar{g}_{E\to I}$ = 1 nS and $\bar{g}_{I\to I}$ = 0.5 nS.; $\bar{g}_{LGN\to E}$ = 11 nS ; $\bar{g}_{LGN\to I}$ = 13.8 nS. In NS3, the excitatory and inhibitory projections are drawn from a flat distribution (each cell receives excitatory and inhibitory projections from neurons of all possible preferred orientations). The synaptic conductances are:

$\bar{g}_{E\to E}$ = 1 nS, $\bar{g}_{I\to E}$ = 6.5 nS, $\bar{g}_{E\to I}$ = 1.8 nS and $\bar{g}_{I\to I}$ = 0.8 nS., $\bar{g}_{LGN\to E}$ = 19.5 nS ; $\bar{g}_{LGN\to I}$ =14.8 nS.

To test the robustness of our results with respect to the correlational structure of the input to V1, we constructed sharpening and no-sharpening models in which all cortical cells received independent input from the LGN. In these models, the retinal and LGN stages described above are replaced by a much simpler scheme in which a V1 cell with preferred orientation φ receives independent Poisson spike trains from N_K LGN neurons (K=E, I). The firing rate of each LGN neuron, denoted $\lambda_K(c,\varphi-\theta)$ where, as above, c is the stimulus contrast and θ is the stimulus orientation, is given by

$$\lambda_K(c,\theta) = r_{baseline} + A_K g(c)\left[\frac{1-\varepsilon_K}{\sqrt{2\pi\sigma_K{}^2}} e^{-\frac{\theta^2}{2\sigma_K{}^2}} + \varepsilon_K\right]. \qquad (14)$$

The parameters in this expression have the following interpretation: A_K controls the maximal spiking rate of each presynaptic LGN cell relative to spontaneous activity, σ_K controls the width of the input orientation tuning curve, and ε_K controls the fraction of LGN inputs that depend on stimulus contrast but not on stimulus orientation. These parameters, along with N_K, were chosen so that the strength of the input matched that used in the original sharpening and no-sharpening models. For both models, N_E =24, N_I =16, A_E=A_I=3.16, and ε_E= ε_I=0.2. In the sharpening model, σ_E=40º, σ_I=45º, $\bar{g}_{LGN\to E}$ = 2.2 nS and $\bar{g}_{LGN\to I}$ =5 nS. In the no-sharpening model, σ_E=σ_I=18º, $\bar{g}_{LGN\to E}$ = 11.4 nS and $\bar{g}_{LGN\to I}$ =15.8 nS. Intracortical conductances were the same as in the original sharpening and no-sharpening models.

All networks were designed to have very similar tuning curve amplitude (33.85 ±1.38 spk/s) and width (σ =14.9º ± 0.82), as measured by fitting a Gaussian function on the population tuning curve averaged over 1008 trials (baseline= 3.1 spk/s ±1.49), and very similar degree of variability, as measured by the Fano Factor (mean Fano = 0.844 ±0.058) .

Data Collection and Analysis

The stimulus was a flashed bar at an orientation of either 89.5º or 90.5º. Stimulus evoked spikes were collected over 500 ms of stimulus presentation. Response statistics were computed after 1008 repetitions of the same stimulus.

Simulation Software

The model was implemented in C and run on an 8-node cluster of Linux machines. Our simulator uses published methods for fourth order Runge-kutta numerical integration of ODEs[11], and a differential equation method for describing GABA and AMPA receptor channel kinetics[12]. The integration time step was 0.5 ms.

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