

New perspectives on the mechanisms for orientation selectivity

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Since the discovery of orientation selectivity by Hubel and Wiesel, the mechanisms responsible for this remarkable operation in the visual cortex have been controversial. Experimental studies over the past year have highlighted the contribution of feedforward thalamo-cortical afferents, as proposed originally by Hubel and Wiesel, but they have also indicated that this contribution alone is insufficient to account for the sharp orientation tuning observed in the visual cortex. Recent advances in understanding the functional architecture of local cortical circuitry have led to new proposals for the involvement of intracortical recurrent excitation and inhibition in orientation selectivity. Establishing how these two mechanisms work together remains an important experimental and theoretical challenge.

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Abbreviations

AC	alternating current
DC	direct current
EPSP	excitatory PSP
GABA	γ -aminobutyric acid
IPSP	inhibitory PSP
LGN	lateral geniculate nucleus
NO	null orientation
PO	preferred orientation
PSP	postsynaptic potential
V1	primary visual cortex

Introduction

The coding of the orientation of visual stimuli is one of the best studied cortical functions. Understanding how orientation tuning emerges in the primary visual cortex (V1) may be a key to understanding how the cerebral cortex is designed to process information. In this review of recent (and classic) work, we highlight the interplay between theory and experiments in an attempt to characterize the mechanisms underlying orientation selectivity.

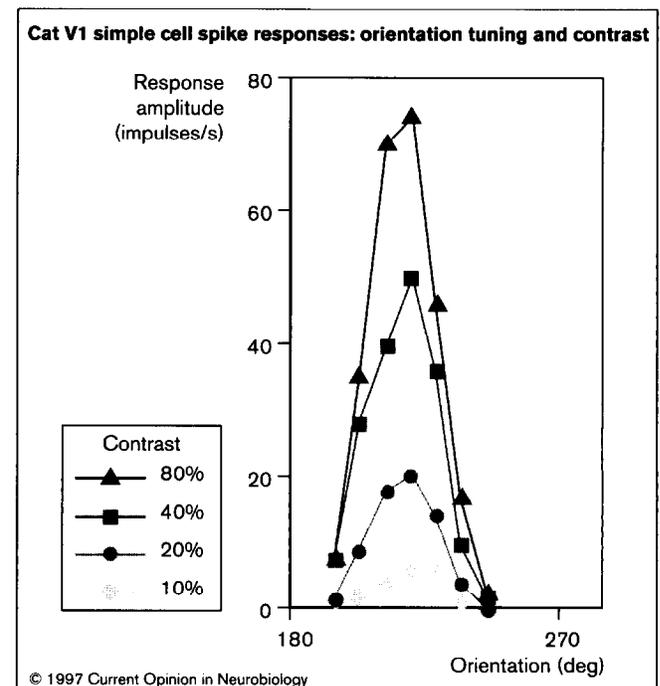
Orientation tuning in visual cortex

One of the crucial events in the coding of visual stimuli by V1 is the transformation of information from more-or-less orientation-insensitive elements to orientation-tuned ones (as measured in the cat [1] and in the monkey [2]). In the retina and lateral geniculate nucleus (LGN; the main visual nucleus in the thalamus), there is weak or

no orientation selectivity [1,3–5]. Therefore, orientation selectivity must result either from the way the thalamic afferents connect to the cortical cells or from the cortico-cortical circuitry.

Figure 1 depicts typical orientation tuning curves of a simple cell in cat V1. The response of the simple cell to a drifting sinusoidal grating that moves across its receptive field is recorded as a function of the stimulus orientation (see e.g. [6]). The response, measured extracellularly as the number of spikes per second emitted by the cell, is maximal at the preferred orientation (PO) of the cell, which is 220° in this case (Figure 1), and falls off sharply as the stimulus orientation departs from the PO. The response decreases to zero at about $\pm 30^\circ$ away from the PO. The half-width of the orientation tuning, which is defined as the half-width at the half-height of the tuning curve, is 15° in this example (Figure 1). Different cells show different degrees of tuning; the mean half-width is about 20° in simple cells of the cat visual cortex [7]. Figure 1 displays another important feature of orientation tuning, namely that the width (but not the height) of the tuning curve is independent of stimulus contrast [6,8].

Figure 1



Orientation tuning and contrast. Discharge response of a simple cell in cat V1 to drifting sinusoidal gratings of optimal spatial frequency. Orientation tuning was measured at four different contrasts (10%, 20%, 40% and 80%), as indicated in the figure. Redrawn from [6].

Orientation selectivity: feedforward or intracortical?

Recent experiments—in particular, the cooling experiment by Ferster *et al.* [9**] and the correlation experiments by Reid and Alonso [5]—have been interpreted [9**,10–12] as providing strong evidence for Hubel and Wiesel's model [1], according to which orientation selectivity results from the specific pattern of convergence of LGN afferents. These findings, therefore, would seem to settle the long-standing issue regarding the mechanisms for orientation selectivity. However, building on advances in our understanding of cortical circuitry, recent theoretical studies by Ben-Yishai *et al.* [13] and Somers *et al.* [14] suggest that orientation selectivity is a cooperative property of local cortical networks. Determining which of these two explanations is correct will have a big impact on our conception of cerebral cortical function. If orientation selectivity results from the specific pattern of convergence of LGN afferents, as proposed by Hubel and Wiesel, this would mean that a fundamental function of the visual cortex arises from the feedforward filtering of sensory input, supporting the view of cortical processing as a hierarchy of feedforward transformations of neural representations. However, if intracortical circuitry plays a crucial role in orientation tuning, then it implies that intracortical dynamics shape the internal representations of the external world in the cortex.

The spatial arrangement of LGN inputs

The first issue in understanding orientation selectivity concerns the organization of the convergent inputs from the LGN to single cortical cells. The receptive fields of both LGN and simple cells in the cortex are characterized by distinct ON and OFF subregions. Cross-correlation analysis of activity in pairs of cells in the cortex and LGN in the cat [5,15] has provided strong evidence that the inputs from the LGN to the different subfields of simple cells are largely segregated to the same 'signature' (i.e. ON to ON and OFF to OFF). In addition, experiments in both cats [5] and ferrets [16] indicate that in many cells there is a high correlation between the axis of alignment of the LGN receptive fields and the PO of the recipient cortical column.

Subfield aspect ratio of LGN afferents

An important quantitative issue is the degree of elongation of the cortical subfield formed by the receptive fields of the LGN afferents. This elongation is quantified by the ratio between the long and short axes of the subfield—the subfield aspect ratio. Estimates based on the geometric dimensions of the simple cell's subfields measured by extracellular responses yield a mean subfield aspect ratio of 4–5 [17]. However, these responses may be influenced by cortical inputs, as well as by the spike threshold; hence, these estimates most probably overestimate the subfield aspect ratio of the combined LGN receptive fields. In fact, the results of Chapman *et al.* [16] are consistent with a low mean aspect ratio. Furthermore, their results highlight the large variability in the degree of alignment of

LGN inputs across different cortical columns, and indicate that this variability is not correlated with the sharpness of orientation tuning in these locations.

Reid and Alonso [5] have attempted to reconstruct the receptive field generated by the LGN afferents to a cortical cell. Their results are, in our opinion, consistent with a subfield aspect ratio of about 2. However, their reconstruction is ambiguous, as it combines data from different simple cell/LGN cell pairs and, in addition, the strength of the LGN afferent connections has not been taken into account (RC Reid, personal communication).

The orientation tuning of the total excitatory postsynaptic potential (EPSP) generated by LGN afferents depends not only on the geometry of their receptive field but also on the spatio-temporal details of the stimulus. In studies using drifting long bars or gratings as the stimulus, the group of LGN cells that are active during the motion of the stimulus across the simple cell's receptive field is the same for all orientations; changing the stimulus orientation changes only the temporal order of their activation. Temporal modulation (the 'AC' component) of the LGN input to the cortical cell may produce a relatively weak signal at the null orientation (NO; the orientation perpendicular to the PO). However, if one assumes that individual LGN cells are untuned, then the time-averaged EPSP (the 'DC' component) will be untuned to orientation [18]. The DC component is particularly large at high contrasts because the dynamic range for increased discharge is much larger than that for suppression of discharge ([18]; A Krukowski, NJ Priebe, KD Miller, *Soc Neurosci Abstr* 1996, 22:642). The peak of the time-dependent LGN response to a drifting pattern, which depends on both the AC and the DC components, will have a substantial positive value at all orientations. In addition, if the stimulus is long, then not only does the subfield aspect ratio contribute to the tuning of the afferent EPSP, but so does the presence of multiple subfields.

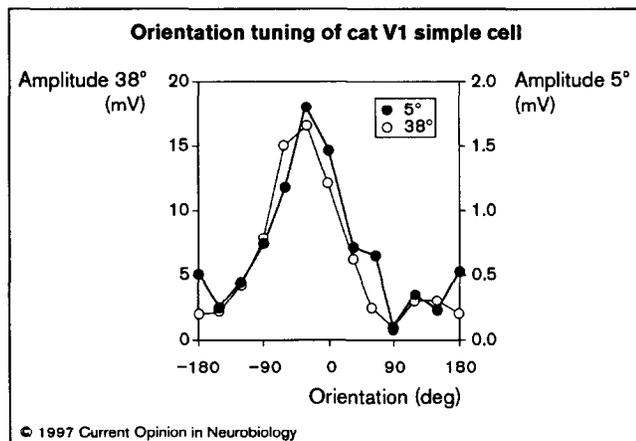
Intracellular measurements

Recently, two groups have used intracellular recordings in an attempt to measure the tuning of the combined LGN input to simple cells in cat visual cortex. Pei *et al.* [19] measured EPSP responses to flashed stationary bars at different latencies. The early components of the EPSPs—which probably represent, primarily, LGN inputs—showed very weak tuning with orientation and yielded an estimated average subfield aspect ratio of only 1.7 [19].

The second group, Ferster and co-workers [9**], attempted to measure directly the tuning of the LGN synaptic input to simple cells in cat V1. To isolate the LGN contribution, they made intracellular recordings of the membrane potentials of simple cells responding to a high-contrast, moving 2 Hz sinusoidal grating while the

cortex was cooled to 10°C or below, which presumably quenched most of the spike activity in the cortex. They compared the recorded membrane potentials to the intracellular responses of the same cells under normal (warm) conditions, in which both LGN and cortical inputs are intact. An example of the results is shown in Figure 2. The postsynaptic potentials (PSPs) in the cooled conditions, presumably dominated by LGN inputs, have a significant orientation dependence. However, it should be noted that in the majority of the cells, the tuning profile of the potential was broad, with a half-width of about 45°, as seen in the example depicted in Figure 2. In many of the cells, the input tuning curve seems to vanish at the NO. This is because the potentials recorded in this experiment were the amplitude of the first harmonic of the response. As discussed above, the actual response (as measured by the peak potential above the background level) includes a DC component that is completely untuned to orientation. Thus, the results from the cooled cortex indicate that the LGN input has a broad profile superimposed on an untuned base line. This finding is consistent with the LGN input being generated by a low aspect ratio.

Figure 2



Intracellular measurement of orientation tuning of the membrane potential of a simple cell in cat V1 in warm and cooled conditions. The shapes of the two tuning curves are similar, although their amplitudes are different. (Note the different right and left vertical scales.) Redrawn from [9**].

Cortical inhibition

The effect of blocking cortical inhibition in a local region of the cortex on the orientation selectivity of simple and complex cells in the cat has been tested by Sillitto's group [20,21] and others [22]; they found a large reduction in the orientation selectivity of the majority of the cells—most simple cells completely lost their orientation tuning. Recently, Pflieger and Bonds [23] have shown that at some concentrations of bicuculline (a type A GABA receptor blocker), an early, orientation-insensitive response component is present in cat complex cells; but the late response component is tuned to orientation. This

finding suggests that both fast inhibition (mediated by type A GABA receptors) and slow inhibition (not affected by bicuculline) play a role in orientation selectivity. The sensitivity of orientation tuning to the reduction of cortical inhibition is a strong indication that this inhibition is involved in the orientation tuning of cortical cells. An apparently contradictory result was reported recently by Nelson *et al.* [24], who blocked GABA receptors on single cortical cells by using intracellular injections of cesium fluoride. They found that the orientation selectivity of these cells was unaffected by the removal of inhibitory inputs. Sato *et al.* [25**] have recently measured the effect of bicuculline blocking of cortical inhibition in monkey V1. They found a marked decrease in orientation tuning of neurons in some output layers (2, 3 and 4B). However, they found no or little effect on the tuning of neurons in the input layers (4C α and 4C β) and in layer 6.

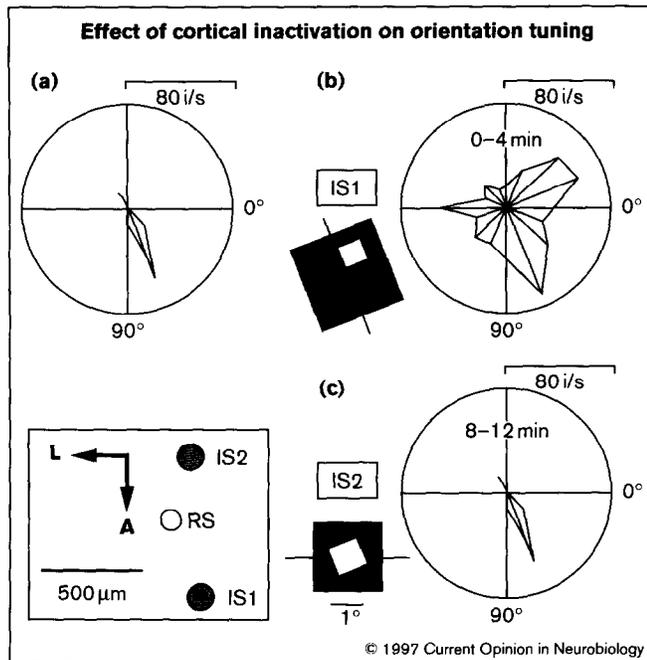
In a recent study, Crook *et al.* [26**] provide new evidence for the existence of cortical inhibition at NO and for its role in orientation selectivity. They blocked cortical activity at distinct orientation columns and measured the effect on the tuning of neurons with similar or different POs located 350–700 microns away in the cortex. They found that the inactivation caused a substantial broadening of orientation tuning in neurons at cross-orientation sites (as illustrated in Figure 3), but had no effect on the tuning at iso-orientation sites.

Intracellular measurements [27,28] have revealed that relatively strong inhibitory PSPs are evoked by a stimulus at the PO. In simple cells, inhibitory postsynaptic potentials (IPSPs) and EPSPs are often generated by antagonistic subfields in a 'push-pull' manner, so that withdrawal of excitation is accompanied by increased inhibition [29]. Surprisingly, these groups found no significant inhibitory inputs (either as hyperpolarizing currents or as shunting conductances) at NO, suggesting that both the EPSPs and the IPSPs of a cortical cell have roughly the same tuning—that is, both peak at the PO and have a very weak amplitude at NO. On the other hand, recent whole-cell recordings [19,30,31] have revealed many cases of inhibitory (as well as excitatory) inputs at NO.

Recurrent cortical excitation

Over the past few years, attention has focused on the role of recurrent cortical excitation in orientation selectivity. Intracellular recordings *in vivo* show that electrical stimulation and visual stimuli evoke multiple EPSP signals that differ in shape and latency. The delayed components come primarily from cortical sources [19,27,28]. The presence of strong excitatory intracortical feedback is supported by anatomical studies that show that the majority of excitatory synapses on spiny stellate cells in cortical layer 4—which are the main targets of LGN inputs to the mammalian visual cortex—are from recurrently connected cortical neurons [32–34]. Estimates of the ratio of the number of intracortical over thalamic excitatory inputs based on

Figure 3



Effect of cortical inactivation on orientation tuning in cat V1. The responses of a single simple cell located at the recording site (RS) to drifting bars at different orientations are shown in polar plots. The plots show the peak responses as a function of the direction of motion that is orthogonal to the bar orientation. The inset shows the recording arrangement. IS1 and IS2 are the remote inactivation sites. Inactivation is caused by microiontophoresis of GABA. The labels A and L indicate the anterior and lateral directions in the cortex, respectively. (a) The control response at RS in the absence of inactivation. (b) The orientation tuning at RS when IS1 is inactivated. The PO of the multiunit activity at IS1 is orthogonal to the cell at RS, as indicated in the diagram. (c) Orientation tuning after inactivation of IS2. The PO of IS2 is 22.5° away from that of RS, as shown in the diagram. The time shown in the polar plots of (a) and (b) indicate the time after the onset of GABA iontophoresis. Adapted from [26**].

anatomy vary between 15 and 4. However, anatomical factors are insufficient to estimate the strength of recurrent cortical excitation, as recent *in vivo* intracellular recordings from spiny stellate cells in cat layer 4 indicate that the amplitude and reliability of thalamo-cortical EPSPs tend to be higher than cortico-cortical EPSPs [35*]. Nevertheless, given their abundance, it is likely that excitatory inputs from adjacent layer 4 cells, as well as from layer 6, constitute the majority of the excitatory inputs to layer 4 cells.

To understand the functional role of the massive cortical excitation in orientation selectivity, it is important to know the columnar architecture of the intracortical circuitry. Unfortunately, not much is known about the orientation specificity of the proximal intracortical connections. Cross-correlation analysis suggests that, on average, the activity of cells with similar POs is more strongly correlated than those with different POs [36]. However, these cross-correlations reflect the degree of common input and

not direct synaptic coupling. Nevertheless, anatomical evidence suggests that the probability of connection between pairs of proximal neurons in the cortex decreases as their separation increases (see [37,38]). Therefore, given the coarse continuity of orientation maps, the probability of connection is expected to decrease as the difference in the PO of the proximal cells increases. Crook *et al.* [26**] found cases in which inactivation at one cortical site greatly reduced the response of neurons with the same PO at a location several hundred microns away. This finding supports the notion of recurrent excitation between cortical neurons with similar POs.

The intracellular measurements of Ferster *et al.* [9**] in the warm conditions reveal that the orientation tuning of the PSP to simple cells has roughly the same shape as the one in the cooled conditions (as shown in Figure 2). This result is remarkable in light of the fact that the total PSP in the warm condition is bigger than the LGN input by a factor of 2–5 (after taking into account the effect of cooling on synaptic efficacy; see the two different vertical scales in Figure 2). Thus, one arrives at the striking conclusion that the cortex substantially amplifies the magnitude of the signal provided by the LGN but does not affect its tuning. These results seem to be at odds with those of Pei *et al.* [19], who observed a considerable sharpening of the tuning of the EPSPs over time, indicating that cortical inputs have narrower tuning than those of the LGN.

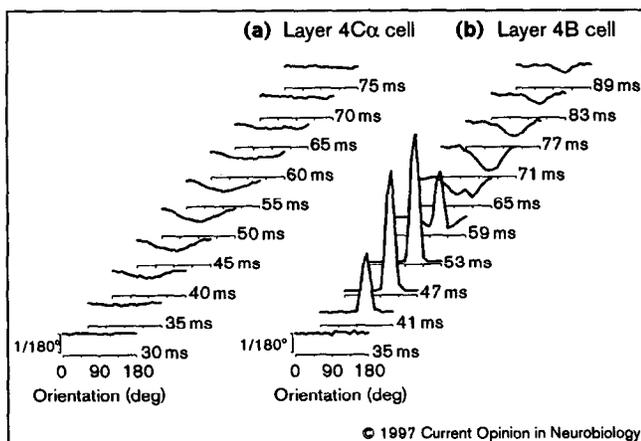
Dynamics of orientation tuning

The issue of whether the character of orientation tuning changes during the time course of the cell's response is central for understanding the mechanisms for orientation selectivity. Unfortunately, this issue is surrounded by considerable experimental uncertainty. The sharpening of the intracellular potential over time observed by Pei *et al.* [19] suggests that extracellular responses might show a similar effect. However, Celebrini *et al.* [39] found that orientation selectivity is fully developed at the very start of the discharge response in virtually all the cells recorded in V1 of awake monkeys. In contrast, Shevelev *et al.* [40] found dynamic changes in both the tuning width and the PO in most of the extracellularly recorded cells in cat visual cortex.

Recently, Ringach *et al.* [41**] searched for dynamic effects of orientation selectivity in macaque V1. In monkey striate cortex, there is a marked difference in the sharpness of the tuning across the cortical layers. In input layer 4C α , most neurons are broadly tuned for orientation, but some have no orientation selectivity at all. However, just above layer 4C, in layer 4B, and in other layers, there are many sharply tuned neurons [25**,42]. Ringach *et al.* [41**] measured the responses of cortical cells to changes in the stimulus orientation. They stimulated the cortical cells using successive brief presentations of gratings of different, randomly chosen orientations and phases, and measured the time-delayed correlations between the

spikes emitted and the orientation presented at a fixed time earlier. Figure 4 shows some of their results. In layer 4C α cells, the tuning of these orientation–response curves is often very broad. This finding is consistent with these cells having weak orientation selectivity. However, in layer 4B and above, many of the sharply tuned cells exhibit orientation–response curves that change dramatically over time. A particular feature to note is the ‘mexican hat’ orientation tuning profile seen in the layer 4B cell presented in Figure 4. In other cells, such as those in layers 2 and 3, the peak of the response tuning shifts over time. Some cells exhibit sharpening of the response tuning over time. It is interesting that this type of sharpening occurs very rapidly, within 5–10 ms of the start of the response.

Figure 4



Orientation tuning dynamics in monkey striate cortex. Extracellular recordings of single cells in monkey VI stimulated by a stream of images of sinusoidal gratings. Orientation tuning was computed as a function of time by reverse correlation. The main results shown in the figure are that (a) layer 4C α cells have simple up and down dynamics and broad orientation tuning, whereas (b) cells in layer 4B (and other output layers) have more elaborate dynamics and sharp orientation tuning. Redrawn from [41**].

Can the Hubel and Wiesel model explain orientation tuning?

Hubel and Wiesel (see [1]) proposed that orientation selectivity arises from the geometric alignment of the receptive fields of the LGN cells that project to a simple cell in the cortex. A major problem with this model is that it does not provide a mechanism for suppressing the excitatory input generated by the LGN in orthogonal orientations. As argued above, the available data indicate that the LGN input has a significant orientation bias but that the degree of its tuning varies considerably across the cell population and is typically broadly tuned, with a half-width of 45° or more. Furthermore, the LGN is expected to provide substantial excitatory input to the cortex even at NO. This scenario is depicted in Figure 5a, in which the tuning of LGN input to a simple cell was calculated under the assumption of an aspect ratio

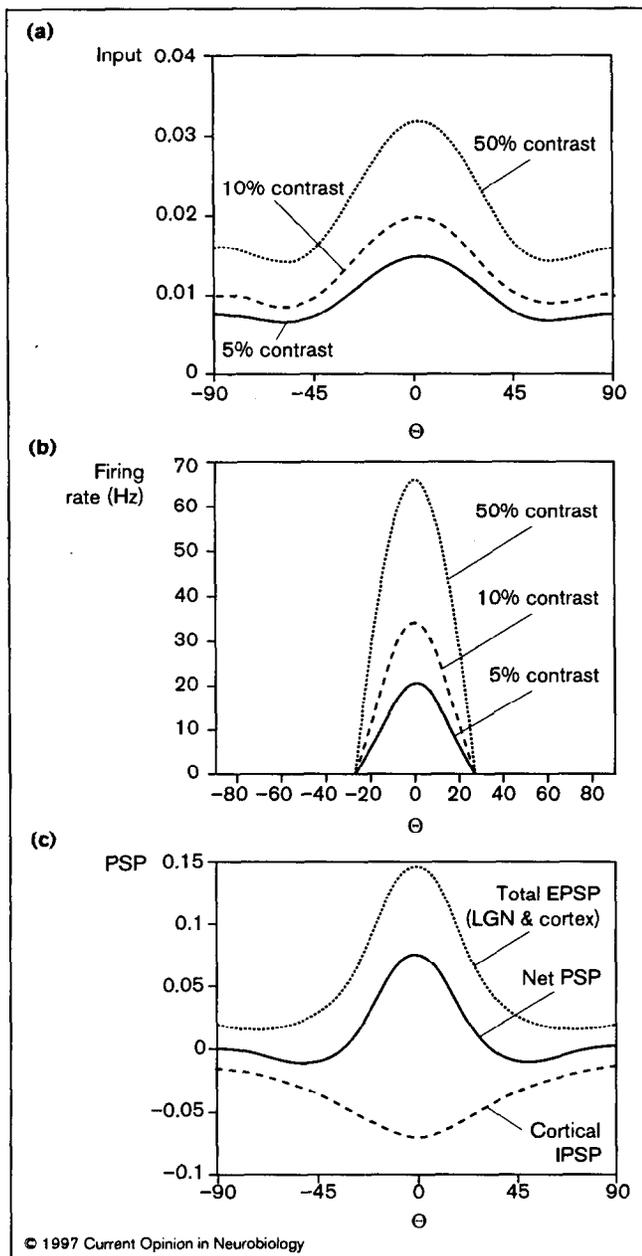
of 2. As the LGN provides only excitation to the cortex [43], there is no afferent mechanism for suppressing the significant excitatory input that is evoked when the stimulus orientation is away from the PO of the cell (except for at very low contrast, where withdrawal of background excitation may act like inhibition). Thus, a pure feedforward mechanism can account for the sharp tuning of the discharge responses only if one assumes that the cortical cell has a high threshold for generating action potentials so that only stimuli close to the PO are able to elicit discharges. An important consequence of the sharpening of the orientation tuning by the ‘iceberg’ effect of the neuronal threshold is that the resultant tuning is highly sensitive to the stimulus contrast. Thus, as illustrated in Figure 5a, it is impossible to reconcile Hubel and Wiesel’s model [1] with the observed sharp, contrast-invariant orientation tuning.

The role of cortical inhibition in orientation tuning

Several mechanisms for orientation selectivity, which are based on cortical inhibition, have been proposed (for reviews, see [44,45]). Of these, the one that is consistent with most of the data is the mechanism of nonspecific inhibition. According to this model [44,45], a cortical cell receives an inhibitory input that is only broadly tuned to orientation and serves to offset the broadly tuned excitatory input from the LGN. This inhibition plays the same role in sharpening the orientation tuning as a neuronal threshold, except that when an inhibitory input is the source of the threshold, it may yield a tuning width that is invariant to contrast if the intrinsic threshold is low. This invariance results from the fact that the activity of the inhibitory cells increases at the same time as the contrast increases, which leads to an increase in the effective threshold, thereby preventing the broadening of the tuning. The nonspecificity of the inhibitory mechanism resolves the apparent contradiction between the extracellular results of Sillito and co-workers [20,21] and others [22,23] and the intracellular results of Nelson *et al.* [24]. In Nelson *et al.*’s experiment, a fixed hyperpolarizing current was injected into uninhibited cells to maintain a normal firing rate. This current could have compensated for the removal of orientation-insensitive inhibition.

However, the nonspecific inhibition model suffers from several drawbacks. To start with, it predicts a substantial inhibitory input at nonoptimal orientations, something that has not been confirmed by most intracellular measurements. Furthermore, the strong inhibition that is needed to account for the sharp tuning will considerably reduce the responsiveness of the cortical cells, and this will yield response rates that are too low to account for the observed responses. The nonspecific inhibitory model asserts that the orientation tuning of the potential of the cortical cells is set by the LGN afferents. It thus predicts that the orientation tuning of a cortical cell is very sensitive to

Figure 5



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Orientation tuning according to the cortical recurrent excitation model. **(a)** Calculated tuning curve of the peak response of the LGN afferents (relative to spontaneous levels) to a simple cell. The LGN afferents form an input receptive field with ON-OFF-ON subfields, each with an aspect ratio of 2. A total of 24 OFF-centered LGN cells comprise the OFF subfield, and 12 ON-centered LGN cells comprise each of the side ON subfields. The displayed curves were generated by smoothing the inputs produced by a particular random realization of the locations of the LGN receptive fields' centers within each subfield. **(b)** The tuning curve of the average discharge rate of a simple cell in response to the stimulus of (a), as calculated by the recurrent network model [13,47], with narrowly tuned excitatory interactions and broadly tuned inhibitory interactions, as depicted in Figure 6a. All three curves have roughly the same shape except for a slight broadening at the edges at high contrasts. **(c)** Tuning of the calculated PSPs of the cell for 50% contrast. θ , stimulus orientation in degrees.

the quality of the LGN tuning. Cells with relatively poor LGN tuning are expected to yield broadly tuned responses (unless this is compensated on a cell-by-cell basis with a stronger inhibitory inputs). Evidently, not only cortical inhibition but also cortical recurrent excitation must be taken into account.

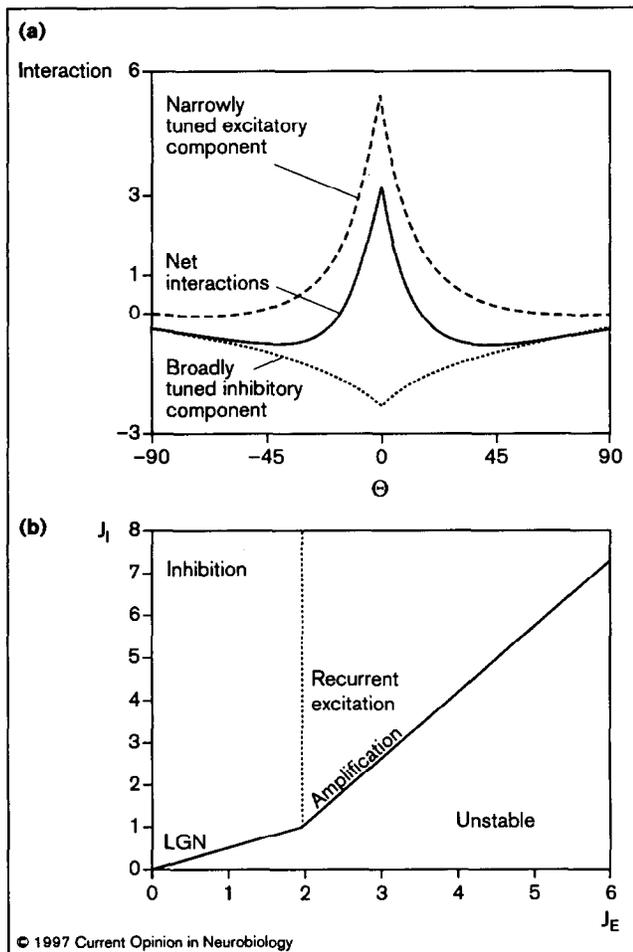
The role of recurrent cortical excitation in orientation selectivity

Two recent theoretical studies ([13,14]; see also [46,47]) have investigated the potential involvement of cortical recurrent excitation in orientation selectivity. These studies have shown that recurrent excitation may be a powerful source of sharp, contrast-invariant orientation tuning. Although the two models differ in detail, they contain the same ingredients: first, relatively weak excitatory orientation bias from the LGN input; second, excitatory connections between nearby orientation columns; and finally, inhibitory connections with a range longer than that of excitatory connections.

According to the recurrent excitation model, a central feature of cortical interactions is that the 'net' interactions between orientation columns (consisting of both excitatory and inhibitory cells) depend on their separation in a 'mexican hat' form: proximal columns excite each other, whereas distal ones inhibit each other (see Figure 6a). The inhibition controls the overall activity level of the whole network of orientation columns. The excitatory modulation ensures that this activity is not spread uniformly over the network but is restricted to a 'spot' of activity shared by the orientation columns that are close to the stimulus orientation. Hence, when the cortex is stimulated by an input from the LGN (which may have only a weak bias in favor of the stimulus orientation), the cortical interactions amplify the LGN excitatory input at the PO and suppress it at nonoptimal orientations. This combination of positive and negative feedback greatly enhances the initial orientation bias supplied by the LGN input, transforming it into a sharply tuned response profile. This mechanism for sharp orientation tuning is similar to symmetry-breaking mechanisms in pattern-forming systems.

The sharp tuning produced in the recurrent excitation network is contrast invariant. The tuning width is determined primarily by the modulation amplitude and the width of the center of the interaction profile. Increasing the contrast increases the positive feedback at the PO, but also the negative feedback at nonoptimal orientations, so that the tuning width remains unchanged (see Figure 5b). An important consequence of this mechanism is that the tuning width is relatively insensitive to the degree of tuning of the LGN input. Thus, even cells with poor alignment of LGN afferents may show sharp tuning. Similarly, sharp tuning may be elicited by visual stimuli that have small aspect ratios.

Figure 6



Phase diagram of the recurrent excitation model. (a) Profiles of the cortical interactions between orientation columns versus the difference in their PO. (b) Schematic phase diagram of the model, showing the different tuning mechanisms in the different regimes of the amplitudes of the excitatory (J_E) and inhibitory (J_I) interactions. When both interactions are weak, the discharge tuning of the cortical network depends on the tuning of the LGN afferents and is expected to be broad and contrast sensitive (the 'LGN' regime). When the excitatory interactions are weak but there is a strong, broadly tuned inhibition, sharp contrast-invariant tuning occurs (the 'Inhibition' regime). For large values of J_E and J_I , tuning is dominated by the strong modulation of the PSP produced by the combination of recurrent excitation and inhibition. In the limit of very weak LGN bias, there is a sharp 'phase transition' separating the regime of recurrent excitation from the other two regimes (i.e. the LGN and inhibition regimes) and is marked by the vertical dashed line. To minimize the relative size of the IPSP at NO, cortical amplification at PO must be strong, and this is achieved for parameters near the line marked 'Amplification'. Beyond this line, the inhibition is too weak to control the activity in the network in reasonable levels (the 'Unstable' regime). Θ , difference in the POs of the two interacting columns.

The recurrent excitation model predicts that orientation tuning should sharpen with time, as the recurrent feedback is expected to be delayed relative to the direct input from the LGN. This effect may be minimized, however, if one assumes that there is an initial, fast

feedforward inhibitory input. A cortical cell may have LGN input that peaks at one orientation and a cortical excitatory feedback that peaks at a different orientation. According to the recurrent excitation model, the PO of the cell will shift from that defined by the LGN input to that determined by the massive cortical feedback. Other dynamic effects predicted by the model include transient activation of intermediate columns in response to switching the orientation of the stimulus [13,46*].

Can the recurrent excitation model account for the intracellular data? The enhancement of tuning in the recurrent model requires strong cortical modulation of the synaptic input but not necessarily cortical amplification. The magnitude of the cortical EPSP evoked by the stimulus in the PO relative to the direct input from LGN may be large or small depending on the amount of inhibition in the circuit, as well as on contrast. The notion of cortical amplification has been advanced as an explanation for the absence of substantial inhibition at null direction and orientation in intracellular measurements [14,48–50]. According to this proposal, cortical feedback strongly amplifies the excitatory input to a cell at the PO but not at NO. Hence, at NO, a weak inhibitory input is sufficient to counter the unamplified LGN input, whereas at the PO, the dominant input is the amplified excitatory signal. On the other hand, strong inhibition is needed to stabilize the network in the face of strong excitatory feedback. These constraints require cortical interactions that produce a PSP profile with strong net inhibition at intermediate angles but weak net inhibition at NOs, as shown in Figure 5c.

The PSP tuning profile predicted by the recurrent excitation model (Figure 5c) is not entirely consistent with Ferster *et al.*'s [9**] broad PSP tuning profile under warm conditions (see Figure 2). In fact, Ferster *et al.*'s [9**] results presents an intriguing puzzle. At the high grating contrast used in their experiment, the membrane potential at the PO must be much higher than the spike threshold. Therefore, how can a broadly tuned potential that is substantially higher than the spike threshold lead to sharp discharge tuning? We should point out, however, that the authors' interpretation of the warm potential data is ambiguous. As the observed tuning refers to the amplitude of the AC modulation of the potential, it is possible that for some range of angles the signal is actually dominated by IPSPs and not by EPSPs. Hence, these data cannot be compared directly with the total PSP profile in Figure 5c. In addition, a full analysis of the experimental results must also take into account the temporal and phase relations between EPSPs and IPSPs in simple cells (A Krukowski, NJ Priebe, KD Miller, *Soc Neurosci Abstr* 1996, 22:642).

Conclusions

Recent experiments [5,9**] have been interpreted as supporting Hubel and Wiesel's model [1]. A closer look at these and other recent experiments [19,25**,26**,41**],

as well as theoretical considerations [13,14] leads us to a very different conclusion. The tuning of the LGN input to cortical cells, as inferred from the intracellular measurements [9**,19] and estimated from theory (see Figure 5), is in many cases much broader than the mean discharge tuning in the cortex. In addition, the tuning of the AC potential in the cooling experiment [9**] is presumably superimposed on an untuned DC component. Sharpening the discharge tuning of the cortical cells by threshold will yield strong dependence of the tuning width on contrast, contrary to experiment (see Figure 1). Therefore, these results reinforce the view that LGN inputs are insufficient to explain orientation tuning. Given the accumulated evidence against inhibition tuned around the NO, the natural resolution of the above problems is that sharp orientation tuning results from a combination of recurrent excitatory feedback from nearby orientation columns and inhibitory inputs from more distal columns.

Over the past years, we have witnessed a substantial advance in our understanding of the intracellular responses of cells in the visual cortex. As the problems discussed above indicate, it is now time for a systematic experimental study of both extracellular and intracellular orientation tuning in the same cells. It is also important to resolve the conflicting results regarding the dynamics of orientation tuning. An important experimental test of the role of cortical excitation would be provided by measuring the effect of changing the aspect ratio of the visual stimulus on the tuning width. Orientation tuning in the visual cortex of primates may be even more dependent on intracortical feedback mechanisms than in cats. The features of the recently observed responses to orientation changes in primate V1 indicate that the emergence of sharp orientation tuning in extragranular layers within V1 may be associated with complex intracortical dynamical processes that have yet to be elucidated.

Establishing a coherent picture of orientation selectivity in the cortex poses an interesting and important challenge for computational neuroscience. Developing a better understanding of the nature of the LGN inputs to cortex, analyzing the spatio-temporal relationships between excitation and inhibition, and considering the effects of the nonlinear dynamics of synaptic transmission are some of the avenues for future research that will advance our understanding of orientation tuning and other functions of the visual cortex.

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References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
 - of outstanding interest
1. Hubel DH, Wiesel TN: **Receptive fields, binocular interaction and functional architecture of the cat's visual cortex.** *J Physiol (Lond)* 1962, **160**:106-154.
 2. Hubel DH, Wiesel TN: **Receptive fields and functional architecture of monkey striate cortex.** *J Physiol (Lond)* 1968, **195**:215-243.
 3. Shou T, Leventhal AG: **Organized arrangement of orientation-sensitive relay cells in the cat's dorsal lateral geniculate nucleus.** *J Neurosci* 1989, **9**:4287-4302.
 4. Vidyasagar TR, Urbas JV: **Orientation sensitivity of cat LGN neurones with and without inputs from visual cortical areas 17, 18.** *Exp Brain Res* 1982, **46**:157-169.
 5. Reid RC, Alonso JM: **Specificity of monosynaptic connections from thalamus to visual cortex.** *Nature* 1995, **378**:281-284.
 6. Sclar G, Freeman R: **Orientation selectivity in cat's striate cortex is invariant with stimulus contrast.** *Exp Brain Res* 1982, **46**:457-461.
 7. Orban GA: *Neuronal Operations in the Visual Cortex.* Berlin: Springer Verlag; 1984.
 8. Skottun B, Bradley A, Sclar G, Ohzawa I, Freeman R: **The effects of contrast on visual orientation and spatial frequency discrimination: a comparison of single cells and behavior.** *J Neurophysiol* 1987, **57**:773-786.
 9. Ferster D, Sooyoung C, Wheat H: **Orientation selectivity of thalamic input to simple cells of cat visual cortex.** *Nature* 1996, **380**:249-252.
 - The authors investigated the orientation tuning of LGN input to cortical neurons. Input layer cells in cat V1 were recorded intracellularly, and orientation tuning of the intracellular potential was measured at 38°C and at 5–10°C. Cortical cooling was used to suppress intracortical inputs, by blocking nerve impulse generation in the cortex. The authors found that the tuning of the potential in the cooled cortex was the same as in the warm one, and they concluded that LGN inputs are the source of orientation tuning.
 10. Hubel D: **A big step along the visual pathway.** *Nature* 1996, **380**:197-198.
 11. Das A: **Orientation in visual cortex: a simple mechanism emerges.** *Neuron* 1996, **16**:477-480.
 12. Reid RC, Alonso JM: **The processing and encoding of information in the visual cortex.** *Curr Opin Neurobiol* 1996, **6**:475-480.
 13. Ben-Yishai R, Bar-Or RL, Sompolinsky H: **Theory of orientation tuning in visual cortex.** *Proc Natl Acad Sci USA* 1995, **92**:3844-3848.
 14. Somers DC, Nelson SB, Sur M: **An emergent model of orientation selectivity in cat visual cortical simple cells.** *J Neurosci* 1995, **15**:5448-5465.
 15. Tanaka K: **Cross-correlation analysis of geniculostriate neuronal relationships in the cat.** *J Neurophysiol* 1983, **49**:1303-1318.
 16. Chapman B, Zahs K, Stryker MP: **Relation of cortical cell orientation selectivity to alignment of receptive fields of the geniculocortical afferents that arborize within a single orientation column in ferret visual cortex.** *J Neurosci* 1991, **11**:1347-1358.
 17. Jones JP, Palmer LA: **The two dimensional spatial structure of simple receptive fields in cat striate cortex.** *J Neurophysiol* 1987, **58**:1233-1258.
 18. Ferster D: **Origin of orientation-selective EPSPs in simple cells of cat visual cortex.** *J Neurosci* 1987, **7**:1780-1791.
 19. Pei X, Vidyasagar TR, Volgushev M, Creutzfeldt O: **Receptive field analysis and orientation selectivity of postsynaptic potentials of simple cells in cat visual cortex.** *J Neurosci* 1994, **14**:7130-7140.

20. Sillito AM: **Inhibitory mechanisms influencing complex cell orientation selectivity and their modification at high resting discharge.** *J Physiol (Lond)* 1979, **289**:33-53.
21. Sillito AM, Kemp JA, Milson JA, Berardi N: **A re-evaluation of the mechanisms underlying simple cell orientation selectivity.** *Brain Res* 1980, **194**:517-520.
22. Tsumoto T, Eckart W, Creutzfeldt OD: **Modification of orientation selectivity of cat visual cortex neurons by removal of GABA mediated inhibition.** *Exp Brain Res* 1979, **34**:351-363.
23. Pflieger B, Bonds AB: **Dynamic differentiation of GABA_A sensitive influences on orientation selectivity of complex cells in the cat striate cortex.** *Exp Brain Res* 1995, **104**:81-88.
24. Nelson S, Toth L, Sheth B, Sur M: **Orientation selectivity of cortical neurons during intracellular blockade of inhibition.** *Science* 1994, **265**:774-777.
25. Sato H, Katsuyama N, Tamura H, Hata Y, Tsumoto T: **Mechanisms underlying orientation selectivity in the primary visual cortex of the macaque.** *J Physiol* 1996, **494**:757-771.
- This is the first study of the contribution of intracortical inhibition to the orientation selectivity of neurons in monkey V1, using the GABA blocker bicuculline, which was administered iontophoretically in different layers of the cortex. Orientation tuning in layers 2, 3, and 4B was markedly broadened by bicuculline. Neurons in the input layers 4C α and 4C β were broadly tuned for orientation and showed little change in orientation tuning with bicuculline. The sharp orientation tuning of layer 6 neurons was unaffected by bicuculline. The implication is that intracortical inhibition plays a significant role in orientation tuning in some, but not all, layers of macaque V1.
26. Crook JM, Kisvarday ZF, Eysel UT: **GABA-induced inactivation of functionally characterized sites in cat striate cortex: effects on orientation tuning and direction selectivity.** *Vis Neurosci* 1997, **14**:141-158.
- This is a study of intracortical interactions in cat V1. Small sites in the cortex were reversibly inactivated by iontophoresis of GABA. The effect of orientation tuning was measured from neurons located 350–700 microns away from the site of inactivation. Orientation tuning was broadened significantly when the POs of the inactivation site and the recording site differed by more than 45°. The broadening was caused by enhancing the response away from the PO of the recording site. No change in orientation tuning was found when the POs of the inactivation site and the recording site were similar.
27. Ferster D: **Orientation selectivity of synaptic potentials in neurons of cat primary visual cortex.** *J Neurosci* 1986, **6**:1284-1301.
28. Douglas RJ, Martin KAC, Whitteridge D: **An intracellular analysis of the visual responses of neurones in cat visual cortex.** *J Physiol* 1991, **440**:659-696.
29. Ferster D: **Spatially opponent excitation and inhibition in simple cells of the cat visual cortex.** *J Neurosci* 1988, **8**:1172-1180.
30. Volgushev M, Pei X, Vidyasagar TR, Creutzfeldt OD: **Excitation and inhibition in orientation selectivity of cat visual cortex neurons revealed by whole-cell recordings in vivo.** *Vis Neurosci* 1993, **10**:1151-1155.
31. Vidyasagar TR, Pei X, Volgushev M: **Multiple mechanisms underlying the orientation selectivity of visual cortical neurones.** *Trends Neurosci* 1996, **19**:272-277.
32. Peters A, Payne BR: **Numerical relationships between geniculocortical cell modules in cat primary visual cortex.** *Cereb Cortex* 1993, **3**:69-78.
33. Ahmed B, Anderson JC, Douglas RJ, Martin KAC, Nelson JC: **Polyneuronal innervation of spiny stellate neurons in cat visual cortex.** *J Comp Neurol* 1994, **341**:39-49.
34. Peters A, Payne BR, Rudd J: **A numerical analysis of the geniculocortical input to striate cortex in monkeys.** *Cereb Cortex* 1994, **4**:215-229.
35. Stratford KJ, Tarczy-Hornoch K, Martin KAC, Bannister NJ, Jack JJB: **Excitatory synaptic inputs to spiny stellate cells in cat visual cortex.** *Nature* 1996, **382**:258-261.
- A study of the connectivity and synaptic physiology of intracortical and afferent inputs to cells in slices of cat V1 using minimal stimulation and pair-recording techniques intracellularly. The authors identified three kinds of synapses onto spiny stellate cells (the input cells of V1): one from the LGN, with a large and highly reliable EPSP; another from layer 6 pyramidal cells onto layer 4, with smaller and less reliable PSPs; and a third type from other layer 4 spiny cells, with intermediate PSP sizes and reliability. The relative sizes and numbers of PSPs recorded in V1 led to the conclusion that the intracortical inputs provide most of the excitation to the simple cells in layer 4.
36. Ts'o DY, Gilbert CD, Wiesel TN: **Relationship between horizontal interactions and functional architecture in cat striate cortex as revealed by cross-correlation analysis.** *J Neurosci* 1986, **6**:1160-1170.
37. White EL: *Cortical Circuits.* Boston: Birkhauser; 1989.
38. Abeles M: *Corticonics: Neural Circuits of the Cerebral Cortex.* Cambridge, UK: Cambridge University Press; 1991.
39. Celebrini S, Thorpe S, Trotter Y, Imbert M: **Dynamics of orientation coding in area V1 of the awake primate.** *Vis Neurosci* 1993, **10**:811-825.
40. Shevelev IA, Sharaev GA, Lazareva NA, Novikova RV, Tikhomirov AS: **Dynamics of orientation tuning in the cat striate cortex neurons.** *Neuroscience* 1993, **56**:865-876.
41. Ringach DL, Hawken MJ, Shapley R: **The dynamics of orientation tuning in macaque V1.** *Nature* 1997, **387**:281-284.
- Extracellular responses of monkey V1 neurons were studied as they evolved over time. The authors measured the responses of cells in different cortical layers to a stream of images with different orientations. Using the reverse correlation technique, they obtained tuning curves of the time-delayed response to changes in stimulus orientations. Layer 4C cells were broadly tuned and had simple dynamics. Upper (2, 3 and 4B) and lower (5 and 6) layer cells had more elaborate dynamics associated with sharpening of orientation tuning and a shift in the response maxima.
42. Leventhal AG, Thompson KG, Liu D, Zhou Y, Ault SJ: **Concomitant sensitivity to orientation, direction and color of cells in layers 2, 3, and 4 of monkey striate cortex.** *J Neurosci* 1995, **15**:1808-1818.
43. Ferster D, Lindstrom S: **An intracellular analysis of geniculocortical connectivity in area 17 of the cat.** *J Physiol (Lond)* 1984, **342**:181-215.
44. Worgotter F, Koch C: **A detailed model of the primary visual pathway in the cat: comparison of afferent excitatory and intracortical inhibitory connecting schemes for orientation selectivity.** *J Neurosci* 1991, **11**:1959-1979.
45. Ferster D, Koch C: **Neuronal connections underlying orientation selectivity in cat visual cortex.** *Trends Neurosci* 1987, **10**:487-492.
46. Hansel D, Sompolinsky H: **Chaos and synchrony in a model of a hypercolumn in visual cortex.** *J Comp Neurosci* 1996, **3**:7-34.
- Simulation study of a network model of a hypercolumn in primary visual cortex, using the same gross architecture as in [13,14] but a more realistic conductance-based Hodgkin–Huxley type model of single neuron dynamics. Analysis of the results shows that the main predictions of the simplified model [13] regarding the statics and dynamics of orientation tuning also apply to this realistic model. The authors also showed that in the conductance-based dynamics, strong feedback interactions can give rise to temporally irregular firing patterns, suggesting that local recurrent intracortical dynamics may contribute to the stochasticity of neuronal responses in the cortex.
47. Hansel D, Sompolinsky H: **Modeling feature selectivity in local cortical circuits.** In *Methods in Neuronal Modeling: From Synapses to Networks*, edn 2. Edited by Koch C, Segev I. Cambridge, Massachusetts: MIT Press; 1997:in press.
48. Douglas R, Koch C, Mahowald M, Martin KAC, Suarez HH: **Recurrent excitation in neocortical circuits.** *Science* 1995, **269**:981-985.
49. Suarez HH, Koch C, Douglas R: **Modeling direction selectivity of simple cells in striate visual cortex within the framework of the canonical microcircuit.** *J Neurosci* 1995, **15**:6700-6719.
50. Douglas RJ, Martin KAC: **A functional microcircuit for cat visual cortex.** *J Physiol* 1991, **440**:735-769.