Neuronal connections underlying orientation selectivity in cat visual cortex

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How do neurons of the visual cortex acquire their acute sensitivity to the orientation of a visual stimulus? The question has preoccupied those who study the cortex since Hubel and Wiesel[1] first described orientation selectivity over twenty-five years ago. At the time, they proposed an elegant and enduring model for the origin of orientation selectivity. Fig. 1A, which is adapted from their original paper and which contains the essence of their model, is by now familiar to most students of the visual system and to many others besides. Yet the model, and the central question that it addresses, is still the subject of intense debate. Competing models have arisen in the intervening years, along with diverse experiments that bear on them.

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The major question that any model must address is how the response of every cortical neuron in the cat is so critically dependent on the orientation of a visual stimulus, even though many neurons, both simple and complex, receive the bulk of their excitatory input from relay cells of the lateral geniculate nucleus (LGN), which are largely insensitive to orientation. When the image of a bar or edge is rotated 90° from the orientation that is most effective in activating a particular cortical cell, the cells in the LGN that provide its visual input hardly change the magnitude of their response: what causes the cortical neuron to cease responding? Hubel and Wiesel's model relies on the spatial relationship of the receptive fields of presynaptic geniculate relay cells. But there are several ways in which intracortical inhibition might contribute as well. We will consider a number of proposals for the origins of orientation selectivity in turn.

The excitatory model of cortical orientation selectivity

In Hubel and Wiesel's model, orientation selectivity is first created in one class of cortical neurons, the simple cells, distinguished by the discrete subregions that can be found in their receptive fields (Fig. 1A). These regions resemble the centers of the receptive fields of ON- and OFF-center neurons in the LGN. Light increment in an ON region or light decrement in an OFF region elicits a response in a simple cell; light decrement in an ON region or light increment in an OFF region suppresses activity. The main difference between the centers of geniculate neurons and the subregions of cortical cells is their shape. Cortical subregions are often elongated, their width being similar to the diameter of the receptive field centers of neurons in the corresponding region of the LGN. From this arrangement, Hubel and Wiesel suggested that a simple cell subregion is generated directly by excitatory synaptic input from a row of geniculate neurons whose receptive field centers overlap the subregion. When a bar of light is oriented so as to fall simultaneously on the receptive fields of all the presynaptic geniculate neurons, their combined activity drives the simple cell above threshold. When the stimulus is oriented improperly, falling on only a few of the geniculate cells' receptive fields, those few alone are too weak to evoke action potentials in the simple cell. In addition, an improperly oriented bar will simultaneously stimulate adjacent subfields. Therefore, an increase in excitation evoked by the bar in one subfield will necessarily be antagonized by a withdrawal of excitation from the other. For example, a bright bar flashed at an angle to the preferred orientation would increase the activity of ON-center cells underlying an ON region and reduce the spontaneous activity of OFF-center

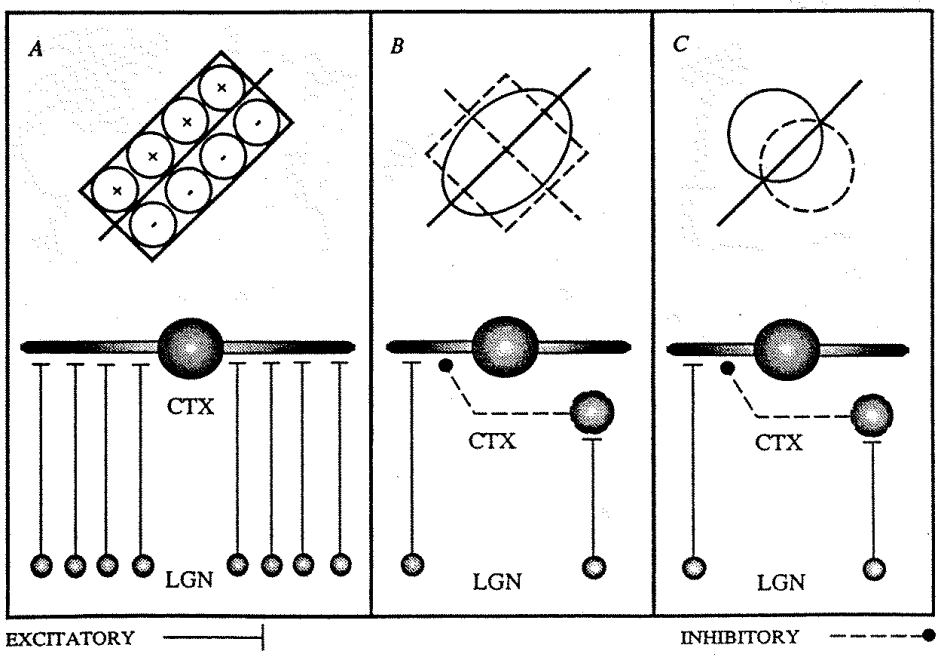

Fig. 1. *Schematic representations of three models for the origin of orientation selectivity in neurons of the visual cortex.* **(A)** *The model proposed by Hubel and Wiesel[1], based on the spatial organization of the receptive fields of a number of presynaptic geniculate neurons.* **(B)** *The cross-orientation model, in which inhibition from orientation-specific cortical neurons with preferred orientation near the null orientation of the postsynaptic neuron prevents the latter from responding to the rather poorly oriented excitation from the LGN.* **(C)** *A model from Heggelund[51] in which non-oriented excitation from the LGN and non-oriented intracortical inhibition confer orientation selectivity onto the postsynaptic cell by virtue of the offset in their receptive fields. See text for further discussion.*

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cells underlying the adjacent OFF region.

Several experiments have confirmed some of the predictions of the model. (1) According to Jones and Palmer[2], the width of orientation tuning of a simple cell judged from extracellular recording can often be predicted from the size and shape of its receptive field, assuming that spatial summation in the receptive field is linear. As one might expect, the longer and narrower the subfields of a given simple cell, the sharper its orientation tuning. For many cells, the relationship holds with a great degree of accuracy, based on the unusually sensitive receptive field maps Jones and Palmer were able to obtain with their reverse correlation technique. (2) All simple cells appear to receive powerful monosynaptic excitation from relay cells of the LGN[3]. (3) The EPSPs evoked in simple cells by bars of light moved through their receptive fields are strongly tuned for orientation[4,5]. (4) Several properties of visually evoked EPSPs[6] can be well fit by quantitative models based on Hubel and Wiesel's proposals[6–9], including the size and shape of the receptive fields of EPSPs, the extremely sharp orientation tuning, the dependence of sensitivity on the length : width ratio of the EPSP's subfields, and the insensitivity of tuning width to inactivation of retinal ON-center cells by the pharmacological agent, APB.

Cross-orientation inhibition

Despite the simplicity of Hubel and Wiesel's model and the experiments that support some of its predictions, it is difficult to prove that the arrangement of the receptive fields of presynaptic inputs is sufficient to produce orientation selectivity on its own. That different mechanisms may operate in different neurons would not be surprising given the complexity of the cortical circuit. Even within the simple cell category, neurons vary widely in dendritic morphology[10,11]. And simple cells are not the only neurons to receive direct input from the LGN; complex cells of layer 3, which have very different receptive field structure than simple cells, do as well[3,12]. Orientation selectivity may be generated independently in different layers within a cortical column[13,14], and it is conceivable that different mechanisms operate at different sites, each with the same preferred orientation.

Inhibition of cortical origin has been suggested as an alternative mechanism underlying orientation tuning. Suppression or inhibition of any sort is difficult to detect in extracellular recordings from cortical cells since most are silent in the absence of an excitatory stimulus. But when the background activity of a cell is elevated by a conditioning stimulus of optimal orientation[15–17] or by excitatory amino acids[18,19], the suppressive effects of a test stimulus can be seen against the background. The observation that stimuli 90° to the optimal produce a strong suppression has given rise to the cross-orientation inhibition model. In this model, a small number of geniculate neurons excite a cortical neuron; neither the individual inputs, nor their summed effects possess strong orientation tuning. Orientation selectivity would then be imposed on the cell via intracortical inhibition; IPSPs would block the response of the cell to any EPSPs evoked by stimuli of the non-preferred orientation – hence the name cross-orientation inhibition. It would arise from other cortical cells with preferred orientations different from that of the cells they inhibit (Fig. 1B).

In extracellular experiments it is difficult to distinguish inhibition from a withdrawal of excitation. It is also difficult to compare the magnitudes of inhibition at the null and the preferred orientations; any inhibition present at the preferred orientation is overwhelmed by the large excitatory responses. Some of the most compelling evidence for the role of inhibition in orientation selectivity, therefore, is provided by experiments in which the action of the principal cortical inhibitory transmitter, γ-aminobutyric-acid (GABA) is blocked pharmacologically. When cortical inhibition is removed by application of *N*-methyl-bicuculline or bicuculline methiodide, orientation selectivity is abolished in some fraction of simple and complex cells[20–23]. It has been concluded that excitatory connections, which are presumably left functional by the drug, are insufficient to produce sharp orientation tuning on their own and require the superimposed effects of cross-oriented IPSPs to provide the high degree of specificity found in cortical cells.

Cross-orientation inhibition is in itself insufficient to explain the origin of orientation selectivity. One cannot invoke orientation-specific neurons to explain the origin of orientation-specific neurons. But if two sets of neurons with orthogonal preferred orientations possessed even a slight bias for orientation by virtue of their excitatory input, mutual inhibition could conceivably sharpen their tuning to the degree observed in most cortical cells. The bias required could come from a mechanism similar to that proposed by Hubel and Wiesel, or as suggested by some, from the reported orientation bias of single retinal ganglion cells[24,25] and geniculate neurons[26].

For most purposes, the assumption that the receptive fields of cat retinal ganglion cells (and of geniculate neurons) possess perfect circular symmetry is a good one. But even in Kuffler's[27] original drawings of retinal receptive fields, some asymmetries are present. The average ratio of the response amplitudes at the optimal and null orientations in geniculate neurons has been reported to be as high as 2 : 1[26]. This bias could provide a substrate for the sharpening effects of cross-orientation inhibition[28]. Soodak, Kaplan and Shapley[29], however, have reported that the ratio of the major and minor axes of the centers of geniculate receptive fields is rarely higher than 1 : 3, and that orientation bias, the ratio of the response to optimal and null oriented stimuli, is as high as 2 : 1 only at relatively high spatial frequencies, when the amplitude of a cortical cell's response would be low.

According to the cross-orientation inhibition model, one would expect when recording intracellularly from cortical neurons to find EPSPs that are relatively poorly tuned for orientation. IPSPs would have a preferred orientation 90° from that of the EPSPs, and inhibition evoked by the null orientation

would prevent the cell from responding to any EPSPs except those evoked by appropriately oriented stimuli. Creutzfeldt, Kuhnt and Benevento[4] and Ferster[5,6], however, found that in many cells, EPSPs are quite well tuned for orientation. Even more striking, IPSPs are also well tuned, not to 90° from the EPSPs' preferred orientation, but to the EPSPs' preferred orientation itself. This strong inhibition is likely to have many functions, including disparity sensitivity, end inhibition and direction selectivity[30]. At the cross-orientation, however, inhibition is much weaker than at the preferred orientation, and is sometimes undetectable.

The intracellular presence of well-tuned EPSPs and of only weak cross-oriented inhibition is surprising given the evidence for cross-orientation inhibition from extracellular and pharmacological experiments. Might the strong IPSPs predicted at the cross-orientation be present, yet remain invisible to a microelectrode in the cell soma? One mechanism that has been proposed is shunting or silent inhibition. When the reversal potential of an inhibitory synapse is at or near the resting membrane potential, activation of a silent synapse by itself would not lead to a change in the membrane potential. Yet a local increase in conductance could attenuate very effectively an EPSP produced by a synapse near to or more distal to the inhibitory synapse[31–33].

The experiments of Ferster[5] place severe constraints, however, on any explanation involving shunting inhibition. Responses evoked by null-oriented stimuli were always recorded both at rest and while the cell was depolarized with DC current injected through the recording electrode. When the membrane potential is raised in this way, inhibitory synapses with reversal potentials near rest should produce large hyperpolarizing IPSPs. In the experiments, inhibition could clearly be detected in response to electrical stimulation of the LGN, and to moving or flashing bars of the optimal orientation; the same procedure, however, revealed little inhibition at the non-optimal orientation.

A large fraction of the geniculate input to cortical cells appears to end on synaptic spines[34]. It is well known, however, that between 5 and 20% of all dendritic spines receive both asymmetrical and symmetrical synapses, that is, presumably both excitatory and inhibitory synapses[35,36]. This highly local synaptic circuit could well underlie a number of specific computations on the excitatory geniculate input, such as orientation or direction selectivity[37], yet due to the electronic properties of spines and dendrites, the action of the local IPSPs, particularly of the shunting type, might well remain invisible in the soma. Perhaps inhibition for the null orientation is located on spines or located distally in the dendritic tree and not conducted electrotonically to the soma, while inhibition for the optimal orientation would be located near the soma. But models and experiments suggest that the electrotonic properties of neurons should allow potentials generated even in the distal dendrites to be visible in the soma[38–40].

An additional problem with shunting IPSPs in local circuits involves the relative timing of EPSPs and IPSPs. To be effective in attenuating EPSPs, a shunt must be open during the early part of the excitatory conductance change underlying the EPSP[33,41]. The rising phase of bicuculline-sensitive IPSPs observable in cortex[42] lasts no longer than 1 ms, however, placing great demands on the accuracy of the relative timing of locally acting IPSPs, particularly when the firing rates of the cortical neurons are likely to be less than 30 s^{-1}.

Where shunting conductance is thought to be important in shaping visual receptive field properties is in direction-selective cells of the rabbit retina. Movement of a bar in the null direction coincides with the activation of an inhibition that at rest shows itself only by the diminution of the visually evoked EPSPs (relative to the EPSPs evoked by movement in the optimal direction). Depolarization of the cell with current, however, does reveal a hyperpolarizing IPSP[43,44].

The effects of bicuculline on orientation selectivity have been attributed to the removal of cross-orientation inhibition, i.e. IPSPs specific to the generation of orientation selectivity. Yet only small IPSPs have been recorded intracellularly in response to null-oriented stimuli. Might the bicuculline experiments be explained by the removal of *all* inhibition, not only from the cortical cell whose orientation selectivity is to be tested, but from

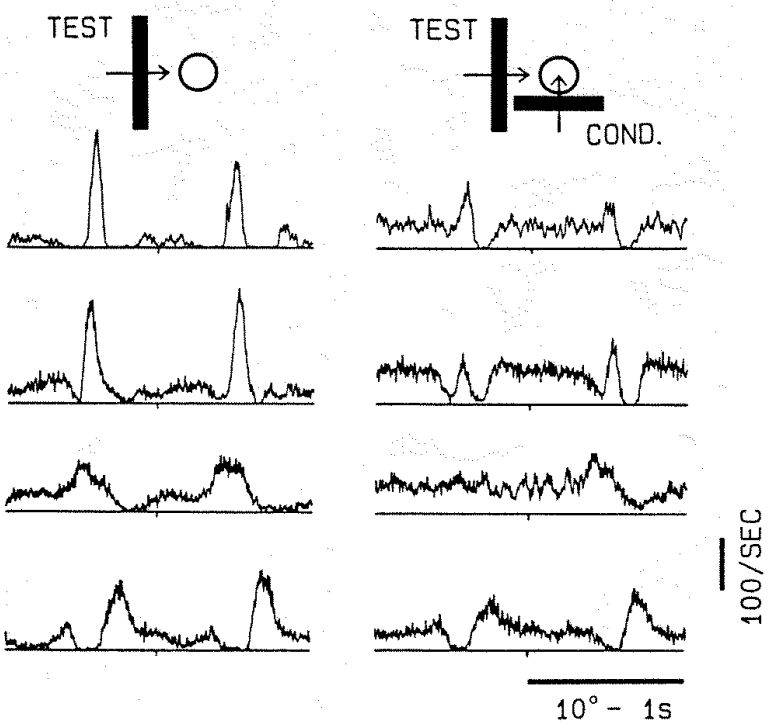

Fig. 2. *Cross-orientation effects in neurons of the LGN. To the left are shown histograms of the responses of four geniculate neurons (two ON-center and two OFF-center) to a bright bar swept repeatedly over their receptive fields. To the right are the responses to the same bright bar, presented simultaneously with a second bar at right angles to the first whose sweep was unsynchronized with respect to the collection of spikes into the histogram. The second bar reduces the size of the peak response to the first, and significantly raises the background activity.*

thousands of neurons in the immediate vicinity, many of which have excitatory connections with each other? A non-specific increase in excitability might raise the threshold of a cortical cell and allow it to respond to almost any stimulus, even the weak, but normally subthreshold one predicted by Hubel and Wiesel to be present at the non-optimal orientation. It has been found[6] that increasing the excitability of a cortical cell by intracellular penetration and application of Ce^{+} – in order to block K^{+} channels and raise the membrane resistance – can result in spikes being evoked at the non-optimal orientation where presumably there were none before.

Bicuculline at high enough concentrations can induce seizures in cortical tissue and is often used for the study of epilepsy[45]. It is suggestive that other cortical receptive field properties, such as direction selectivity, disappear rapidly after the application of bicuculline, with a time course similar to the disappearance of the response to iontophoretically applied GABA. The suppression of orientation selectivity, in contrast, requires extended application, and for some neurons has incomplete or no effect in area 17[22,46] and area 18[47]. This mirrors the presence of large IPSPs evoked at the preferred orientation and small IPSPs evoked at the null orientation. Perhaps the rapid loss of direction selectivity does reflect the loss of inhibition specific to that receptive field property, while the loss of orientation selectivity some time later reflects a non-specific increase in excitation of a group of cortical cells.

That an improperly oriented stimulus can suppress the activity of a cortical cell could mean that a specific inhibition is evoked by that stimulus. Experiments of this kind, however, place important constraints on the properties of the inhibitory interneurons that would mediate the inhibition. A null-oriented stimulus in one eye, for example, is reported not to affect the response to an optimally oriented stimulus in the other eye[48,49]; although see Ref. 50. The interneurons must therefore be monocular, and their synapses must be electrically isolated within the dendritic tree from the excitatory geniculate input from the opposite eye.

Some of the suppressive effects of null-oriented stimuli could also be explained by their effects on the response of geniculate neurons. Fig. 2 shows an example of a cross-orientation effect on the response of four geniculate cells to a moving bar. The peak response is reduced and the background activity greatly increased by a second, orthogonal stimulus, resulting in a large decrease in signal-to-noise ratio. A geniculate origin for the effect of null-oriented stimuli would also explain the monocularity of the effect. We suggest, therefore, that while the issue of cross-orientation inhibition remains controversial given the conflicting evidence, this type of inhibition may make only a small contribution to the orientation selectivity of cortical cells.

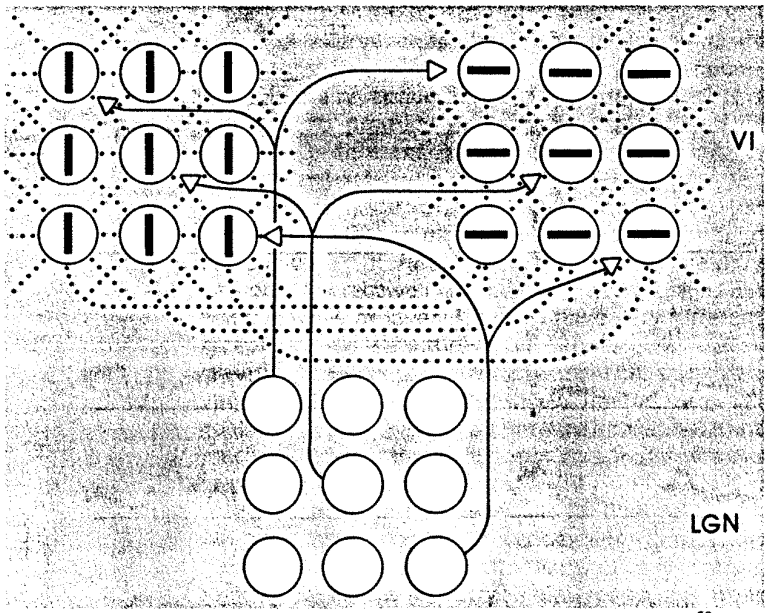

Fig. 3. *Skeleton version of the eclectic model proposed by one of us[60]. The cortical interneurons acquire orientation selectivity via two different inhibitory systems superimposed on the Hubel and Wiesel type of excitatory system (not shown). Local inhibition among cells – most likely within the same orientation column – with the same preferred orientation (but spatially offset in a range of directions perpendicular to the preferred orientation) strongly reduces the response of the cells to non-optimal orientations, in agreement with Ferster's intracellular data (see also Orban[59]). Cross-inhibition between cells with overlapping receptive fields but orthogonal preferred orientations sharpens orientation tuning. Recent anatomical evidence has revealed extensive synaptic contacts among GABAergic interneurons in monkey cortex[64]. Since each neuron contributes partially to the orientation selectivity of its neighbors, no non-oriented cells are required. Excitatory pyramidal cells are easily incorporated into this scheme.*

Alternative models

In simple cells, the spatial organization of inhibition evoked by stimuli at the preferred orientation takes a very specific form. Superimposed on each ON subfield is OFF inhibition, and on each OFF subfield, ON inhibition[30,51–53]. Each inhibitory mechanism is centered on the corresponding excitatory subfield, but is broad enough so that it partially overlaps adjacent subregions. Some have suggested that this inhibition arises from neurons with geniculate-like, non-oriented receptive fields (Fig. 1C)[51,52,54]. Were this so, the inhibition could generate orientation selectivity by suppressing responses to stimuli at 90° to the preferred orientation; such a stimulus would fall across the receptive fields of both the excitatory and inhibitory inputs simultaneously. So far, however, no significant population of non-oriented cells that could provide such inhibition has been reported in cat striate cortex[55], and there is little evidence for monosynaptic inhibition of cortical cells from the LGN[3,56,57]. It is conceivable, however, that the model could operate in the monkey visual cortex, given the numerous cells in layer 4c with non-oriented geniculate-like receptive fields[58]. In the cat, it is most likely that this inhibition arises from inhibitory simple cells with orientation preference similar to that of their postsynaptic targets, but with ON regions wherever the postsynaptic cell has OFF regions, and vice versa[30,53]. Even though the neurons supplying the inhibition have the same orientation preference as the postsynaptic cell, inhibition from cells with spatially displaced subfields

could still contribute to orientation selectivity[59].

This type of inhibition plays an important role in a model recently proposed by one of us, which combines in an eclectic manner features from each of the three models discussed (Fig. 3)[60]. In this model, which is currently being explored in computer simulations, the basic excitatory scheme of Hubel and Wiesel[1] (not shown in Fig. 3) provides for some orientation selectivity via synaptic input from an appropriately oriented row of geniculate cells. The postulated inhibition between simple cells of similar orientation could significantly sharpen orientation tuning independently of the length : width ratio of the excitatory subfields of the cortical cell. Inhibitory interneurons receiving direct geniculate excitation (most likely basket or clutch cells[61,62]) would inhibit each other in an orientation-specific manner. They would have simple receptive fields and code for the same orientation, but their receptive fields would be spatially displaced in the direction perpendicular to the preferred orientation. For instance, each neuron in the left group of cells in Fig. 3 forms inhibitory connections with its neighbors to the left and to the right, but not with those above or below. Thus, if a vertical bar excites one of these cells, no inhibition from neighboring cells will prevent it from firing. At the non-optimal orientation, all cells would be weakly excited – by geniculate input – but would receive some inhibition from all neighboring cells of the same preferred orientation as itself. This requires that the neighboring cells respond slightly to the non-optimal stimulus. Conversely the group of neurons at the top right of Fig. 3, which are selective for horizontal stimuli, have no inhibitory interconnections in the left–right direction, but inhibition in the up–down direction. Thus, if one would record from any of these neurons intracellularly, one would see IPSPs tuned to the optimal orientation, in agreement with Ferster[5].

A second system of inhibition between cells of different orientation, but with spatially overlapping receptive fields, could be superimposed onto the first (i.e. cross-orientation inhibition). This second inhibitory component could sharpen orientation tuning by suppressing responses at the non-optimal orientations. Since the excitatory input at the non-optimal orientation would be already strongly reduced from its peak at the optimal orientation, a small amount of cross-oriented inhibition would suffice to completely block the response of the cell.

Given the eclectic nature of this model, it seems to account for most of the experimental data from both the excitatory and inhibitory schools of thought. The model relies on massive feedback among cortical neurons to yield orientation selectivity, which may better reflect the cortical anatomy than earlier models, which tend to be of the feed-forward type. Interestingly, this model can be described by a Hopfield type of neural network formalism[63]. It may well be that variations of this network, duplicated across different cortical layers and areas explain orientation selectivity not only in simple but also in complex cells.

One method that may shed light on this issue is computer simulation. Koch and Van Essen (in preparation) are currently modelling the foveal X-system of the cat from the detailed mosaic of beta cells in the retina that are now available[64] to layer 4c of the striate cortex (see also Soodak[65]). We believe that important insights can come from this approach if such simulations incorporate the high degree of convergence and divergence evident in the geniculocortical and intracortical projections, and if they are based on realistic models of the electrophysiological properties of nerve cells.

Concluding remarks

The problem of the appearance of orientation selectivity in the visual cortex has attracted so much attention in part because it represents a complex function of neocortex which is nevertheless well-defined, when so much of the function of other cortical areas remains a mystery. What better place to understand the way in which the neuronal interconnections of the cortex underlie cortical function? But even after 25 years of study, a resolution to the problem has not emerged. Whether one or the other of the various models discussed here will prove true, or as is often the case, some combination is actually present in the cortex, or whether some entirely new mechanism will appear, remains to be seen.

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