Orientation selectivity: models and neural mechanisms

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Introduction

The focus of this article is mechanisms of orientation selectivity in the visual cortex. Although many scientists have been working on this issue for many years, it resists a final comprehensive explanation. (Here we will emphasize what is known about striate cortex, V1, of macaque monkeys, but many important sources cited are from cat visual cortex experiments). In V1 there is a transformation to orientation tuned elements (in cat area 17, Hubel and Wiesel, 1962; in monkey V1, Hubel and Wiesel, 1968; Schiller et al., 1976; DeValois et al., 1982). Prior to V1, in the retina and LGN, there is weak or no orientation selectivity in single cells. Some authors have suggested the ellipticity of the LGN receptive fields as a possible source of orientation bias in the cortex (Shou and Leventhal, 1989; Smith et al., 1990; Vidyasagar, 1992). However, direct comparisons of the amount of orientation tuning expected from single LGN inputs to the amount of orientation tuning seen in single cat cortical cells reveals that ellipticity in single LGN cells to be a relatively small part of the story (Reid and Alonso, 1995). It has been thought from the time of its discovery that 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. Much has been learned about basic principles of cortical neurophysiology on account of the intense investigation of orientation selectivity.

1 Feedforward models

There are two poles of thought on the problem of orientation selectivity: feedforward filtering on the one hand, and attractor states on the other. My own view, based on experimental results and also on our own modeling is somewhere in the middle – perhaps a good label for our view of the cause of orientation selectivity in V1 would be recurrent network filtering. However, first in time, and first discussed here, is the feedforward view that is descended from the pioneering work of Hubel and Wiesel (1962). From the time of its publication, Hubel and Wiesel’s (1962) feedforward model has been a dominant idea in this field. 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. For example, in the often-encountered edge-sensitive orientation-tuned cells, Hubel and Wiesel postulated that a row of ON cells from the LGN would provide excitatory input and an adjacent parallel row of OFF cells would provide excitation for the opposite sign of contrast. There is some direct support for this kind of neural architecture in the ferret visual cortex (Chapman et al., 1991), and from dual recordings in LGN and cortex in the cat (Reid and Alonso, 1995). The experiment on cooling of cat V1 by Ferster et al. (1996) is an important result that was interpreted to mean that there is substantial orientation tuning of the collective thalamic input to a cortical neuron, consistent with the HW model. Chung and Ferster (1998) reach similar conclusions based on different experiments. In spite of all this evidence, several authors agree that the HW model predicts rather little orientation selectivity, and therefore does not account for the visual properties of V1 cells (Ferster, 1988; 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, so their firing rate during visual stimulation clips at zero spikes/sec, even at fairly low stimulus contrast. Under these conditions LGN cells would 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 would cause it to have a non-zero response at 90° from the optimal orientation. In fact, the HW model predicts that the total number of spikes elicited by a stimulus could be the same at 90° as at 0°, though the spikes would be more spread out in time at 90° (Sompolinsky and Shapley, 1997; Troyer et al., 1998). Computational simulations of the HW model have demonstrated that this analysis is correct (Sompolinsky and Shapley, 1997; McLaughlin et al., 2000), as illustrated in Figure 1 where the orientation selectivity of an HW model is shown. But many cortical cells respond little
or not at all at 90° from peak orientation, so we must conclude that the HW convergence mechanism is only a part of the story of cortical orientation selectivity.

Figure 1 --Plot of HW Model Orientation Tuning Curve from Sompol. And Shapley (1997--Current Opinion Paper) Near Here

One may wonder why the experiments of Ferster et al. (1996), and Chapman et al. (1991), are not decisive pieces of evidence for a large amount of orientation selectivity as a consequence of LGN convergence. Here is our analysis of these experiments. The cooling experiments of Ferster et al. measured the first order Fourier component (F1) in the intracellular voltage response to a drifting grating from a neuron in a cooled cortex, and found it to be as tuned for orientation as in the warm cortex. But this does not account for the tuning of the mean spike rate which is tuned as much as the F1 component of the spike rate. So to account for the orientation selectivity that is observed in the mean spike rate, it would have been necessary for Ferster et al. to measure also the orientation selectivity of the mean intracellular voltage response in the cooled cortex. They did not do this in the cooling experiments reported in 1996, probably for technical reasons. But suppose they had measured the DC response in the cooled cortex. And suppose HW model does describe the LGN input to V1 neurons. Then we can predict that Ferster et al should have found the mean (or DC) intracellular voltage response to be only weakly selective for orientation (based on the analysis of the HW model in the previous paragraph).

One can go further and analyze why Ferster et al. found so much orientation tuning for the F1 component. The only mechanism for orientation selective response for an F1 response in the HW model is different spatial frequency resolution along the two axes of the elliptical receptive field. The reason is, the HW model has no inhibition; it is a purely excitatory model. Thus, (if indeed the HW model applied) Ferster et al. probably observed orientation selectivity in the cooled cortex only because the spatial frequency of the grating they used was too high for the elliptical LGN array to resolve it along the long axis. However, the spatial frequency was not too high for the summed LGN input to resolve it along the minor axis. To put it another way, if orientation selectivity for the F1 Fourier component depended on feedforward LGN convergence as in the HW model, it would be very strongly dependent on spatial frequency. But in the normally functioning cortex, it is not (Palmer and Davis, 1981; Webster and DeValois, 1987). The conclusion of all these considerations of the cooling experiment is that it is not decisive evidence for a feedforward explanation of orientation selectivity. Similar arguments could be presented concerning the Chung and Ferster (1998) paper.

Now we turn to the Chapman et al. (1991) paper about rows of LGN terminals found in layer 4 of ferret V1 cortex, after cortical inactivation with muscimol. This was offered as evidence that the HW model could explain orientation selectivity in ferret layer 4. But later we learned that layer 4 neurons in ferret V1 are very broadly tuned for orientation –about half of the neurons in layer 4 are almost completely non-selective (Chapman and Stryker, 1993; Weliky and Katz, 1997). Therefore, the results of Chapman et al. (1991, 1993) are completely consistent with the conclusions of the analysis by Sompolinsky and Shapley (1997) and Troyer et al. (1998) that the HW convergence mechanism would make neurons only very weakly selective for orientation.

2 Cortical Inhibition

One possible addendum to the HW model, to increase the orientation selectivity greatly, has been known for a long time. One can obtain increased orientation selectivity by adding inhibition: either push-pull inhibition (Palmer and Davis, 1981; Tolhurst and Dean, 1990; Ferster, 1988, 1992; Troyer et al., 1998) or some other kind of cross-orientation inhibition (Bonds, 1989; Somers et al., 1995; Ben-Yishai et al., 1995; McLaughlin et al., 2000). But given what is known about V1 connections (Lund, 1988; Callaway, 1999) this inhibition must come through cortical interneurons rather than directly from the thalamic afferents. Experiments on intracortical inhibition in V1 have given mixed results. Initially, Sillito’s (1975) experiments with bicuculline suggested that intracortical inhibition might be 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 intracellually have been interpreted to mean that inhibition onto a single neuron is not necessary for that neuron to be orientation tuned. However,
the role of intracortical inhibition has been supported by the work of Bonds and collaborators. They have studied interactions between stimuli at different orientations (Bonds, 1989), the effects of blocking activity in infragranular layers (Allison, Pfleger and Bonds, 1995), and the effects of GABA on orientation selectivity (Allison, Casagrande, and Bonds, 1995). More recently, Eysel and his collaborators have accumulated a body of evidence in cat cortex for the important role of inhibition in causing a sharpening of orientation selectivity (Crook et al., 1998). All of this analysis of the role of inhibition is best done layer by layer in V1, because different layers have very different patterns of feedforward input and recurrent excitatory and inhibitory connections.

A theory of orientation tuning in cat cortex has been offered by Troyer et al. (1998; cf. Lauritzen et al., 2001) that attempts to explain orientation tuning in terms of specific “push-pull” inhibition (Palmer and Davis, 1981; Ferster, 1988, 1992; Tolhurst and Dean, 1990). However, the main mechanism for sharpening of orientation tuning in the Troyer et al. model is cortico-cortical inhibition that is broadly tuned for orientation. This is not the feature of inhibition emphasized by the authors. They instead emphasize that the inhibition is phase-sensitive, but the phase sensitivity is irrelevant for the sharpening of orientation tuning. In the Troyer et al. model there is moderately tuned LGN convergent excitation from an HW mechanism, 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. Therefore, this model is one of a class of cortico-cortical interaction models for orientation selectivity. Like the Ben-Yishai et al. (1995) model discussed below, it achieves contrast invariant orientation selectivity.

More recently, we have developed a large-scale model of four hypercolumns in layer 4cα of macaque V1. The model incorporates known facts about the physiology and anatomy of V1. This model accounts for many visual properties of V1 neurons, especially orientation selectivity. Inspired by the Somers et al (1995) and Ben-Yishai et al. (1995) models, it seeks to account for the same set of phenomena as these models but with more biological realism. One novelty in our model is that 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, 1999). The model places the “footprints” of synaptic excitation and inhibition on the pinwheel latticework that is revealed by cortical optical imaging (Blasdel, 1992; Bonhoeffer and Grinvald, 1993). This is illustrated in Figure 2. In its focus on the visual-functional consequences of the pinwheel organization, our model resembles the analysis of Dragoi et al. (2001) on cortical plasticity's dependence on cortical cell location within pinwheels. Our model causes significant sharpening and also diversity of orientation selectivity and produces simple cells. Orientation tuning bandwidth is approximately contrast-invariant in this model (McLaughlin et al. 2000; Wielaard et al., 2001). The most significant 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 (the opposite of push-pull). This happens because inhibition onto a model cell is a sum from many neural sources and it is likely that each of these sources is a cortical inhibitory cell with a fixed phase preference that is different from those of neighboring neurons. This view of the non-selective nature of local cortico-cortical inhibitory interactions is similar to the conclusions of Das and Gilbert (1999). It is also consistent with the measured phase insensitivity of measured inhibition (Borg-Graham et al., 1998; Anderson et al., 2000). Anderson et al. state that their data support a push-pull, that is, phase-sensitive inhibition model. However, a close scrutiny of their data reveals that much of the measured inhibitory conductance (in response to drifting gratings) is a phase-insensitive elevation of inhibition, as predicted by the McLaughlin et al. model (a point discussed in Wielaard et al, 2001).
3 Cortical excitation and attractor models

The idea that cortico-cortical excitatory feedback plays a crucial role in orientation tuning has been put forward most forcefully by theorists of brain function. There are three well-known papers that make the case for this cortico-cortical feedback. One is the paper by Somers et al. (1995) that presents an elaborate computational model for orientation tuning. Another is the paper by Douglas et al. (1995) that argues for the importance of recurrent excitation in cortical circuits, and its role in orientation tuning as an example. This is related to the papers by Douglas and Martin (1991) on the “canonical microcircuit” of V1 cortex, which described a cortical circuit with a large amount of recurrent excitation. A third paper in this genre is the work of Ben-Yishai et al. (1995). Ben-Yishai et al. offer an analytical model from which they make several qualitative and quantitative predictions. One of their important theoretical results is that one cannot predict contrast invariance of orientation tuning (as measured by Sclar and Freeman, 1981) with feedforward models, but the feedback model of Ben-Yishai et al., with recurrent excitation and inhibition, does exhibit contrast invariance. Another of their results is that if recurrent feedback is strong enough, one will observe the “marginal phase” state in which V1 behaves like a set of attractors for orientation.

The attractor states of recurrent excitatory models are discussed not only by Ben-Yishai et al. (1995) but also in the paper by Tsodyks et al. (1999). The concept is that very weakly orientation-tuned feedforward signals can be massively sharpened by strong recurrent excitatory feedback causing the cortex to respond to any visual signal by relaxing into a state of activity governed by the pattern of cortico-cortical feedback. A similar idea has been proposed by Adorjan et al. (1998). We believe this theory, like the pure feedforward theory, has trouble explaining some important data, for instance the existence of simple cells in which response waveforms follow the time course of the stimulus faithfully. Nevertheless, testing the attractor and feedforward models more carefully should be a major goal of future research.
4 Response Dynamics
In an attempt to provide a database to test models of orientation selectivity, Dario Ringach, Mike Hawken, and Shapley applied the subspace reverse correlation method (Ringach et al., 1997). 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 Figure 3. One main result from the use of this technique is that there is evidence for a slightly delayed inhibition or suppression in orientation selectivity’s time evolution. Also, in a few neurons one observes a shift of the peak orientation with time. These are the so-called “shifter” cells first described by Shevelev et al. (1993). Shifter cells are the exception not the rule. However, often in highly selective cells in the output layers of the cortex one observes a delayed suppression at the orientation that is the peak orientation early in the response. The suppression causes it to become the least preferred orientation late in the response.

Recently a paper from David Fester’s lab, by Gillespie et al. (2001), has challenged the results of our earlier paper on orientation dynamics (Ringach et al., 1997). Gillespie et al. reported that the intracellular membrane voltage’s orientation tuning, in cat cortical neurons, did not change with time. They concluded that feedforward mechanisms could explain their observations. However, my colleagues and I believe that Gillespie et al.’s results were different from ours mainly because the time resolution of their measurements was inadequate to pick up the rapid recurrent cortico-cortical interactions that sharpen orientation tuning. The time dependence of orientation tuning is illustrated for one macaque 4cα neurons in Figure 4, row (a).

Figure 4. Time dependence of orientation tuning in a macaque 4cα neuron and in the large-scale model of McLaughlin et al. (2000). In the first 4 panels, p(θ,τ) is graphed. In the fifth panel the circular variance (CV) is shown. Row (a) is for a typical 4cα neuron; row (b) is from the model. The dotted line in the rightmost (b) panel is the CV vs time for a pure feedforward model.

Figure 4 also illustrates that a feedforward model would produce little orientation selectivity in the reverse correlation experiment. This is illustrated in the rightmost panel of row (b), in which circular variance 9CV) is drawn for the full model (solid line) and for a pure feedforward model (dotted line). Circular variance is a global measure of selectivity, as defined below.

5 Bandwidth and Circular Variance
There are different ways to measure orientation selectivity, and they can tell us about different aspects of orientation selectivity. A traditional method is to determine the half-bandwidth of the tuning curve around the peak of the tuning (e.g., Schiller et al., 1976; DeValois et al., 1982). This tells one the shape of the tuning curve near the peak. However, there are important questions about mechanisms that depend upon the global shape of the tuning curve at all orientations. Various vector-averaging measures have been devised by different investigators. We favor the use of circular variance, a measure that is used in circular statistics (Mardia, 1972). The circular variance of the spike rate m(θ) is:

$$CV[m] = 1 - \left| \frac{\int m(\theta) \exp(2i\theta) d\theta}{\int m(\theta) d\theta} \right|$$
Circular variance is $1 - \{\text{relative modulation of } m(\theta) \text{ as a function of } \theta \}$. The relative modulation is the ratio of the best fitting Fourier component of the orientation tuning curve (with period $= 180$ deg), divided by the average response. For a flat tuning curve CV=1. For a very highly tuned tuning curve, CV $\rightarrow 0$. CV reflects wide-angle responses that the bandwidth does not. Other investigators have also used global measures for selectivity that are related to circular variance. For instance, Chapman and Stryker (1993) and Dragoi et al. (2001) use an Orientation Selectivity Index OSI that is effectively $1 - \text{CV}$. One possibility we will be seeking to evaluate in proposed experiments and also in analysis of our cortical model is whether the bandwidth of a V1 neuron is linked more to feedforward input, while circular variance is influenced more by cortico-cortical interactions.

6. Conclusions

In our view, cortico-cortical inhibition is a crucial ingredient in the emergence of orientation selectivity in the visual cortex. The orientation preference of each neuron, and the orderly orientation preference map, are likely to be consequences of the pattern of feedforward convergence. However, the selectivity observed in steady state experiments and even moreso in orientation dynamics experiments, cannot be achieved by a purely feedforward model. At present we cannot yet evaluate the relative importance of cortico-cortical excitation in enhancing orientation selectivity. It may play a role for some neurons. For cortical simple cells, the results of our modelling indicate that cortico-cortical excitation has to be relatively weak compared to the LGN excitation (see Wielaard et al., 2001). However, it is highly likely that cortico-cortical excitation plays a bigger role in complex cells of V1 (Chance et al., 1998).