**Supplementary Notes**

**Analysis of pinwheel stability and recording site variability**

*Supplementary Fig. 2* illustrates an analysis of the reliability of pinwheel center localization, and the effect of variability in electrode localization on the relationship between the tuning of conductance and the local orientation distribution.

*Supplementary Fig. 2a* shows the orientation angle maps in the region immediately surrounding each of our pinwheel cell recordings (two cells were recorded from the same location), calculated from all eight stimulus orientations (left) and from orientations 0°, 45°, 90° and 135° (right). The location of the pinwheel in the four orientation map never deviated from the location in the eight orientation map by more than 38 µm, the radius of the circles overlaid on the maps. As shown previously¹, the locations of some pinwheels differ substantially, as shown by the arrows in case 5. However, we only targeted pinwheels with minimal differences. Analysis of maps computed from the other four intermediate stimulus orientations had differences in pinwheel location of similar magnitude (data not shown). *Supplementary Fig. 2b* shows the effects of error in recording location on our analysis of the relationship between the local map structure and the tuning of conductance (g). For each of our recording locations, we created a “cloud” of 500 locations, with random scatter having a 2D gaussian distribution. We then calculated the map OSI from each of these locations. Next, we fit a line to the relationship between map OSI and g OSI, just as we had for the original map locations. The top three panels are histograms of the slope of the linear relationship calculated from each of the 500 random locations having random error drawn from gaussian distributions with standard deviations of 49, 73 and 98 µm (from top to bottom). The downward
arrow marks the slope of the fit to the data using our original localization (see Fig 3d).

The bottom panel shows the mean +/- SEM of the slopes from these simulations for errors of size (SD) ranging from 13-244 µm. Up to a SD of 73 µm, the real slope value falls within the mean + one SD of the distribution, suggesting that errors of localization up to ~75 µm would have been unlikely to change our estimate of the slope of the dependence of synaptic conductance tuning on the local map structure.

**Detailed model descriptions**

**Single-compartment model neuron.** We started with a single neuron model similar to that described by Destexhe et al. The dynamics of the membrane potential $V$ is described by

$$C_m \frac{dV}{dt} = -g_L (V - E_L) - \sum_{int} I_{int} - \frac{1}{a} I_{syn},$$

where $I_{syn}$ and $I_{int}$ denote the synaptic and the intrinsic voltage-dependent currents, $g_L$ and $E_L$ denote the leak conductance and its reversal potential, $C_m$ denotes the membrane capacitance, and $t$ the time. In the single cell model the parameter values are $g_L = 22.74$ nS, $E_L = -80$ mV, and $C_m = 0.5 nF$. In the network model, we set $C_m = 0.35 nF$, and we chose $g_L^E = 15.7$ nS for the leak conductance of the excitatory and $g_L^I = 31.4$ nS for the inhibitory cells, $E_L = -70$ mV.

Each current $I_{int}$ is described by a Hodgkin-Huxley equation
\[ I_m(t) = \bar{g}m^m(t)h^h(t)(V(t) - E), \]

where \( \bar{g} \) is the peak conductance, \( E \) is the reversal potential, and \( m(t) \) and \( h(t) \) are the activation and inactivation variables. We included three voltage dependent currents: a fast Na\(^+\) current and a delayed-rectifier K\(^+\) current for the generation of action potentials, and a slow non-inactivating K\(^+\) current responsible for spike frequency adaptation. Active conductances (cf. ref. 3) are given by:

The Na\(^+\) current:

\[ I_{Na} = \bar{g}_{Na}m^3h(V - E_{Na}) \]

\[ \frac{dm}{dt} = \alpha_m(V)(1 - m) - \beta_m(V)m \]
\[ \frac{dh}{dt} = \alpha_h(V)(1 - h) - \beta_h(V)h \]

\[ \alpha_m = \frac{-0.32(V - V_T - 13)}{\exp(-(V - V_T - 13)/4) - 1} \]
\[ \beta_m = \frac{0.28(V - V_T - 40)}{\exp((V - V_T - 40)/5) - 1} \]

\[ \alpha_h = 0.128\exp(-(V - V_T - V_S - 17)/18) \]
\[ \beta_h = \frac{4}{1 + \exp(-(V - V_T - V_S - 40)/5)} \]

Parameters: \( V_T = -58 \text{ mV} \), \( V_S = -10 \text{ mV} \) and \( \bar{g}_{Na} = 17.87 \mu S \) and \( E_{Na} = 50 \text{ mV} \).

The ‘delayed-rectifier’ K\(^+\) current:

\[ I_{Kd} = \bar{g}_{Kd}n^4(V - E_K) \]
\[ \frac{dn}{dt} = \alpha_n(V)(1 - n) - \beta_n(V)n \]
\[
\alpha_n = \frac{-0.032(V - V_T - 15)}{\exp(-(V - V_T - 15)/5) - 1} \quad \beta_n = 0.5 \exp(-(V - V_T - 10)/40)
\]

Parameters: \( E_K = -90 \, mV \), \( \bar{g}_{K,i} = 3.46 \, \mu S \).

The non-inactivating \( K^+ \) current:

\[
I_M = \bar{g}_M n(V - E_K) \quad \frac{dn}{dt} = \alpha_n(V)(1-n) - \beta_n(V)n
\]

\[
\alpha_n = \frac{0.0001(V + 30)}{1 - \exp(-(V + 30)/9)} \quad \beta_n = \frac{-0.0001(V + 30)}{1 - \exp((V + 30)/9)}
\]

Parameters: \( \bar{g}_M^E = 0.28 \, \mu S \) for excitatory and \( \bar{g}_M^I = 0.1 \, \bar{g}_M^E \) for inhibitory neurons (network model), i.e. spike-frequency adaptation is reduced for inhibitory neurons.

Single cell model: \( \bar{g}_M^E = 0.25 \, \mu S \).

**Synapses.** The synaptic currents were computed using

\[
I_{syn}(t) = \sum_j \bar{g}_j g_j(t)(V(t) - E_j),
\]

where \( g_j \) and \( E_j \) are the time-dependent conductance and the reversal potential for the \( j \)-th synapse, and \( \bar{g}_j \) is a scale factor (values are given below). Parameters were \( E_j = E_E = 0 \) mV and \( E_j = E_I = -80 \) mV for the excitatory and inhibitory synapses. In the network model we furthermore distinguish between a fast AMPA (\( \alpha \)-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid)-like and a slow NMDA (N-methyl-D-aspartate)-like excitatory
component. The dynamics of the fast excitatory and of the inhibitory synaptic
cconductance is described by

\[ \frac{d}{dt} g_j(t) = -\frac{g_j(t)}{\tau_j} + W_j \sum_k \delta(t-t_j^k), \]

where \( \tau_j \) is the time-constant of the \( j \)-th synapse (\( \tau_j=\tau_E=5 \) ms and \( \tau_j=\tau_I=6 \) ms for fast excitatory and inhibitory synapses), \( W_j \) is a ‘synaptic weight’ describing the impact of a single spike, and where the afferent spike train with spike times \( t_j^k \) is described by the sum of \( \delta \)-functions. For a single spike (at \( t=0 \)) we have

\[ W_j = \frac{1}{\tau_j^2} \int_0^\infty dt \, g_j(t). \]

The values for \( W_j \) are given below.

In the network model the total excitatory postsynaptic potential is the sum of a fast AMPA-like component (as modelled above) and a slow NMDA-like component with equal weight integrated over time. The dynamics of the NMDA-like part is modelled as a difference of Gaussians\(^4\), that is for a presynaptic spike at \( t=0 \text{ ms} \), the NMDA-like conductance follows

\[ g_j(t) = g_{NMDA}(t) = \frac{1}{\tau_1 - \tau_2} (\exp(-t/\tau_1) - \exp(-t/\tau_2)) \]

with time constants \( \tau_1=80 \text{ ms} \) and \( \tau_2=2 \text{ ms} \).
It proved to be necessary to include a slow excitatory component into the network model to achieve network stability while being in a strongly recurrent mode\textsuperscript{5,6}. Nevertheless, the slow NMDA-like excitatory component did not by itself change the tuning of conductances in any way compared to the case where the slow component was absent.

**Deducing the inhibition with the single cell model.** Using our optically imaged orientation maps of cat V1, we first estimated the probability \( P(\Delta \theta; x) \) of a neuron at location \( x \) with preferred orientation \( \theta \) making a synaptic connection to a neuron with preferred orientation \( \theta' \), with \( \Delta \theta \) being the distance between \( \theta \) and \( \theta' \) on the half-circle. We assumed that the connection probability is rotationally symmetric in cortical space and depends on the distance via an alpha-function (see text Fig. 4d). Depending on the preferred orientation of the neuron at \( x \) and the orientation distribution of the local network neighbourhood, this induces the orientation distribution \( P(\Delta \theta; x) \). We then computed the local input OSI as the OSI of the orientation histogram compiled from all pixels with distances not larger than 250 \( \mu \text{m} \) and averaged \( P(\Delta \theta; x) \) over all locations \( x \) with the same local input OSI (bin size 0.1) to obtain an averaged connection probability \( P(\Delta \theta; \text{OSI}) \).

The conductances induced by the model synapses scale linearly with the presynaptic firing rate. In the single cell model we set \( g_j = 1 \) for all synapses and adjusted the synaptic weights \( W_j \) so that mean excitatory and inhibitory synaptic conductances of size \( g_L \) and \( 2g_L \) are induced by presynaptic Poisson spike trains of 7000 sp/s and 3000 sp/s. Then, we calculated the membrane potential of the model cell by means of numerical integration of the membrane potential equation using the simulation software NEURON.
(fixed step size of $\Delta t = 0.25$ ms). For every combination of input firing rates we calculated membrane potential traces of 10 s simulated time. The spike response was then characterized by the time-averaged firing rate; the sub-threshold response was characterized by the mean membrane potential with the action potentials being removed, i.e., removing the membrane potential from 2 ms before to 3 ms after each crossing of the threshold (-54 mV).

The tuning curve of the mean excitatory conductance for a given position in the map (as characterized by its local input OSI) was then calculated using the equation

$$
\left\{ g^E (\theta; \theta_{pr}) \right\} = W^E_{syn} \cdot \left[ f^E_{bg} + p f^E_{aff} (\theta; \theta_{pr}) +right.
\int_{\pm 90} (1 - p) \int d\theta' P(\Delta \theta = \theta' - \theta_{pr}; OSI) \cdot f^E_{rec} (\theta; \theta_{pr} = \theta') \right]$$

Here, $\theta_{pr} = 0$ deg is the preferred orientation of the model neuron, $\theta$ is the stimulus orientation, $P(\Delta \theta = \theta' - \theta_{pr}; OSI)$ is the estimated connection probability, and $f^E_{bg}$ determines the excitatory conductance in the absence of visual stimulation. We chose $f^E_{bg} = 6000$ sp/s. The feedforward input was described by

$$
f^E_{aff} (\theta; \theta_{pr}) = 3.5 \cdot f^E_{bg} \left[ 0.1 + 0.9 \exp \left( -\frac{(\theta - \theta_{pr})^2}{2\sigma^2_{aff}} \right) \right],$$

with $\sigma^2_{aff} = 25$ deg. For the recurrent excitatory input induced by a stimulus of orientation $\theta$, we assumed
with $\sigma_{\text{rec}}^2 = 20$ deg. Parameter $p$ was 0.35, i.e. 65% of the stimulus induced excitation were due to local recurrence. For a pinwheel neuron this leads to an excitation induced by the stimulus of approximately $1.5g_L$ for the preferred orientation. For orientation domain neurons this leads to an excitation of approximately $2.5g_L$, also for the preferred orientation.

Given the mean excitatory conductance and our characterization of the model neuron’s response, we determined for each stimulus orientation $\theta$ the smallest synaptic inhibitory conductance $<g^I(\theta; \theta_{pr}=0)>$ necessary to obtain an orientation tuned spike response given by

$$f^E(\theta; \theta_{pr}) = 3.5 \cdot f^I \cdot \exp\left(-\frac{(\theta - \theta_{pr})^2}{2\sigma_{\text{rec}}^2}\right),$$

i.e., we enforce self-consistency by requiring that the shape of the tuning curve for the spike output matches the firing rate tuning curve $f^E_{\text{rec}}$ assumed for computing the excitatory conductance. For the fit we used the mean-squared error between the desired output firing rate and the firing rate given by our characterization of the model neuron. Note that the much higher absolute value of the $f^E_{\text{rec}}$ is due to integration over multiple presynaptic neurons in the local neighbourhood which may also fire with individual peak responses of around 20 sp/s.
We then calculated the total inhibitory conductance by taking the sum of the synaptic inhibitory conductance and the time-averaged conductance $g_M = \bar{g}_M n$ for the corresponding output firing rate. This is because the latter also hyperpolarizes the neuron and - due to its reversal potential of $E_K = -90$ mV - is likely to contribute to the experimentally measured inhibitory conductances.

The resulting total mean excitatory and inhibitory conductances were normalized to obtain the conductance tuning curves $g_e(\theta)$ and $g_i(\theta)$ (see definitions below). The strong background input leads to an effective membrane time constant of $\tau = 5.8$ ms.

**Supplementary Fig. 3a** shows the dependence of $g_i$ and $g_e$ on the local input OSI together with the data points from the experiments. **Supplementary Fig. 3b** shows that the tuning of the spike response is indeed independent of the local input OSI, but the tuning of the sub-threshold voltage response becomes less tuned as the local input OSI decreases, i.e. closer to pinwheels. These results are summarized in **Supplementary Fig. 3c** for all pinwheel and domain locations. **Supplementary Fig. 3d,e** show that the proposed mechanism of balancing the excitation by local inhibition is also almost independent of the strength of the excitation, i.e. a co-varying inhibition can preserve sharp and location invariant spike tuning.

**Network model architecture.** Our network model consists of two coupled two-dimensional layers of excitatory and inhibitory Hodgkin-Huxley-type point neurons. For simulations with the artificial orientation map, we used a grid of $128 \times 128$ neurons for the excitatory layer and $1/3 \times 128^2$ neurons placed at random locations in the inhibitory
layer. For simulations with the optically imaged orientation maps we used a grid of 90 x 90 neurons for both layers. The two 90 x 90 grids of excitatory and inhibitory ‘inner’ neurons (corresponding to an area 2.25 x 2.25 mm$^2$) were then centered within a larger 114 x 114 grid (corresponding to an area 2.8 x 2.8 mm$^2$) of preferred orientations in order to avoid boundary effects. Neurons that are not ‘inner’ neurons are called ‘surrounding’ neurons. These neurons are not modelled explicitly; they contribute to the lateral excitation and inhibition according to an average spike tuning. Thus the network model contained 75% excitatory and 25% inhibitory cells. All model cells received afferent, recurrent and background synaptic currents.

**Connection probabilities in the network model.** Every neuron receives afferent input from $N_{AF} = 50$ excitatory synapses. Each excitatory neuron receives recurrent excitatory input from $N_{EE} = 60$ and recurrent inhibitory input from $N_{EI} = 40$ neurons. Each inhibitory neuron receives its recurrent excitatory input from $N_{IE} = 60$ and recurrent inhibitory input from $N_{II} = 20$ neurons. All recurrent connections to a given neuron were sampled based on a rotationally symmetric probability distribution having the shape of a Gaussian:

$$P(x) = \begin{cases} 
0 & \text{for } |x| = 0 \text{ (no self - connections)} \\
\frac{1}{\sqrt{2\pi\sigma^2}} \exp\left(-\frac{x^2}{2\sigma^2}\right) & \text{for } 0 < |x| \leq 500\mu m \\
0 & \text{otherwise,}
\end{cases}$$

where $x$ is the distance in $\mu m$ and $\sigma = 125 \mu m$. For our artificial map we assume an average distance of 500 $\mu m$ between pinwheel centres.
The connection probabilities reflect the measured local circuitry. Long-range connections have not been incorporated into the model. In the network model we set $W_j=0.3$ for all synapses. The following values were used for the different types: $\bar{g}^E_{Aff}=38g_L$ (afferent synapses to an excitatory neuron), $\bar{g}^I_{Aff}=32g_L$ (afferent synapses to an inhibitory neuron), $\bar{g}^{EE}=2.12\bar{g}^E_{Aff}$ (recurrent excitation of excitatory neurons), $\bar{g}^{IE}=\bar{g}^{EE}$ (recurrent excitation of inhibitory neurons), $\bar{g}^{EI}=2.8\bar{g}^E_{Aff}$ (recurrent inhibition of excitatory neurons), and $\bar{g}^{II}=0.8\bar{g}^E_{Aff}$ (recurrent inhibition of inhibitory neurons). The $\bar{g}_j$ for an individual synapse is determined by normalizing the above values with respect to the number of synapses of the corresponding type connected to the neuron. The recurrent connections were modelled to have a finite axonal time delay that was Gaussian distributed with mean $4 \text{ ms}$ and standard deviation $2 \text{ ms}$ for excitatory neurons and mean $1.25 \text{ ms}$ and standard deviation $1 \text{ ms}$ for inhibitory neurons. Delay times below the time resolution were set to the integration time step (instantaneous delay).

The model neurons additionally receive background synaptic inputs. The synaptic background conductances $g_{bg}$ are described by a stochastic process similar to an Ornstein-Uhlenbeck process with the following update rule:

$$g_{bg}(t + \Delta t) = g_{bg}^0 + [g_{bg}(t) - g_{bg}^0] \exp(-\Delta t / \tau) + A \cdot N(0,1).$$

$g^0$ is the average conductance, $\tau$ is the background synaptic time constant, $A$ is the amplitude coefficient and $N(0,1)$ is a normally distributed random number with zero
mean and unit standard deviation. The amplitude coefficient has the following analytic expression

\[
A = \sqrt{\frac{D \cdot \tau}{2} \left[1 - \exp\left(-\frac{2 \Delta t}{\tau}\right)\right]}
\]

where \( D \) is the diffusion coefficient

\[
D = 2 \frac{\sigma^2}{\tau}.
\]

Numerical values for the background conductances are \( \tau = \tau_e = 2.7 \, ms \) for excitatory and \( \tau = \tau_i = 10.5 \, ms \) for the inhibitory time constant, \( \sigma = \sigma_e = 0.01g_L \) for the variance of the excitatory, \( \sigma = \sigma_i = 0.01g_L \) for the variance of the inhibitory conductance and \( g_0 = g_{e0} = 0.56g_L \) for the mean excitatory and \( g_0 = g_{i0} = 1.84g_L \) for the mean inhibitory conductance. The reversal potential for the background conductances is \( E_e = -5 \, mV \) and \( E_i = -70 \, mV \). This choice of parameters set the effective membrane time constant of the model excitatory neurons to \( \tau_e = 5.2 \, ms \) and of the model inhibitory neurons to \( \tau_i = 4.2 \, ms \).

**Afferent input:** The feedforward input consists of Poisson spike trains with a maximal firing rate of 30 Hz. The afferent firing rate \( f_{Aff} \) as a function of stimulus orientation is given by a Gaussian distribution added to a baseline

\[
f_{Aff}(\theta; \theta_{pr}) = 30 \, sp/s \left[ (1 - f_{base}) \cdot \exp\left(-\frac{(\theta - \theta_{pr})^2}{2\sigma^2}\right) + f_{base} \right],
\]
where $\theta_{pr}$ is the preferred orientation of the neuron, $\theta$ is the orientation of the stimulus, $\sigma=27.5^\circ$ for excitatory cells and $\sigma=35^\circ$ for inhibitory cells, and $f_{\text{base}}=0.1$ for both inhibitory and excitatory cells alike. The preferred orientation $\theta_{pr}$ as a function of cortical position was chosen according to the optically recorded orientation maps, or, for comparison, according to the orientation map from McLaughlin et al\textsuperscript{7}. The latter consists of four pinwheels with alternating ‘handedness’ with periodic boundary conditions.

**Recurrent input:** Recurrent input to the inner neurons is provided via the recurrent excitatory (fast and slow component) and inhibitory connections. If input from ‘surrounding’ neurons is required, this input was given by Poisson processes with the time independent firing rate

$$f(\theta; \theta_{pr}) = \exp\left(-\frac{(\theta - \theta_{pr})^2}{2\sigma^2}\right),$$

where $\theta_{pr}$ is the preferred orientation at the map location from which the postsynaptic neuron receives its recurrent input from, $\theta$ is the orientation of the stimulus, and $\sigma = 20^\circ$.

No self-consistency was enforced with the firing rate of the inner neurons, but for all simulation results shown in this paper the maximal firing rate of the ‘surrounding’ neurons did not differ by more than 5 Hz from the firing rate averaged over all inner neurons with $\theta_{pr}=\theta$.

**Analysis of the membrane potential, the firing rate and the excitatory and inhibitory conductances.** The network was simulated (Matlab; Mathworks, Natick, MA; fixed step size of $\Delta t=0.25$ ms) until it reached a stationary state (~200 ms). Then the membrane
potential, the afferent conductances, the recurrent excitatory and recurrent inhibitory conductances, and the conductances of the non-inactivating $K^+$-current were recorded for each neuron for a period of 2 s. This was done for nine different stimulus orientations equidistant between $-80^\circ$ and $+80^\circ$. Then spikes were counted, the average membrane potential trace was calculated after removing the spikes from each trace (2 ms before and 4 ms after the peak of each spike). To quantify the position of each neuron in the orientation map, the local input OSI for the preferred orientation map was calculated (see single cell model). The mean membrane potential, the firing rate and the conductance tuning for all pinwheel ($0.1 < OSI < 0.3$) and orientation domain ($0.8 > OSI > 0.6$) neurons were always calculated only for neurons having a preferred orientation differing no more than $3^\circ$ from the stimulus orientation $\theta$. Afferent and recurrent excitatory synaptic conductances (but not background) were pooled for the total excitatory conductance. Recurrent inhibitory synaptic conductances (but not background) and the time-averaged conductance $g_M = \bar{g}_{\text{sli}} n$ of the non-inactivating $K^+$ current were pooled for the total inhibitory conductance. After the network reached its stationary state, the membrane potential, the firing rate and the excitatory, inhibitory, and total conductances were normalized as follows:

$$f_{\text{norm}}(\theta; x) = \frac{f(\theta; x) - f_{\text{bg}}(x)}{\max(f(\theta; x) - f_{\text{bg}}(x))}$$

$$g_{\epsilon}(\theta; x) = \frac{g_{\text{Aff}}^E(\theta; x) + g_{\text{Rec}}^E(\theta; x)}{\max(g_{\text{Aff}}^E(\theta; x) + g_{\text{Rec}}^E(\theta; x), g_M^I(\theta; x) + g_{\text{Rec}}^I(\theta; x))}$$
Here, $f(\theta;x)$ is the firing rate of a neuron at location $x$ in response to stimulus $\theta$ and $f_{bg}(x)$ is the firing rate of that without visual stimulation. The $g_{Aff}^E(\theta;x)$, $g_{Rec}^E(\theta;x)$, $g_{Aff}^I(\theta;x)$, and $g_{Rec}^I(\theta;x)$ are the time-averaged conductances for the afferent excitation, the recurrent excitation, the recurrent inhibition and the non-inactivating $K^+$ current of an excitatory neuron at location $x$ when the stimulus $\theta$ is presented. The OSIs of all normalized tuning curves were then calculated individually for all cells and were either averaged over the population of pinwheel and orientation domain neurons (Fig. 5d and Supplementary Fig. 4) or plotted as a function of the local input OSI (Fig. 5e,f).

Supplementary Fig. 4 shows the tuning curves for the inhibitory conductances, the excitatory conductances, the membrane potential and the firing rate for pinwheel and orientation domain locations. Supplementary Fig. 5 shows the considerable effect of varying the strength of the afferent vs. the recurrent conductance. Using the parameters above, including balanced recurrent excitation and inhibition, we find a strong increase in the OSI of the membrane potential but little increase in the OSI of the firing rate (Wilcoxon rank sum test: p>0.1) in orientation domains compared to pinwheels. Our model is operating in a regime where recurrent excitation significantly contributes to the responses of neurons; the model regime is not in a ‘marginal phase’⁸, where the afferent
input ‘selects’ predefined response patterns and where the tuning width is strongly
determined by the tuning of intracortical inputs. In the model’s parameter regime,
predetermined response patterns or ‘attractor states’ do not exist and weakly tuned
afferent input is not sharpened. Rather, moderately tuned afferent input is sharpened by
the cortical network: whereas the afferent input has a half width at half height (HWHH)
of 32.4°, the tuning of the cortical excitatory cells shows a significantly narrower range of
tuning, with an average HWHH of 26.1°. On the other hand, if we change parameters
such that we use a much stronger recurrent inhibition (dashed lines in Supplementary
Fig. 5), while at the same time increasing the afferent excitatory strength7, the model
produces sharpening of the membrane potential and of the firing rate tuning in pinwheels.
In this feedforward regime, the OSI of excitatory conductance tuning changes little with
map location, because excitation is predominantly feedforward, whereas the OSI of the
inhibitory conductance is larger in the domains than close to pinwheels, because most of
the inhibitory conductance is derived from lateral connections. The broad inhibition in
the pinwheel region then accounts for the sharpening of the tuning curve of the spike
response and the membrane potential at pinwheels. The feedforward mode, however, is
not supported by the data, as it would require the OSI of ge to remain approximately
constant with map location. In particular, the slope of ge for the feedforward regime does
not fall within the 95% confidence interval from the measured values. Furthermore, this
parameter regime predicts location-dependent spike tuning, with sharper orientation
tuning at pinwheels compared to orientation domains, which is not observed
experimentally.
Supplementary Figure Legends

Supplementary Fig. 1. Examples of data used to measure synaptic conductances and passive neuronal properties. (a) Two raw traces (resting and during -0.2 nA current injection) of a cell’s response to visual stimulation for each of 8 orientations and two directions (upper and lower row). For each cell, the same protocol was performed under 3-4 different levels of intracellularly injected current, and repeated 3-5 times. In this example, data was neither adjusted for junction potential nor for series resistance compensation. (b) Current-voltage relationships for the same cell as in a, obtained every stimulus cycle and used to monitor the cell’s biophysical properties. Traces consist of 3 averaged runs of 6 steps of current injection (range -0.3 to 0.2 nA, 100 ms duration).

Supplementary Fig. 2. Analysis of accuracy of pinwheel targeting. (a) Orientation angle maps in the region of the map immediately surrounding each of our pinwheel center recording sites. The left panel in each case is the angle map computed from all eight stimulus orientations, which were used in all analysis. The right panels show the maps computed from only the four cardinal stimulus orientations (shown schematically at the top of the figure). The circle on each map is centered on the location of the pinwheel in the eight orientation map, and has a radius of 38 µm. None of the pinwheel locations in the four orientation maps falls outside this circle, and many fall on exactly the same pixel. There is some variability in the location of other, non-targeted pinwheel centers, as demonstrated by the black arrows in case 5. However, the orientation representation near the recording locations is extremely stable. These figures attest to our choosing extremely stable sites for intracellular recordings. The scale bar represents 500 µm. (b) Effect of random errors in electrode targeting on the analysis of the OSI of the local map.
Top three panels are histograms of the slope of linear fits to the relationship between g OSI and map OSI calculated from 500 randomly offset locations surrounding our determination of the actual recording site. The standard deviation (SD) of the gaussian distribution from which the random locations were drawn is indicated to the upper left. The slope of the real data points is indicated by the downward arrow. The bottom panel plots the mean (+/- SEM) of the distribution of slopes obtained at each value of SD of the gaussian distribution. The mean +1 SD contains the real slope value for all gaussian SDs up to 73 µm.

**Supplementary Fig. 3.** Results from the single cell model, demonstrating that inhibition balances excitation and produces sharp tuning across the orientation map. (a) Tuning of $g_i$ and $g_e$ as a function of the local input OSI together with the data points from the experiments. (b) Tuning of the $V_m$ and spike response as a function of the local input OSI. The $V_m$ OSI increases as the local input OSI becomes larger, i. e. as cell locations move from a pinwheel center to an orientation domain. The OSI of the spike response, however, remains constant. (c) Average OSIs for $g$, $g_i$, $g_e$, $V_m$, and the spike response for pinwheels (OSI<0.3) and orientation domains (OSI>0.7). (d) Tuning of the excitatory conductance ($g_e$, blue curves) and the matched inhibition ($g_i$, red curves) for increasing levels of excitation (dashed and continuous lines). The tuning curve for inhibition must be scaled in order to balance an increase in total excitation, but its shape remains similar. (e) Ratio of the OSIs of $g_i$ and $g_e$ for an orientation domain (thick line) and a pinwheel location (thin line) as a function of the strength of excitation (peak conductance at the preferred orientation for an orientation domain neuron). Spike tuning remains constant
for a broad range of excitatory strengths if it is properly balanced by inhibition. This is shown by the almost constant ratio of OSIs for the conductances.

**Supplementary Fig. 4.** Results from the network model, demonstrating tuned conductances and sharp spike tuning at pinwheels and orientation domains. (a) Tuning of $g_i$ and $g_e$ in a pinwheel neuron population and in an orientation domain population. (b) Tuning of $V_m$ and spike rate in pinwheels and in orientation domains.

**Supplementary Fig. 5.** Results from the network model, demonstrating that balanced recurrent excitation and inhibition are required for location invariant orientation tuning. Effects of two different parameterizations of the recurrent synaptic strength are shown. (a) $V_m$ and spike rate OSIs as a function of map location. (b) $g_e$ and $g_i$ OSIs as a function of map location. The dashed lines show results of a simulation in which recurrent inhibition is particularly strong, and the main synaptic excitatory conductance comes from the feedforward afferents. The solid lines show results of a simulation in which the excitatory contributions from afferent and recurrent connections are almost equal and inhibition does not dominate. Note that spike tuning is invariant with map location in the balanced case but less invariant in the feedforward case. (Solid line: peak afferent excitatory conductance to peak recurrent excitatory conductance was approx. 1.3:1; peak recurrent excitatory conductance to peak recurrent inhibitory conductance was approx. 1:4. Dashed line: peak afferent excitatory conductance to peak recurrent excitatory conductance was approx. 3:1; peak recurrent excitatory conductance to peak recurrent inhibitory conductance was approx. 1:11).
Supplementary References


