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Orientation selectivity in visual cortex by fluctuation-controlled criticality

Louis Tao*, David Cai^{†‡}, David W. McLaughlin^{†‡§}, Michael J. Shelley^{†§}, and Robert Shapley^{†§}

[†]Courant Institute of Mathematical Sciences and [§]Center for Neural Science, New York University, New York, NY 10012; and *Department of Mathematical Sciences, New Jersey Institute of Technology, Newark, NJ 07102

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Within a large-scale neuronal network model of macaque primary visual cortex, we examined how intrinsic dynamic fluctuations in synaptic currents modify the effect of strong recurrent excitation on orientation selectivity. Previously, we showed that, using a strong network inhibition countered by feedforward and recurrent excitation, the cortical model reproduced many observed properties of simple and complex cells. However, that network's complex cells were poorly selective for orientation, and increasing cortical self-excitation led to network instabilities and unrealistically high firing rates. Here, we show that a sparsity of connections in the network produces large, intrinsic fluctuations in the cortico-cortical conductances that can stabilize the network and that there is a critical level of fluctuations (controllable by sparsity) that allows strong cortical gain and the emergence of orientation-selective complex cells. The resultant sparse network also shows near contrast invariance in its selectivity and, in agreement with recent experiments, has extracellular tuning properties that are similar in pinwheel center and iso-orientation regions, whereas intracellular conductances show positional dependencies. Varying the strength of synaptic fluctuations by adjusting the sparsity of network connectivity, we identified a transition between the dynamics of bistability and without bistability. In a network with strong recurrent excitation, this transition is characterized by a near hysteretic behavior and a rapid rise of network firing rates as the synaptic drive or stimulus input is increased. We discuss the connection between this transition and orientation selectivity in our model of primary visual cortex.

hypercolumns | numerical simulation | V1 | circular variance

Orientation selectivity and spatial summation (1) are two fundamental attributes of visual processing performed by the mammalian primary visual cortex (V1). V1 is the first cortical area along the visual pathway where neurons are strongly selective for stimulus orientation. Moreover, measurements of orientation selectivity for individual neurons, such as the tuning curve bandwidth (half-width at half maximum), circular variance (CV),[†] and orientation selectivity index, often show near independence of stimulus contrast. V1 neurons are also classified as either “simple” or “complex” based on their spatial summation properties. Simple cells respond to visual stimulation in an approximately linear fashion, whereas complex cells respond more nonlinearly. For example, when driven by drifting sinusoidal gratings, a simple cell follows the temporal modulation of the drifting grating as the grating moves across the receptive field of the neuron, whereas complex cells show an elevated but unmodulated response. Quantitatively, simple and complex cellular responses are often differentiated by the modulation ratio F1/F0 (at preferred stimulus orientation, the ratio of the first Fourier component and the mean) of the cycle-averaged firing rate (2): cells with F1/F0 > 1/2 are simple and cells with F1/F0 < 1/2 are complex.

How orientation selectivity arises in V1 has not been fully elucidated (3, 4). According to the classical Hubel and Wiesel picture (5), orientation selectivity arises directly from the convergence of lateral geniculate nucleus (LGN) afferents. However, modeling based on the Hubel and Wiesel, or “feedforward,” picture

shows that the degree of selectivity provided by the convergent LGN inputs alone is insufficient to explain extant data (3). Some form of cortical processing seems necessary.

V1 responses have been investigated through a variety of models of differing architectures and coupling schemes. Modifications of the feedforward scheme have used Hebbian ideas to posit cortical circuitry with highly specific cortical inhibition. The push-pull model (6) is an example of such a modification: intracortical inhibition is anticorrelated with the excitatory synaptic drive. Other models without highly feature-specific coupling demonstrate that selectivity can arise from the sharpening of weakly tuned feedforward excitation by broadly tuned intracortical inhibition (see, e.g., refs. 7–9). The so-called marginal phase, which can evince contrast invariance, arises when cortical excitation is sufficiently strong to allow symmetry-breaking states (10).

Previously, we studied how simple and complex cell responses arise in a large-scale neuronal network model of an input layer 4C α of macaque V1 (11–13). The model represents a 1-mm² local patch with four orientation hypercolumns containing O(10⁴) conductance-based, integrate-and-fire (I&F) neurons: 75% excitatory and 25% inhibitory. The cortical architecture, the LGN drive, and the cortico-cortical synaptic couplings are constrained whenever possible by anatomical and physiological measurements. In this large-scale model, a continuum of simple and complex cellular responses arises from the varying trade-offs between cortico-cortical and geniculate excitation: the most simple of the model neurons are driven strongly by the LGN and are “linearized” by strong cortical inhibition (12). In its published form, this model operated in a regime where its membrane conductances and potential had large fluctuations over its mean (see figures 4 and 5 of ref. 13). Indeed, it was these fluctuations that drove the network activity because the trial-averaged membrane potential was below the firing threshold,[‡] as has also been observed experimentally (14). The origin of these fluctuations, which helped stabilize the network responses, was in large part external through the inclusion of membrane conductances (in addition to the cortico-cortical ones) driven by stimulus-

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Abbreviations: CV, circular variance; V1, primary visual cortex; F1, first Fourier component; F0, mean component; LGN, lateral geniculate nucleus; ES, excitatory simple; EC, excitatory complex; PSC, postsynaptic conductance; AMPA, α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid.

[†]To whom correspondence may be addressed. E-mail: cai@cims.nyu.edu or david.mclaughlin@nyu.edu.

[‡]Orientation selectivity for drifting grating stimuli is measured by CV. Let $m(\theta)$ denote the time-averaged firing rate as a function of stimulus angle θ ; $m(\theta)$ is π -periodic. CV is defined as $CV[m] = 1 - \frac{|\int_0^\pi m(\theta) e^{2i\theta} d\theta|}{\int_0^\pi m(\theta) d\theta}$. CV lies between 0 and 1. Poorly tuned cells have CVs near 1, whereas well tuned cells have CVs near 0.

[§]We note that Eq. 1 of the integrate-and-fire dynamics can be cast in the form $\dot{v}_j = -g_j^{-1}(t)[v_j - V_j^*(t)]$ (see *Methods*). The effective reversal potential, V_j^* , is necessarily greater than the voltage threshold V_T whenever the neuron fires. We say the dynamics are mean-driven whenever the trial averaged $\langle V_j^*(t) \rangle > V_T$; i.e., the mean of the synaptic input is sufficient to drive the neuron to fire. The time-average $\bar{V}_j^* \equiv T^{-1} \int_0^T V_j^*(t) dt$ can also be used if the rate of the input is time-homogeneous. For $\langle V_j^* \rangle < V_T$, we say the dynamics are fluctuation-driven, because temporal fluctuations in the drive are needed for spiking.

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in this model. Fig. 3*a* shows the distribution of CV for the ES and the EC populations separately. Both populations are broadly distributed in CV, having ES cells somewhat better tuned than EC cells, in qualitative agreement with experimental data (17). Fig. 3*b* indicates that the orientation selectivity of the populations is approximately contrast invariant: the CV at medium stimulus contrast is on average equal to the CV at low stimulus contrast, with the data showing a large scatter (consistent with the experimental measurements^{§§}). As already discussed, a distinct feature of previous versions (13) of this large-scale model was the clear differences in orientation selectivity relative to cortical location. As seen in Fig. 3*c*, there are differences in selectivity near and far from pinwheel centers, but they are slight. This roughly invariant selectivity in extracellular spiking across the cortical surface is in agreement with experimental measurements (16, 22, 23), as are the differences in tuning of cortical conductances near (broadly tuned) and far (more narrowly tuned) in cortical conductances (16, 23).

In producing these model results, large regions of the synaptic coupling strength parameter space were explored. We find that, to have roughly contrast invariant, selective complex cells, there must be strong recurrent excitation (13), with large intrinsic temporal fluctuations (15). To understand the effect of intrinsic synaptic fluctuations on network dynamics, we systematically varied the network sparsity through varying the connection probability p , thus varying N_{eff} (see *Methods*).

We find that, without sufficient intrinsic synaptic fluctuation, the complex cells tend to be bistable in the presence of the strong recurrent excitation needed for amplification and orientation tuning, with the bistability leading to network properties that are biologically unrealistic, such as only a very few extremely well tuned complex cells with absolute-refractory-limited high firing rate, whereas the rest of the complex cells remain unresponsive. This behavior is illustrated by comparing model networks with differing N_{eff} . Fixing the stimulus orientation, the stimulus contrast was slowly increased from zero to 100% contrast and then decreased back down to zero. For each cell, the number of spikes produced during the period of contrast increase was recorded, as were the number of spikes produced during contrast decrease. Let ΔN_{spikes} denote the difference in the two, so that positive ΔN_{spikes} means that the cell spiked more during contrast decrease than during contrast increase. Fig. 4 shows, for two networks with different N_{eff} , the distributions of ΔN_{spikes} of the ES and the EC populations. For the sparsely coupled, fluctuation-driven network [Fig. 4*a*; $N_{\text{eff}} = 96$ with $N_E = 72$ and $N_I = 24$ (24, 25)], the distribution of ΔN_{spikes} is symmetric about 0, revealing no difference in the system response to contrast increment or decrement. However, in the (mean-driven) network with larger N_{eff} (Fig. 4*b*; $N_{\text{eff}} = 768$ with $N_E = 576$ and $N_I = 192$), the distribution of ΔN_{spikes} for the EC population is skewed; that is, the EC cells are firing more during contrast decrement. This behavior is symptomatic of a hysteretic system. Note that this hysteresis increases as N_{eff} is further increased (data not shown). To isolate and understand the effect of intrinsic fluctuations, we turn next to an idealized, statistically homogeneously coupled model and examine how synaptic fluctuations transform the input–output relation of strongly recurrent networks.

Fluctuation-Controlled Criticality. We now consider a reduced, very idealized model network wherein 50% of the neurons receive feedforward drive (mimicking simple cells), 50% receive strong intracortical excitation (mimicking complex cells), and the coupling is statistically homogeneous (see *Methods*). Both populations receive the same, strong, cortico-cortical inhibition, i.e., $S_{\text{EI}} = S_{\text{II}}$ being relatively large. We focus on the effect of fluctuations by ignoring detailed time dependencies of the visual drive; all simple cells receive the same feedforward drive of mean

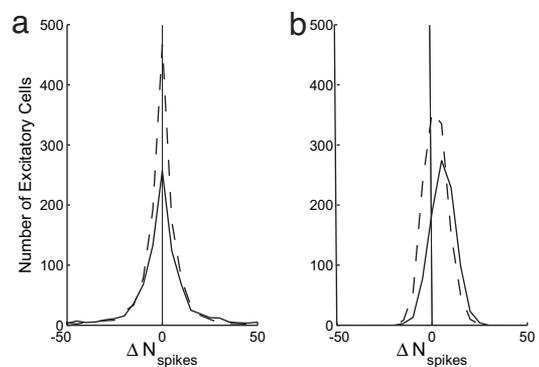


Fig. 4. Hysteresis in the V1 model. Shown is the distribution in ΔN_{spikes} (the difference in the number of spikes during stimulus contrast decrement and increment; see text) for the ES (dashed lines) and EC (solid lines) populations. (a) $N_{\text{eff}} = 96$ ($p = 0.02344$). (b) $N_{\text{eff}} = 768$ ($p = 0.1875$). All other model parameters are the same as in Fig. 1. The complex cells of the densely coupled, $N_{\text{eff}} = 768$ network show evidence of hysteresis in stimulus contrast. The stimulus contrast was increased linearly in time from 0% (at $t = 0$) to 100% ($t = 5$ s) and then decreased linearly in time from 100% to 0% (ending at $t = 10$ s).

$G_{\text{input}} = f\nu_0$. For networks with the same network synaptic coupling strengths but of different N_{eff} (i.e., the average coupling strengths remain the same as N_{eff} is varied), Fig. 5*a* displays the complex cell population firing rate as a function of the mean drive G_{input} . These firing rate curves are obtained by first increasing and then decreasing the feedforward input. In the $N_{\text{eff}} = 200$ network, hysteresis is observed as we ramp up and then down the strength of the feedforward drive. This behavior is well captured by the solution of our kinetic theory analysis (data not shown) (15). The transition is a saddle-node bifurcation in G_{input} for the mean population firing rate. As N_{eff} is decreased (while strengthening individual synapses to keep the effective network drive constant), the region of bistable behavior in G_{input} becomes smaller and smaller, until the bistability disappears completely and a smooth firing rate curve is observed (e.g., the curve for $N_{\text{eff}} = 50$). In particular, at the critical point at which the bistability disappears, the gain in the response curve is the most rapid, as can be seen in Fig. 5*a*.

The transition also occurs when we change the relative contributions of fast and slow excitation. NMDA receptors act on a longer time-scale, and each postsynaptic conductance (PSC) has a smaller temporal variance than a PSC mediated by α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors. Therefore, by beginning with a sparse system and very little NMDA excitation, there are large fluctuations, but these fluctuations diminish and the system becomes more mean-driven, with an increasing proportion of NMDA excitation. Fig. 5*b* displays the complex cell population firing rate as a function of G_{input} in networks of fixed $N_{\text{eff}} = 25$ but with different NMDA/AMPA ratios. Again, the firing rate curves are obtained by increasing and decreasing G_{input} . In the case with no AMPA, hysteresis is observed, and the dynamics is bistable. In the case with no NMDA, there is no hysteresis, and the dynamics is dominated by fluctuations and below the near-critical state. We note that the results in Fig. 5*a* and *b* also hold in densely coupled networks where a probability of synaptic failure introduces an effective sparsification to the network connectivity, and so induces a similar bifurcation structure.^{§§}

^{§§}It is important to note that there can be important differences in network response between the effective sparsification via synaptic failure and that via sparse connections that are fixed and independent of time. For example, synaptic failure induces a much more statistically homogeneous dynamic behavior in a network, whereas the sparse but fixed connections can have clustered dynamics associated with specific causal links over

^{§§}Shapley, R., Johnson, E., Hawken, M. & Kang, K. (2002) *Soc. Neurosci. Abstr.* **32**, 720.8.

Fig. 5b, if synaptic inputs are not sufficiently strong to drive the complex cells up to the higher firing upper-branch.

Our analysis suggests that pharmacological manipulations of the cortical network can change its operating point. For example, a mean-driven, hysteretic state can be moved toward the critical state by decreasing Λ , the relative contributions of slow and fast excitation. Similarly, we can move a nonselective network strongly dominated by fluctuations toward the critical state by increasing Λ . Although the N_{eff} and Λ of the V1 cortical network are not known, our work suggests that the cortical network operates near a fluctuation-controlled critical state.

Methods

We use systems of conductance-based integrate-and-fire neurons, whose individual membrane potentials $v_P^j(t)$ follow

$$\frac{dv_P^j}{dt} = -g_L(v_P^j - V_R) - g_{PE}^j(t)(v_P^j - V_E) - g_{PI}^j(t)(v_P^j - V_I), \quad [1]$$

where $P = E, I$ for the excitatory and inhibitory neurons, $j = 1, \dots, N_P$. The m th spike time, t_m^j , of the j th model neuron, is determined by $v_P^j(t_m^j) = V_T$; $v_P^j(t_m^j + \tau_{\text{ref}}) = V_R$, where τ_{ref} is an absolute refractory period. Here, the membrane potentials of the excitatory (E) [inhibitory (I)] neurons are denoted by $v_E^j(t)$ [$v_I^j(t)$], where the superscript j indexes the spatial location of the neuron within the network. g_L , g_{PE} , and g_{PI} are the leak, excitatory, and inhibitory conductances, respectively. We use normalized, dimensionless potentials with $V_I = -2/3$, $V_T = 1$, $V_R = 0$, and $V_E = 14/3$ (11). We take $\tau_{\text{ref}} = 3$ ms (1 ms) for excitatory (inhibitory) neurons. Eq. 1 can be rewritten as $dv_P^j/dt = -g_{PT}^j(t)[v_P^j - V_S^j(t)]$, where $g_{PT}^j \equiv g_L + g_{PE}^j + g_{PI}^j$ is the total membrane conductance and $V_S^j \equiv (g_L V_R + g_{PE}^j V_E + g_{PI}^j V_I)/g_{PT}^j$ is an effective reversal potential.

The time-dependent conductances arise from the LGN input and from the cortical network activity of the excitatory and inhibitory populations, and have the general form

$$g_{PE}^j(t) = F_{PE}(t) + [(1 - \lambda^j)S_{PE} + S_{PE}^0] \cdot \sum_k a_{j,k} (p_k^j/p) \sum_l G_E(t - t_l^k), \quad [2]$$

$$g_{PI}^j(t) = F_{PI}(t) + S_{PI} \sum_k b_{j,k} (p_k^j/p) \sum_l G_I(t - t_l^k), \quad [3]$$

where $F_{PE}(t) = \lambda^j g_{lgn}^j(t)$ [the conductance $g_{lgn}^j(t)$ denotes the feedforward forcing from the LGN (see the supporting text of ref. 13 for details)] and $F_{PI}(t) = c_{\text{inh}} \sum_i G_I(t - s_i^j)$ is a stimulus-independent inhibition driven by homogeneous Poisson spike trains. The PSCs have the form $G(t) = \Theta(t)[\exp(-t/\tau_d) - \exp(-t/\tau_r)]/(\tau_d - \tau_r)$, where Θ is the Heaviside function (G is normalized to have unit time integral). The time constants are $\tau_r = 1, 2$, and 1 ms and $\tau_d = 5, 80$, and 10 ms for excitatory AMPA and NMDA and

inhibitory GABA_A, respectively. (Note that orientation-tuning properties in our model are similar for $\tau_d = 3$ and 5 ms for AMPA and GABA_A, respectively.) For excitatory synapses, $G_E(t) = (1 - \Lambda)G_{\text{AMPA}}(t) + \Lambda G_{\text{NMDA}}(t)$, where Λ denotes the fractional contribution of NMDA receptors, $\Lambda = 25\%$.

The kernels $a_{j,k}$ and $b_{j,k}$ describe the spatial structure of the cortical coupling and are normalized to have unit sum. The parameter $\lambda^j \in [0, 1]$ in these equations indicates heuristically how the distribution of simple and complex cells is set in our models and characterizes the simple-complex nature of the j th neuron (with $\lambda^j = 0$ the most complex, $\lambda^j = 1$ the most simple; S_{PE}^0 models weak cortical excitatory couplings for simple cells), by setting the strength of LGN drive relative to the strength of the cortico-cortical excitation. The parameter λ^j is distributed uniformly in $[0, 1]$ for our large-scale V1 model.

The factor p_k^j/p controls the degree of sparsity in network connectivity, while simultaneously scaling up the strength of individual connections as connectivity is made more sparse. To wit, p_k^j is chosen to be 1 with probability p and zero otherwise, and is fixed for individual realizations of the network. On average, each neuron is coupled presynaptically to $N_{\text{eff}} = pN$ other neurons. By scaling the strength of a single postsynaptic connection by p , sparser networks have stronger PSCs, with the mean and the variance of a PSC induced in a single cell scaling as $1/N_{\text{eff}}$. As the distributional average of p_k^j/p is one, the parameters S_{PE} and S_{PI} denote overall network synaptic strengths. To examine the effects of synaptic fluctuations, we study networks with the fixed network coupling parameters S_{PP} but differing levels of sparsity. We take $S_{EI} = S_{II}$ so that the cortical inhibition is the same for both excitatory and inhibitory neurons.

The network architecture is a simplified version of the model of ref. 13. The basic cortical architecture, LGN drive, and cortico-cortical couplings are described in detail in refs. 11, 12, and 13. However, unlike these previous works, here we do not model feedback from other layers or extrastriate areas, which was modeled as activity-dependent feedback in ref. 13. We set the synaptic coupling parameters to be fixed constants instead of being Gaussian distributed about a mean. Inhibitory effects are modeled as a sum of local and global contributions: $b_{j,k} = (1/2) \times (b_{j,k}^0 + 1/N_{\text{eff}})$. The first term is the local one with $b_{j,k}^0$ a Gaussian in the distance between neurons j and k and is normalized to unity, and the second is global and scaled with N_{eff} . Unlike our previous model, we do not consider a second, long inhibitory synaptic time course.

For the idealized model, we have $g_{PE}^j(t) = \eta^j G_{\text{input,PE}}(t) + S_{PE}^{\sigma} \sum_k (p_k^j/pN) \sum_l G_E(t - t_l^k)$, and $g_{PI}^j(t) = S_{PI} \sum_k (p_k^j/pN) \sum_l G_I(t - t_l^k)$, with $\eta^j = 1$, $\sigma = s$ for $j = 1, 2, \dots, N_P/2$ and $\eta^j = 0$, $\sigma = c$, otherwise. $N_E = 75\%N$, $N_I = 25\%N$, and $N = 1600$ is the total number of neurons in the network. $N_{\text{eff}} = pN$. $G_{\text{input,PE}} = f \sum_i G_E(t - t_i^k)$, where $\{t_i^k\}$ is a Poisson spike train with constant rate ν_0 .

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