

The dynamics of visual responses in the primary visual cortex

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Abstract: There is a transformation in behavior in the visual system of cats and primates, from neurons in the Lateral Geniculate Nucleus (LGN) that are not tuned for orientation to orientation-tuned cells in primary visual cortex (V1). The visual stimuli that excite V1 can be well controlled, and the thalamic inputs to V1 from the LGN have been measured precisely. Much has been learned about basic principles of cortical neurophysiology on account of the intense investigation of the transformation between LGN and V1. Here we present a discussion of different models for visual cortex and orientation selectivity, and then discuss our own experimental findings about the dynamics of orientation selectivity. We consider what these theoretical analyses and experimental results imply about cerebral cortical function. The conclusion is that there is a very important role for intracortical interactions, especially cortico-cortical inhibition, in producing neurons in the visual cortex highly selective for orientation.

Keywords: V1 cortex; orientation selectivity; computational model; untuned suppression; tuned suppression; dynamics

Introduction

Orientation tuning, as an emergent property in visual cortex, must be an important clue to how the cortex works and why it is built the way it is. There is a transformation in behavior, from neurons in the Lateral Geniculate Nucleus (LGN) that are not tuned for orientation to orientation-tuned cells in V1 cortex (for example, in cat area 17, Hubel and Wiesel, 1962; in monkey V1, Hubel and Wiesel, 1968; Schiller et al., 1976; De Valois et al., 1982). We have learned about basic principles of cortical neurophysiology from the intense investigation and constructive disagreements about the mechanisms of the orientation transformation

between LGN and V1 as discussed below. Here we will present our own findings about the dynamics of orientation selectivity, and contrast our results and conclusions with others. Our results suggest that intracortical interactions, especially cortico-cortical inhibition, play an important role in producing highly selective neurons in the cortex.

Theories of orientation selectivity

The rationale of our experiments came from considering different models or theories for visual cortical function, so it makes sense to begin with theory. There are two poles of thought about theoretical solutions for the problem of orientation selectivity: feedforward filtering on the one hand, and attractor states where networks develop

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“bumps of activity” in the orientation domain as a response to weakly oriented input on the other (Ben-Yishai et al., 1995). Our own view based on our experimental work, and also on recent theoretical work (Troyer et al., 1998; Chance et al., 1999; McLaughlin et al., 2000; Tao et al., 2004; Marino et al., 2005) is that the major cause of orientation selectivity in V1 is recurrent network filtering. We believe that feedforward excitation induces an orientation preference in V1 neurons but that cortico-cortical inhibitory interactions within the V1 network are needed to make V1 neurons highly selective for orientation.

Feedforward model of orientation selectivity

The first model offered chronologically, and first discussed here, is the feedforward model that is descended from the pioneering work of Hubel and Wiesel (1962). The HW model has the great virtue of being explicit and calculable. It involves the addition of signals from LGN cells that are aligned in a row along the long axis of the receptive field of the orientation-selective neuron, as in Fig. 1. Such connectivity is likely the basis of orientation preference (the preferred orientation) but whether or not feedforward connectivity can account for orientation selectivity (how much bigger the preferred response is than responses to nonpreferred orientations) is a more difficult question. There is some support for a feedforward neural architecture based on studies that have determined the

pattern of LGN input to V1 cells. In the ferret visual cortex Chapman et al. (1991) inhibited cortical activity with Muscimol, a GABA agonist, and observed the spatial pattern of LGN inputs to a small zone of V1. Reid and Alonso (1995) did dual recordings in LGN and cat V1 and mapped the overlapping receptive fields of cortical cells and their LGN inputs. The experiment on cooling of cat V1 to block cortical activity by Ferster et al. (1996) is somewhat analogous to the Chapman et al. (1991) study with the technical improvement of intracellular recording of synaptic current in V1 cells; it was interpreted to mean that there is substantial orientation tuning of the collective thalamic input to a cortical neuron, consistent with the HW feedforward model. In spite of all this evidence, there is general agreement that the HW model predicts rather weak orientation selectivity, and therefore does not account for the visual properties of those V1 cells that are highly selective (Sompolinsky and Shapley, 1997; Troyer et al., 1998; McLaughlin et al., 2000).

The reason for the shortfall of orientation selectivity in the HW model has been discussed before. LGN cells have a low spontaneous rate but are quite responsive to visual stimuli. An LGN cell's firing rate during visual stimulation by an optimal grating pattern has a sharp peak at one temporal phase and dips to zero spikes/s at the opposite temporal phase. Such nonlinear behavior depends on stimulus contrast; at very low stimulus contrast the LGN cells' minimum firing rate may not go down as low as zero spikes/s. But at most

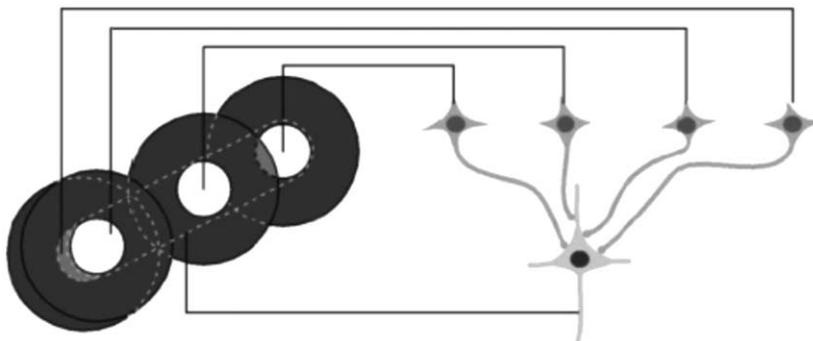


Fig. 1. Classic feedforward model from LGN to simple cells in V1 cortex. Adapted with permission from Hubel and Wiesel (1962). Four LGN cells are drawn as converging onto a single V1 cell. The circular LGN receptive fields aligned in a row on the left side of the diagram make the receptive field of the cortical cell elongated.

stimulus contrasts used in experiments on cortex (that is contrast >0.1) the LGN cells' firing rate will hit zero on the downswing. This clipping of the spike rate at zero spikes/s makes the LGN cells act like nonlinear excitatory subunits as inputs to their cortical targets (Palmer and Davis, 1981; Tolhurst and Dean, 1990; Shapley, 1994). Since the HW model simply adds up the LGN sources, its summation of the clipped LGN inputs results in a nonzero response at 90° from the optimal orientation. Computational simulations of feedforward models with estimates of LGN convergent input derived from the work of Reid and Alonso (1995) support this analysis (Sompolinsky and Shapley, 1997; McLaughlin et al., 2000). An example is given in Fig. 2, which shows a computation of the summed excitatory synaptic input from an HW model onto a cortical cell (cf. Sompolinsky and Shapley, 1997). Such a model produces a substantial LGN input to a cortical cell at 90° from the preferred orientation, as seen in the figure. However, highly selective V1 cells respond little or not at all at 90° from peak orientation. Therefore, feedforward convergence can be only a part of the story of cortical orientation selectivity.

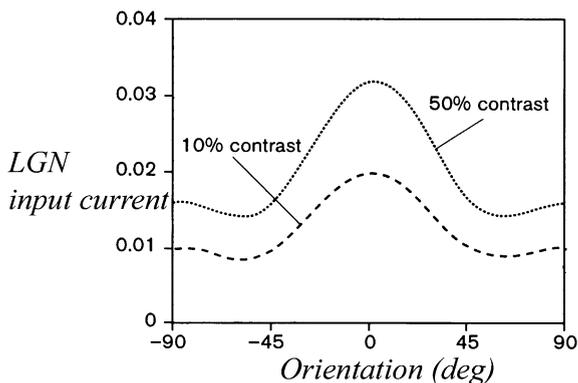


Fig. 2. Orientation tuning curve of the synaptic current evoked by the LGN input to a cortical cell, relative to spontaneous levels of LGN input calculated from a feedforward model (Sompolinsky and Shapley, 1997). In this model, the LGN afferents formed an ON-OFF-ON receptive field. Each subregion had an aspect ratio of 2. A total of 24 OFF-center cells comprised the OFF subfield, while 12 ON cells comprised each ON subregion, in the model. The pattern of wiring was based on the experimental results of Reid and Alonso (1995).

It might be supposed that one could rescue the feedforward model by setting the spike threshold just high enough that the off-peak LGN input would be sub-threshold (Carandini and Ferster, 2000). However, this strategy will only work for one contrast. One can infer this from Fig. 2. If one adds a threshold that makes the 10% contrast curve highly selective, the 50% contrast curve will have a very broadly tuned response. This has been pointed out often before (cf. Ben-Yishai et al., 1995; Sompolinsky and Shapley, 1997; Troyer et al., 1998). To understand cortical orientation selectivity we must answer the theoretical question: how does V1 reduce large feedforward responses at orientations far from the preferred orientation, like those illustrated in Fig. 2? The important experimental issue therefore is, what is the global shape of the orientation tuning curve? This focuses attention on global measures of orientation selectivity like circular variance (Ringach et al., 2002) or 1 minus circular variance, sometimes called the orientation selectivity index (Dragoi et al., 2000). Kang et al. (2004) showed that global measures like circular variance or orientation selectivity index are equivalent to informational measures of discriminability of widely separated orientations, an important function for visual perception.

Models with cortical inhibition and excitation

There is a well-known addition to the HW model that would increase the orientation selectivity greatly. One can obtain increased orientation selectivity by adding *inhibition* that is more broadly tuned for orientation than excitation. The inhibition can be either spatial-phase-specific, so-called push-pull inhibition (Palmer and Davis, 1981; Ferster, 1988, 1992; Tolhurst and Dean, 1990; Troyer et al., 1998), or some other kind of cross-orientation inhibition (Bonds, 1989; Ben-Yishai et al., 1995; Somers et al., 1995; McLaughlin et al., 2000). What matters for explaining orientation selectivity is not the phase specificity of the inhibition but the breadth of tuning. Thalamo-cortical synapses are thought to be purely excitatory (Freund et al., 1989; Callaway, 1998), so the inhibition must come through cortical interneurons

rather than directly from the thalamic afferents. Experiments about intracortical inhibition in V1 have given mixed results. Initially, Sillito's (1975) and Sillito et al. (1980) experiments with bicuculline, a GABA antagonist, suggested that intracortical inhibition is necessary for orientation tuning. However, the interpretation of these results is moot because of possible ceiling effects. Subsequent experiments of Nelson et al. (1994) blocking inhibition intracellularly were interpreted to mean that inhibition onto a single neuron is not necessary for that neuron to be orientation tuned. There is some question about this interpretation because in the Nelson experiments the blocked cells were hyperpolarized, mimicking the effect of sustained inhibition. Somewhat later, an important role for intracortical inhibition was indicated by pharmacological experiments (Allison et al., 1995; Sato et al., 1996; Crook et al., 1998).

There are several models that explain cortical orientation selectivity in terms of broadly tuned inhibition and more narrowly tuned excitation. One such theory of orientation tuning in cat cortex (Troyer et al., 1998) explains orientation selectivity in V1 in terms of "push-pull," that is spatial-phase-specific, inhibition (Palmer and Davis, 1981; Ferster, 1988, 1992; Tolhurst and Dean, 1990). However, the phase specificity is not the main reason the Troyer et al. model generates orientation selectivity. The mechanism for sharpening of orientation tuning in the Troyer et al. (1998) model is cortico-cortical inhibition that is broadly tuned for orientation. In the Troyer et al. model there is broadly tuned LGN convergent excitation as in the HW model, and then more broadly tuned inhibition that cancels out the wide angle responses but that leaves the tuning curve around the peak orientation relatively unchanged. In having broadly tuned inhibition and more narrowly tuned excitation, this particular model resembles many other cortico-cortical interaction models for orientation selectivity (Somers et al., 1995; Ben-Yishai et al., 1995; McLaughlin et al., 2000).

More recently, our colleagues David McLaughlin and Michael Shelley and their colleagues (McLaughlin et al., 2000; Wielaard et al., 2001; Shelley et al., 2002) designed a realistic network model for macaque V1. They constructed a

large-scale model (16,000 neurons) of four hypercolumns in layer 4c α of macaque V1 incorporating known facts about the physiology and anatomy. This model accounts for many visual properties of V1 neurons, among them orientation selectivity. One innovation in this model is its realism: the spatial strength of connections between neurons is taken to be the spatial density of synaptic connections revealed by anatomical investigations of cortex (e.g., Lund, 1988; Callaway, 1998). This model causes significant sharpening of orientation selectivity of V1 neurons compared to their feedforward LGN input. The mechanism of sharpening of orientation tuning is, as in the Troyer et al. (1998) model, broadly tuned inhibition. The big difference between this model and that of Troyer et al. (1998) is that in the McLaughlin et al. model the inhibitory conductance input to a cell is phase-insensitive (and not push-pull). This is a consequence of the realistic simulation of cortical anatomy: because inhibition onto a model cell is a sum from many inhibitory neurons and each cortical inhibitory cell has a fixed phase preference that is different from that of other inhibitory neurons. This view of the nonselective nature of local cortico-cortical inhibitory interactions is supported by the measured phase insensitivity of synaptic inhibitory conductance in V1 neurons (Borg-Graham et al., 1998; Anderson et al., 2000, discussed in Wielaard et al., 2001). Another distinguishing feature of the large-scale model of McLaughlin et al. (2000) is that it provides a mechanism for diversity in orientation selectivity that has been observed (Ringach et al., 2002).

Others have suggested that cortico-cortical excitatory interactions play a crucial role in orientation selectivity. Somers et al. (1995) presented an elaborate computational model for orientation tuning that includes both recurrent cortical excitation and inhibition as crucial elements. Douglas et al. (1995) argued for the importance of recurrent excitation in cortical circuits, reinforcing the message of Douglas and Martin (1991) on the "canonical microcircuit" of V1 cortex. A third paper in this genre was Ben-Yishai et al. (1995). Ben-Yishai et al. offered an analytical model from which they make several qualitative and quantitative predictions. One of their theoretical results is

that if recurrent feedback is strong enough, one will observe a “marginal phase” state in which V1 behaves like a set of attractors for orientation. The attractor states in recurrent excitatory models are discussed not only in Ben-Yishai et al. (1995), but also in Tsodyks et al. (1999). The concept is that the tuning of very weakly orientation-tuned feed-forward signals can be massively sharpened by strong recurrent excitatory feedback. In such a network, the neurons will respond to any visual signal by relaxing into a state of activity governed by the pattern of cortico-cortical feedback. A similar idea was proposed in Adorjan et al. (1999). Our motivation was to try to decide between the different cortical models by performing and analyzing experiments on cortical orientation dynamics.

Cortical orientation dynamics

In an attempt to provide data to test models of orientation selectivity, we used a reverse correlation method developed originally by Dario Ringach. The idea was to measure the time evolution of orientation selectivity extracellularly in single V1 neurons, with a technique that drove most cortical neurons above threshold. The technique is illustrated in Fig. 3. The input image sequence is a stimulus “movie” that runs for 15–30 min. Grating patterns of orientations drawn randomly from a set of equally spaced orientations around the clock (usually in 10^0 steps) are presented for a fixed time (17 ms = 1 frame at a 60 Hz refresh rate in the early experiments reported in Ringach et al., 1997, and 20 ms = 2 frames at 100 Hz refresh rate in the more recent experiments reported in Ringach et al., 2003; Xing et al., 2005). Each orientation is presented at eight spatial phases and the response is phase averaged. For each fixed time interval between a spike and a preceding stimulus, the probability distribution for orientation is calculated by incrementing the orientation bin corresponding to the orientation that precedes each of the N spikes, and then dividing the bin counts by N . N is usually of the order of 5000 spikes. This is done for each value of time interval between spike and stimulus to create a sequence of orientation

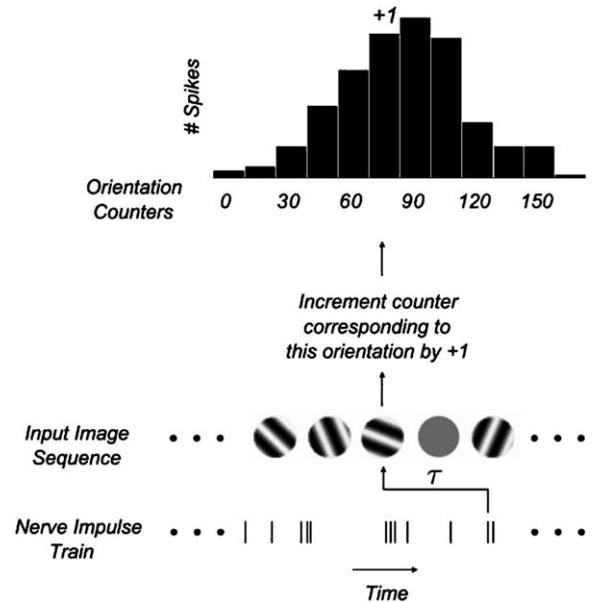


Fig. 3. Reverse correlation in the orientation domain. The input image sequence runs for 15–30 min. Grating patterns of orientations drawn randomly from a set of equally spaced orientations in the interval $[0^\circ, 180^\circ]$ (usually in 10° angle steps) are presented for 20 ms each (2 frames at 100 Hz frame rate). Each orientation is presented at eight spatial phases; response is phase averaged. For each time offset, the probability distribution for orientation is calculated by incrementing the orientation bin corresponding to the orientation that precedes each of the N spikes, and then dividing the bin counts by N . N is usually of the order of 5000 spikes. This is done for each time offset τ to create an “orientation selectivity movie.” In these experiments an additional pattern is added — a blank stimulus at the mean luminance of the grating patterns. This allows us to create a baseline with which the responses at different angles can be compared. Adapted with permission from Shapley et al. (2003).

tuning curves, one for each time interval — an “orientation selectivity movie.”

In more recent experiments on orientation dynamics (Ringach et al., 2003; Xing et al., 2005), we used a refined technique that allowed us to uncover the mechanisms of orientation selectivity. As shown in Fig. 3, an additional pattern is added to the sequence — a blank stimulus at the mean luminance of the grating patterns. This allows us for the first time to measure untuned excitation and inhibition because, with this new technique, one can estimate whether the effect of one of the oriented patterns is greater or less than that of the blank pattern. If the probability of producing a

spike by a pattern of orientation θ is greater than that of a blank, we view as evidence that a pattern of orientation θ produces net excitation, while if the probability of producing a spike by a pattern of orientation θ is less than that of a blank, we take this as an indication of inhibition. Specifically, we take $R(\theta, \tau) = \log[p(\theta, \tau)/p(\text{Blank}, \tau)]$. If the probability that angle θ evokes a spike is greater than that of a blank screen, then the sign of R is $+$. If the probability that angle θ evokes a spike is less than that of a blank screen, then the sign of R is $-$. If all angles evoke a response above the response to a blank, then $R(\theta)$ will have a positive value for all θ . A visual neuron equally well excited by stimuli of all orientation angles would produce a constant, positive $R(\theta)$.

The shape of the orientation tuning curve $R(\theta, \tau)$ changes with time, τ , and this dynamic behavior has a number of important properties that are revealed in Fig. 4 for a representative V1 neuron. The black curve is a graph of $R(\theta, \tau)$ at the time offset τ_{peak} when the orientation modulation depth, that is the difference between R_{max} and R_{min} , reaches its maximum value. The red and blue

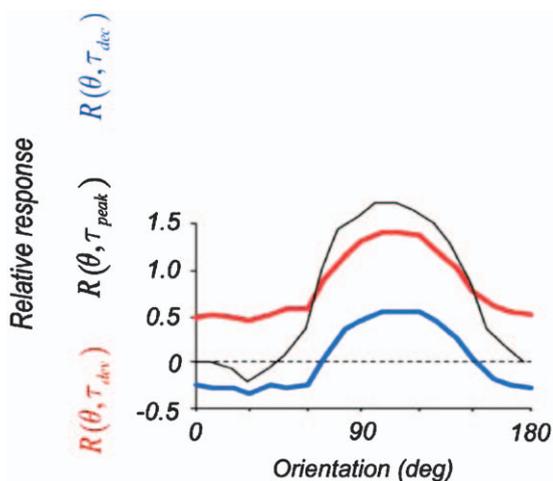


Fig. 4. Dynamics of orientation tuning in a representative V1 neuron. The black curve is a graph of $R(\theta, \tau)$ at the time offset τ_{peak} when the orientation modulation depth reaches its maximum value. The red and blue curves are graphs of $R(\theta, \tau)$ at the two times before and after τ_{peak} at which orientation modulation is half maximal: the red curve is at τ_{dev} , the earlier of the two times, and the blue curve is for τ_{dec} , the later time. Adapted with permission from Shapley et al. (2003).

curves are graphs of $R(\theta, \tau)$ at the two times bracketing τ_{peak} at which the orientation modulation depth is half the maximum value; the red curve is at the development time τ_{dev} , the earlier of the two times when the modulation depth first rises from zero to half maximum, and the blue curve is at the declining time τ_{dec} when the response has declined back down to half maximum from maximum. One striking feature of these curves is that the dynamic tuning curve at the earlier time, $R(\theta, \tau_{\text{dev}})$, has a large positive pedestal of response, a sign of untuned or very broadly tuned excitation early in the response. This is just what one might predict from the analysis of feedforward models (see Fig. 2), if indeed the earliest response measurable were predominantly feedforward excitation. But then, as the response evolves in time, the maximum value of $R(\theta, \tau)$ at the preferred orientation grows only a little, while the responses at nonpreferred orientations decline substantially. Thus, Fig. 4 demonstrates that the maximum orientation modulation depth occurs at a time when inhibition has suppressed nonpreferred responses. Because such inhibition suppresses all responses far from the preferred orientation, we infer that this is untuned inhibition. It is also reasonable to infer that tuned excitation near the preferred orientation counteracts the untuned inhibition to maintain the peak value of $R(\theta, \tau)$.

While bandwidth often has been the focus of interest in previous research, it is rather the global shape of the tuning curve at all orientations that differentiates between different theoretical mechanisms. One simple way to study the global shape of the tuning curve is to compare the response at the preferred orientation with the response at orthogonal-to-preferred. Therefore, we studied $R(\theta_{\text{pref}}, \tau)$ and $R(\theta_{\text{ortho}}, \tau)$ in a population of V1 neurons because these features of the dynamical tuning curves are related to the overall shape of the tuning curve and lead to insight about the role of inhibition in the time evolution of orientation selectivity. The average behaviors of $R(\theta_{\text{pref}}, \tau)$, $R(\theta_{\text{ortho}}, \tau)$ averaged over a population of 101 neurons are depicted in Fig. 5. An important feature is the positive sign of $R(\theta_{\text{pref}}, \tau)$ and $R(\theta_{\text{ortho}}, \tau)$ early in the response, indicating that, on average, V1 cells tended to respond to *all*

orientations early in the response. This is a feature that is consistent with the idea that at early times feedforward input as in Fig. 2 controls the response. Another important feature of the data

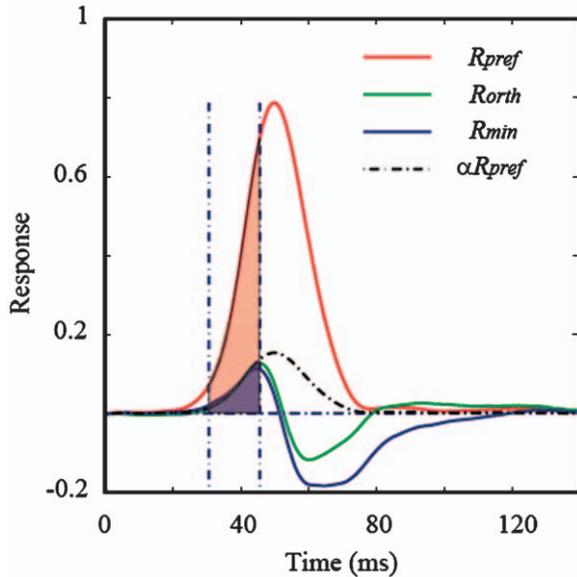


Fig. 5. Time course of the population-averaged (101 cells) response to preferred orientation (R_{pref} , red curve), to orthogonal orientation (R_{orth} , green curve) and to the orientation where the response was minimum (R_{min} , blue curve) in responses to stimuli of large size. Black dash-dot curve (αR_{pref}) is the rescaled R_{pref} . The time course of each cell's responses was shifted so that its half-max rise time of R_{pref} is at 41 ms. Adapted with permission from Xing et al. (2005).

is that the time course of $R(\theta_{ortho}, \tau)$ was different from $R(\theta_{pref}, \tau)$. Note especially in the time courses in Fig. 5 the downward turn of $R(\theta_{ortho}, \tau)$ just before $R(\theta_{pref}, \tau)$ reached its peak value. Eventually $R(\theta_{ortho}, \tau)$ declined to negative values meaning that later in the response orientations far from the preferred orientation were suppressive not excitatory. If the entire response were dominated by feedforward input, one would expect that preferred and orthogonal responses would have the same time course simply scaled by the relative sensitivity. Therefore, the results in Fig. 5 qualitatively rule out an explanation of the time evolution of orientation selectivity in terms of feedforward inputs alone.

The results about the population averages in Fig. 5 support the hypothesis that there is untuned suppression generated in the cortex that is rapid, but still somewhat delayed with respect to the early excitatory input. The untuned suppression contributes to the amount of orientation selectivity at the time when the neuron is most selective. These results could be explained with a theory in which feedforward excitation drives the early weakly selective response. Evidence in favor of weakly selective excitation was obtained by Xing et al. (2005) when they analyzed the orientation dynamics into a sum of excitation and untuned and tuned suppression. The orientation tuning of the excitatory term is shown in Fig. 6 where it is compared to the predicted broad tuning curve for

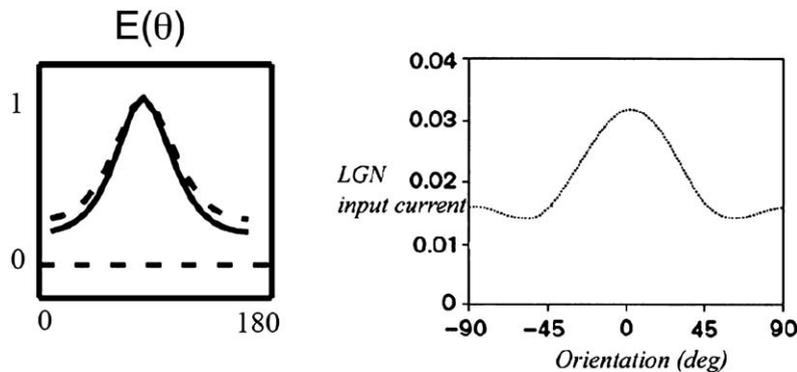


Fig. 6. The time course of measured excitation compared with the prediction of feedforward LGN input current. The orientation dependence of tuned excitation is plotted in the left hand panel, redrawn from Xing et al. (2005). Note especially the broad tuning with nonzero response at orthogonal-to-preferred. The dashed curve is for responses to stimulus of optimal size; the solid curve is for a large stimulus of $2-4 \times$ the diameter of an optimal stimulus. The right panel reproduces the theoretical prediction of Fig. 2.

feedforward input, from Fig. 2. Sharpening of this broadly tuned input occurs when, with a very short delay, relatively rapid intracortical inhibition reduces the response at all orientations, acting like an untuned suppression. Data from intracellular recording in V1 indicate that a wide variety of patterns of cortico-cortical inhibition may influence orientation selectivity (Monier et al., 2003).

Discussion: inhibition and selectivity

The data in Figs. 4 and 5 from the orientation dynamics experiments demonstrate that early excitation in V1 is very broadly tuned for orientation, just as predicted for models of feedforward convergence like the HW model (see Fig. 2). Indeed in simulations of the dynamics experiments with a large-scale network model of V1, McLaughlin et al. demonstrated that feedforward excitation generates dynamical orientation tuning curves with very high circular variance, meaning poor selectivity, at all time offsets between stimulus and spike (see McLaughlin et al., 2000, Fig. 2). Therefore, to us, an important question about orientation selectivity in V1 is, as we have stated it above, how does the cortex suppress the feedforward excitation far from the preferred orientation? Our experimental results show that untuned inhibition in the cortex answers the question for those V1 neurons that are highly selective for orientation. The inhibitory signals must be fairly rapid, though not quite as fast in arrival at the V1 neuron as the earliest excitatory signals. Also, inhibition appears to persist longer than excitation, as illustrated in Fig. 5. A more comprehensive and detailed analysis of the dynamics of orientation selectivity, and in particular of untuned suppression, can be found in Xing et al. (2005).

Additional compelling evidence for the important role of inhibition in orientation selectivity has come from experiments on intracellular recording from neurons in cat V1 (Borg-Graham et al., 1998; Monier et al., 2003). Furthermore, the very elegant pharmacological experiments in macaque V1 cortex by Sato et al. (1996) established that when cortical inhibition was weakened by pharmacological competitive inhibitors, neuronal orientation

selectivity was reduced because the response to off-peak orientations grew stronger relative to the peak response (cf. especially Fig. 8 in Sato et al., 1996). This is further support for the idea that the feedforward excitatory input is very broadly tuned in orientation, and that cortical inhibition suppresses the responses far from the preferred orientation. As presented earlier, the importance of broadly tuned cortical inhibition has been suggested also in computational models of the cortex (Troyer et al., 1998; McLaughlin et al., 2000; Wielaard et al., 2001).

Untuned suppression and cortical inhibition

To judge whether or not cortico-cortical inhibition is the source of untuned suppression requires more detailed considerations. When we stimulated a cell with a stimulus of optimal size (0.45° radius on average in our data), we most likely activated a compact region of V1 (Van Essen et al., 1984; Tootell et al., 1988). This region in V1 cortex corresponds to the cell's local neighborhood (Angelucci et al., 2002). That we see a strong untuned suppression even with a stimulus of optimal size suggests that the untuned suppression mainly comes from the center mechanism and the local circuitry within a cortical hypercolumn. This is consistent with the recent anatomical findings (Angelucci et al., 2002; Marino et al., 2005) that a V1 cell gets most of its inhibitory synaptic input from a local area in the cortex of approximate diameter of 100–250 μm . Untuned suppression exists in all layers as well as in simple and complex cell groups (Xing et al., 2005). This suggests that untuned suppression is a general mechanism in primary visual cortex (Ringach et al., 2002; Shapley et al., 2003; Xing et al., 2005). Broadly tuned cortico-cortical inhibition that arises locally in the cortical circuitry is the likely source of the untuned suppression we have measured (Troyer et al., 1998; McLaughlin et al., 2000; Tao et al., 2004). There are other candidate mechanisms for untuned suppression in V1, for instance synaptic depression at the thalamo-cortical synapses, as proposed by Carandini et al. (2002). The fact that untuned suppression is stronger in layer 4B and

layer 5 than in the main thalamo-recipient layers (layer 4C and layer 6) suggests that the untuned suppression is mainly from cortico-cortical effects instead of from thalamic-cortical effects (Xing et al., 2005). Furthermore, the untuned suppression we measured had short persistence (Xing et al., 2005), while rapid synaptic depression has 200–600 ms recovery time (Abbott et al., 1997). So the time course of untuned suppression is unlike what has been assumed for synaptic depression (e.g., Carandini et al., 2002). A likely possibility is that fast cortical inhibition is the source of the untuned suppression.

Cortico-cortical excitation and selectivity

There is a possibility that tuned cortico-cortical excitation may contribute also to enhancement of orientation selectivity by boosting the response only around the preferred orientation. The possibility that cortico-cortical excitation could enhance orientation selectivity was suggested previously in theories of V1 (Ben-Yishai et al., 1995; Somers et al., 1995). However, we did not observe a substantial sharpening of the excitatory input during the time evolution of orientation selectivity (Xing et al., 2005). Therefore, the orientation dynamics data suggest that the role of tuned cortical excitation is less than that of untuned inhibition in generating selectivity in V1.

Comparison with other studies

In the Introduction we reviewed previous experiments that were taken to support a completely different point of view, namely that the pattern of feedforward thalamic input is enough to determine orientation selectivity. Our results as a whole are not consistent with this viewpoint. There are in the literature two studies with dynamical stimuli that have been interpreted as supporting the feedforward theory. Gillespie et al. (2001), recording intracellularly in cat V1, reported that the bandwidth of orientation tuning curves did not change with time in their dynamic experiments. As stated above, we think that examining bandwidth misses the point that the crucial question in orientation

selectivity is how the orthogonal response is suppressed by the cortex. Interestingly, Gillespie et al. (2001, Figs. 2h, and 3b, f, j) do report a change in the intracellular baseline with time that reinforces our observations on the dynamic growth of inhibition. Therefore, our interpretation of the results of Gillespie et al. (2001) is that they support the concept that inhibition plays an important role in enhancing orientation selectivity, by untuned inhibition.

In a study that purports to assign a dominant role to feedforward connections in orientation, Mazer et al. (2002) recorded extracellularly in V1 of awake macaques, and used a reverse correlation technique very similar to the one we introduced in 1997 (Ringach et al., 1997). However, unlike the results we have presented here, Mazer et al.'s results were interpreted to indicate that the orientation tuning curves measured dynamically did not change shape with time. Because they did not have a baseline stimulus, as we did with the blank stimuli in the stimulus sequence, Mazer et al. (2002) could not measure the presence of untuned suppression, or broadly tuned excitation either. Therefore, their conclusions about the time course of orientation dynamics were not well supported by the data they had available.

Diversity

The diversity of orientation selectivity is interesting. Others have also reported data that indicate wide diversity of orientation tuning in cat V1 (Dragoi et al., 2000) and in ferret V1 (Chapman and Stryker, 1993) when the orientation tuning curves were analyzed with global measures of selectivity like those we have employed. There is a need for understanding what are the functional consequences for visual perception of the wide diversity of orientation tuning that is observed. This question was considered by Kang et al. (2004) in a paper that applied a new technique for measuring information transmission by populations of neurons. Kang et al. concluded that diversity of orientation selectivity could make the cortical population better at discriminations of different orientation differences. It is also plausible that the

visual cortex is not only designed for tasks like orientation discrimination, and that diversity of orientation selectivity may be a result of specializations of neurons in other stimulus dimensions besides orientation.

Orientation selectivity and cortical circuits

Our view of V1 is that it is a nonlinear dynamical system and one of its tasks is to find local stimulus features in the neural image of the visual scene relayed to V1 from the eye through the LGN. Different sources of excitation drive the activity in V1 cells: local thalamo-cortical projections, local-circuit cortico-cortical excitation, long-distance horizontal V1 axons, and also feedback. Different sources of intracortical inhibition contribute to the selectivity of V1 neurons: local-circuit inhibition, inhibition mediated by signals from long-distance intrinsic V1 horizontal connections (Gilbert and Wiesel, 1983; Rockland and Lund, 1983; Crook et al., 1998; Roerig and Chen, 2002), and feedback from extra-striate cortex (Angelucci et al., 2002) that drives inhibitory interneurons in the local circuit. While feedforward excitation must play a role in giving V1 cells preferences for particular orientations, intracortical inhibition makes some V1 cells highly selective for their preferred orientation over all others.

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