Complex Receptive Fields in Primary Visual Cortex

LUIS M. MARTINEZ and JOSE-MANUEL ALONSO

In the early 1960s, Hubel and Wiesel reported the first physiological description of cells in cat primary visual cortex. They distinguished two main cell types: simple cells and complex cells. Based on their distinct response properties, they suggested that the two cell types could represent two consecutive stages in receptive-field construction. Since the 1960s, new experimental and computational evidence provided serious alternatives to this hierarchical model. Parallel models put forward the idea that both simple and complex receptive fields could be built in parallel by direct geniculate inputs. Recurrent models suggested that simple cells and complex cells may not be different cell types after all. To this day, a consensus among hierarchical, parallel, and recurrent models has been difficult to attain; however, the circuitry used by all models is becoming increasingly similar. The authors review theoretical and experimental evidence for each line of models emphasizing their strengths and weaknesses. NEUROSCIENTIST 9(5):317–331, 2003. DOI: 10.1177/1073858403252732

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In 1938, Hartline introduced the term "receptive field" to name a region of the retina where a change in light brightness modified the firing rate of a retinal ganglion cell. Kuffler (1953) would later demonstrate that Hartline's receptive field had a specific spatial structure known as center surround, and in a series of studies to follow, Hubel and Wiesel (1959, 1961, 1962, 1965) would extend the term receptive field to other cells in visual cortex and would demonstrate that receptive-field structures become increasingly complex at successive stages of the visual pathway.

In comparison with the retina and the lateral geniculate nucleus (LGN), the primary visual cortex turned out to have a remarkable variety of receptive fields. Hubel and Wiesel classified cortical receptive fields into two main categories—simple cells and complex cells—after admitting that "new varieties [of receptive fields] are continually appearing, and it is unlikely that the ones we have listed give anything like a complete picture of the striate cortex" (Hubel and Wiesel 1962, p 109). Hubel and Wiesel's (1962) simple receptive fields had a very characteristic spatial structure. Like cells in the LGN, they had separate *on* and *off* subregions that could be mapped with small spots of light. Unlike geniculate cells, the *on* and *off* subregions were elongated and parallel instead of circular and concentric (Fig. 1). Simple receptive fields were identified based on four criteria:

1. they were subdivided into distinct excitatory and inhibitory regions,
2. there was summation within the separate excitatory and inhibitory parts,
3. there was antagonism between excitatory and inhibitory regions, and
4. it was possible to predict responses to stationary or moving spots of various shapes from a map of the excitatory and inhibitory areas.

In contrast, complex receptive fields formed a much more diverse population and were identified by exclusion. Figure 2 shows three different types of complex receptive fields, as illustrated in Hubel and Wiesel (1962). Those three examples share very little in common. Cell *A* generates *on-off* responses throughout the entire receptive field. Cell *B* responds exclusively to a black horizontal bar. Cell *C* has partially separated *on* and *off* regions, but unlike a simple cell, its receptive field cannot be mapped with light spots.

Hubel and Wiesel's (1962) description did not provide a quantitative test to clearly distinguish between simple and complex receptive fields. The lack of such a test would later become a serious problem and eventually would lead to the proposal of many different classification criteria (Palmer and Rosenquist 1974; Schiller and others 1976; Henry 1977; Henry and others 1983; Tanaka 1983; Toyama and others 1981a, 1981b; see

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Neuroscience and Motor Control Group (Neurocom), Department of Medicine, Campus de Oza, Universidade de A Coruña (LMM). Department of Biological Sciences, SUNY College of Optometry (J-MA).

Address correspondence to: Jose-Manuel Alonso, Department of Biological Sciences, SUNY-Optometry, New York, NY 10036 (e-mail: jalonso@mail.sunyopt.edu).

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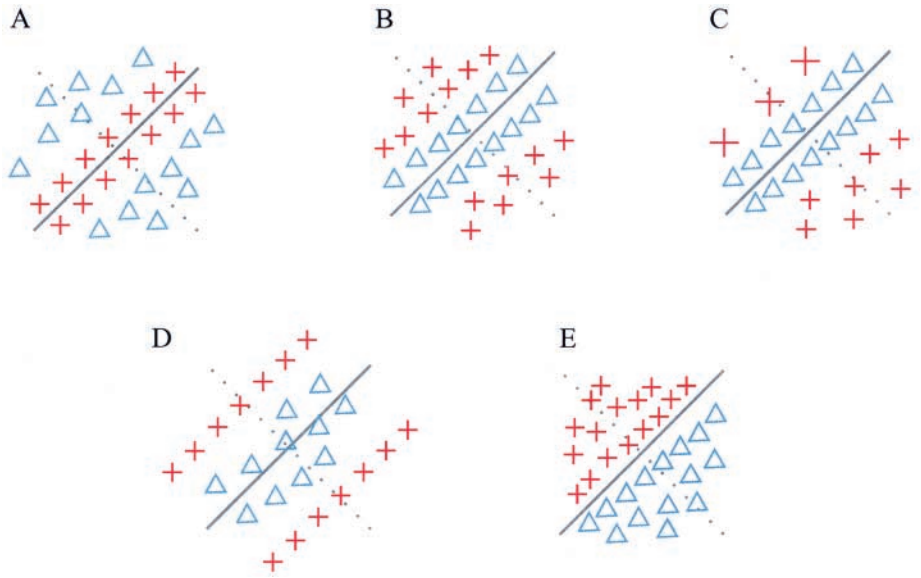

Fig. 1. Simple receptive fields (colored version of Figure 2 from Hubel and Wiesel 1962). Red crosses represent on subregions and blue triangles off subregions.

Orban 1984 for review). Simple cells and complex cells have been classified based on the presence and degree of overlap of *on* and *off* subregions (Fig. 3*A*), spontaneous activity level, response amplitude, length summation, responses to patterns of random dots, responses to moving light and dark bars, responses to moving edges, responses to drifting or contrast-reversal gratings, and responses to flashed bars and reverse correlation maps (Fig. 3*B*; see Skottun and others 1991a); Mechler and Ringach 2002, for review). To give an example of the confusion, cell *B* of Figure 2 could probably be classified by different authors as complex (e.g., Hubel and Wiesel 1962), simple (e.g., Skottun and others 1991a), S1 (e.g., Schiller and others 1976; Martin and Whitteridge 1984; Jones and Palmer 1987; see Orban 1984, for review) or Eoff (e.g., Tanaka 1983). Similarly, cell *C* could probably be classified either as complex (e.g., Hubel and Wiesel 1962) or simple (e.g., Debanne and others 1998).

A successful quantitative test to classify simple and complex cells was introduced by De Valois and colleagues (1982) and further refined by Skottun and others (1991a). The so-called response modulation method provided a mixed quantification of the 2nd and 3rd criteria of Hubel and Wiesel (1962) and rendered two cell populations that roughly corresponded to simple and complex cells (Fig. 3*C*). This new classification was based on the different response modulation of simple cells and complex cells to drifting sinusoidal gratings. Although simple cells tend to modulate their firing rate in phase with the stimulus, complex cells elevate their firing with little or no modulation. Thus, the ratio between the amplitude of the first Fourier harmonic and the mean spike rate can be used as a quantitative index of "response non-linearity" or "receptive-field complexity" (Fig. 3*C*). Unfortunately, this test is not free of limitations. The "response modulation" does not provide any information on the receptive-field geometry (e.g., number and shape of subregions), and therefore it cannot distinguish an X-geniculate cell from a simple cell even if thalamic and cortical cells are clearly different populations. Moreover, granted a nonlinear relationship between synaptic current and firing rate, cells with identical synaptic inputs and intrinsic properties can still show a bimodal distribution of response modulation (Mechler and Ringach 2002).

The difficulty in classifying receptive fields is not simply a problem of semantics. It is closely related to fundamental questions such as, how are simple and complex receptive fields generated? And what is the role of simple and complex receptive fields in visual processing? Possible answers to the first question have been intensively investigated over the past decades by experimental and computational neuroscientists. In this article, we review the history of ideas and receptive-field models that, for didactic purposes, we divide into three categories: hierarchical, parallel, and recurrent. Each of these models has made important contributions to our current understanding of how complex receptive fields are generated, and, as recently noticed by Kevan Martin (2002), all models have become progressively similar. In this review, we attempt to identify the barriers that keep them apart, driving us away from consensus.

What Makes a Complex Cell Complex?

Before we review the different models, it is important to further clarify what we mean by "complex cell." Although simple cells (as defined by Hubel and Wiesel 1962) have relatively similar receptive-field structures (Fig. 1), complex cells are very diverse, and the term clearly embraces different populations (Fig. 2). Here, a

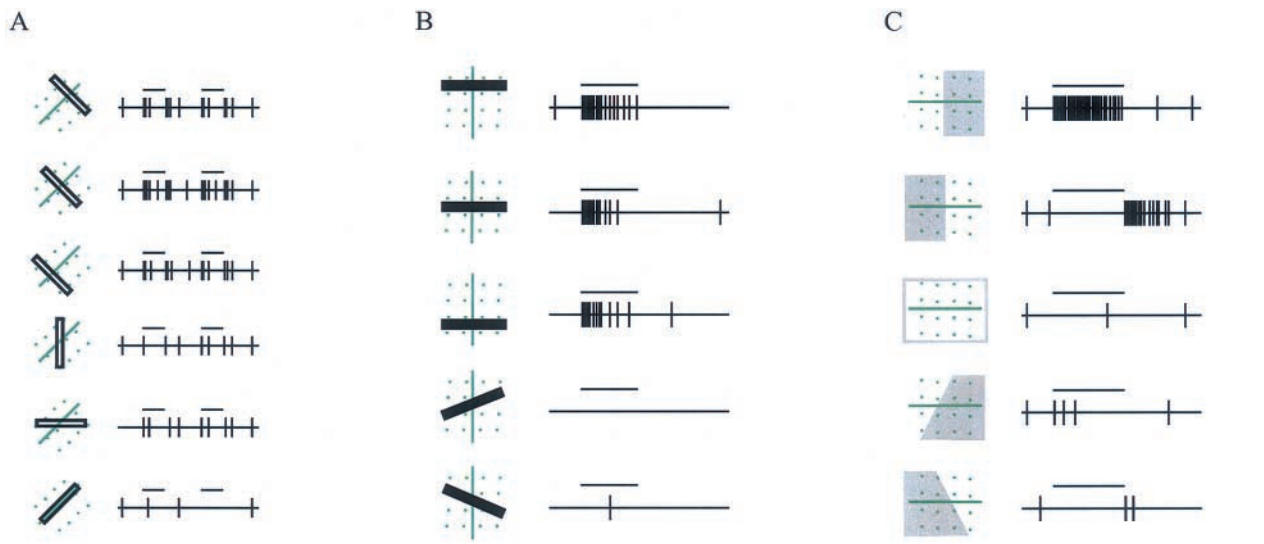

Fig. 2. Three different types of complex receptive fields (modified version of Figures 4, 6, and 7 of Hubel and Wiesel 1962). Complex cells are a very heterogeneous population. Cell *A* generates on-off responses throughout the entire receptive field. Cell *B* responds exclusively to a black horizontal bar. Cell *C* has partially separated on and off regions but the receptive field cannot be mapped with small spots of light. The green icons on the *left* represent the complex receptive field with the stimuli (flashed bars or borders) overlaid.

simple cell is defined by the four Hubel and Wiesel (1962) criteria (see above), and a complex cell is defined by exclusion: any cortical neuron that does not have a simple receptive field.

In our view, a large body of evidence indicates that simple cells are a separate population from the rest of cortical cells in cat visual cortex.

1. Simple cells are segregated in specific cortical layers (e.g., the overwhelming majority of cells in layer 4 have simple receptive fields; Hubel and Wiesel 1962; Gilbert 1977; Gilbert and Wiesel 1979; Hirsch and others 1998, 2002; Martinez and others 2002; but see Orban 1984).
2. The overwhelming majority of spiny stellate cells in cat area 17 are simple cells (Kelly and Van Essen 1974; Gilbert and Wiesel 1979; Martin and Whitteridge 1984; Hirsch and others 1998, 2002; Martinez and others 1999, 2002).
3. At least in layer 4, all simple cells have receptive-field structures consistent with a push-pull organization (e.g., within each subregion, stimuli of the opposite contrast evoke synaptic responses of the opposite sign; Ferster 1988; Hirsch and others 1998, 2002; Martinez and others 1999, 2002).
4. All simple cells receive direct geniculate input; in contrast, only a minority of complex cells does (most complex cells in the superficial layers and layer 5 do not receive direct geniculate input [e.g., Ferster and Lindstrom 1983; Martin and Whitteridge 1984; Alonso and Martinez 1998]).
5. The majority of simple cells generate responses that are roughly linear (Hubel and Wiesel 1962; Movshon and others 1978b; Skottun and others 1991a; Ferster 1994; Carandini and others 1997; Lampl and others 2001a).
6. Mechler and Ringach (2002) have recently suggested that it is necessary to show a bimodal distribution of cortical response properties to prove that simple cells are a separate class from complex cells. Although their argument is valid, statistical tools cannot be taken as the only possible source of proof. Neuroscientists do not discuss whether pyramidal cells and spiny stellate cells are different classes, and to our knowledge, nobody has shown that soma shapes are bimodally distributed.

It should also be emphasized that most of the evidence cited above comes from studies in cat visual cortex (using Hubel and Wiesel criteria for classification). Over the past two decades, we have accumulated enormous knowledge on the anatomy and physiology of cat area 17, although essential data in primate are still missing. Following are a few examples of unknowns in primate:

1. There are very few studies that measured the receptive-field structure of cells that receive direct geniculate input (as identified by electrical stimulation or cross-correlation analysis; Bullier and Henry 1980).
2. We have almost no data on intracellular physiology in vivo (Anderson and others 1993).
3. There are very few studies correlating neuronal morphology and receptive-field properties (McGuire and others 1991; Anderson and others 1993).
4. There is still no agreement on whether there is a laminar segregation of simple cells and complex

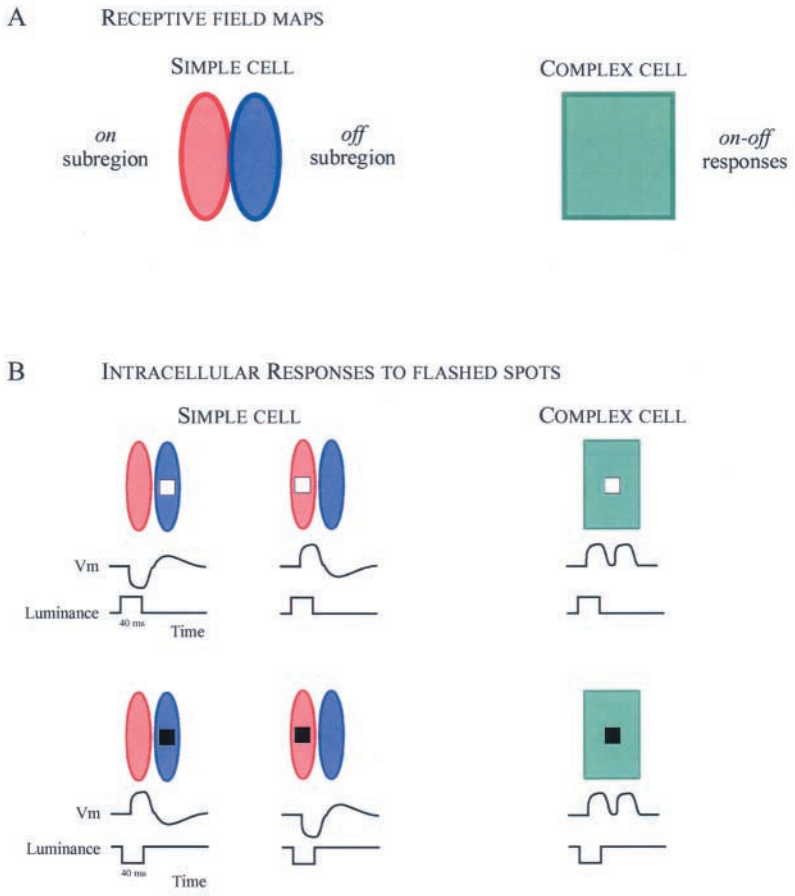

Fig. 3. Simple and complex cells differ in their receptive-field structure and their responses to static and moving stimuli. *A*, Idealized receptive-field maps of a simple cell and a complex cell. *B*, Cartoon of the intracellular responses from a simple cell to small spots (light/dark) presented on each subregion (*left* and *middle*, the traces show a push-pull arrangement of excitatory and inhibitory inputs in the simple receptive field). Complex cells lack segregated *on* and *off* regions; small spots (light/dark) evoke *on* and *off* (push-push) responses throughout the entire receptive field (*right*). *C*, Cartoon representing the responses of a simple cell and a complex cell to drifting sinusoidal gratings.

cells in layer 4C (e.g., layer 4C has mostly simple cells and unoriented cells [Hubel and Wiesel 1968; Bullier and Henry 1980]; layer 4C has simple cells and complex cells in similar proportions (Ringach and others 2002]).

Consistent with this reasoning, most of the work revised here is based on studies of cat visual cortex or computational models based on the physiology of the cat visual cortex.

Complex Receptive-Field Models

Hierarchical Models

The hierarchical model was inspired by a systematic study and comparison of the receptive-field properties in the LGN and primary visual cortex (Hubel and Wiesel 1959, 1961, 1962, 1965). According to this model, simple receptive fields are constructed from the convergence of geniculate inputs with receptive fields aligned in visual space. In turn, complex receptive fields originate from the convergence of simple cells with similar orientation preferences (Fig. 4*A*).

Hubel and Wiesel's (1962) proposal was summarized in a drawing instead of a computational model. Consequently, many of the circuitry details were not precisely described. In their drawing, the possibility that both excitatory and inhibitory inputs could be involved in the construction of cortical receptive fields was left open by writing,

> The suppression of firing . . . is presumed to be the result of withdrawal of tonic excitation. . . . One should, however, consider the possibility of direct inhibitory connections. . . . Up to the present the two mechanisms have not been distinguished, but there is no reason to think that both do not occur. (p 142–3)

Moreover, they emphasized that, "Proposals such as those of Text-figs. 19 and 20 [model drawing] are obviously tentative and should not be interpreted literally" (p 144). Accordingly, the model has evolved in the past few years to account for new experimental discoveries that, mostly, emphasize the role played by local inhibitory inputs in shaping functional response properties in the cortex (Sillito 1975, 1977, 1979; Pei and others 1994; Allison and others 1996; Crook and others 1998; Borg-Graham and others 1998; Hirsch and others 1998; Murthy and Humphrey 1999; Anderson and others 2000; Martinez and others 2002; see Ferster and Miller 2000 for review).

It is important to make a clear distinction between *conceptual* models (general hypothesis) and *computational* models (computer simulations) because they greatly differ in the specifics and the scope of their predictions. In this review, we make this distinction to avoid unnecessary confusion that could make models seem more different than they really are.

Conceptual Model. The idea of the hierarchical model was proposed four decades ago but is far from being outdated (Kandel and others 2000). New findings over the years have kept the model alive and strong. First, as predicted by Hubel and Wiesel (1962), simple cells were found to receive direct geniculate inputs (Bullier and Henry 1979; Mustari and others 1982; Ferster and Lindstrom 1983; Tanaka 1983; Martin and Whitteridge 1984; Reid and Alonso 1995; Alonso and Martinez 1998) and be overwhelmingly present in cortical layer 4

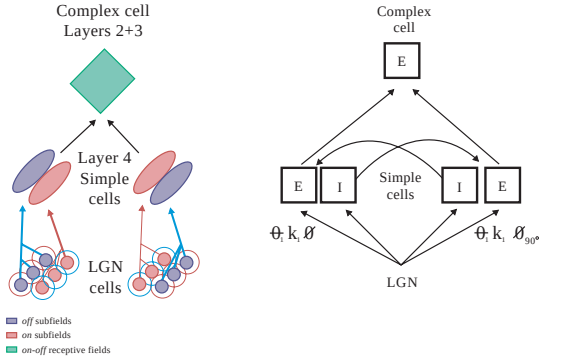

Fig. 4. Hierarchical models put forth the idea that simple cells and complex cells represent two successive stages of cortical processing. *A*, Simple receptive fields are generated in layer 4 from the convergent input of geniculate neurons with receptive fields aligned in visual space. Complex receptive fields are, in turn, generated in the superficial layers by pooling the input from layer 4 simple cells with similar orientation preferences. *B*, Newer versions of the model incorporate more elaborated intracortical circuitry. E and I stand for excitatory and inhibitory neurons, respectively. θ = orientation; k = spatial frequency, ϕ = spatial phase. Full details are given in the text.

(Kelly and Van Essen 1974; Gilbert and Wiesel 1979; Martin and Whitteridge 1984; Hirsch and others 1998, 2002; Martinez and others 1999, 2002). Second, in vivo recordings from neurons that were intracellularly labeled demonstrated that most layer 4 simple cells project to the superficial layers of the cortex (Gilbert and Wiesel 1979; Martin and Whitteridge 1984; Hirsch and others 1998; Martinez and others 1999, 2002) where most cells are complex (but see Orban 1984).

The combination of morphological and physiological data provided strong support for the hierarchical model; however, some of the very same authors that provided the data remained skeptical. For example, Martin and Whitteridge wrote in their classic 1984 paper, "Thus, although we agree that the superficial layer cells may use as their substrate the layer 4 cell receptive fields, this does not necessarily imply an S-to-C progression" (p 495; e.g., simple cells in layer 4 could target simple cells in the superficial layers).

This skepticism was based on several findings that seriously challenged the hierarchical model. First, some complex cells were found to receive direct geniculate input (Hoffmann and Stone 1971; Bullier and Henry 1979; Ferster and Lindstrom 1983; Tanaka 1983; Martin and Whitteridge 1984), indicating that complex cells were "not so different" from simple cells after all (see parallel models below). Second, several studies failed to find evidence for excitatory connections from simple cells to complex cells (Toyama and others 1981a, 1981b; Ghose and others 1994; Freeman 1996). Finally, by making injections of cobalt chloride in the LGN, Malpeli and colleagues concluded that it was possible to inactivate layer 4 simple cells without affecting the response of

complex cells in the superficial layers (Malpeli 1983; Malpeli and others 1986; Mignard and Malpeli 1991).

Some of the criticisms to the hierarchical model have been addressed over the years. By recording from vertically aligned layer 4 and layers 2+3 cells within the cortex, evidence for excitatory connections from simple cells to complex cells was found (Alonso and Martinez 1998). Furthermore, by making injections of GABA in the LGN, the complex cells described by Malpeli were found to be a minority; most layers 2+3 complex cells are not visually driven when the thalamic input to layer 4 is inactivated (Martinez and Alonso 2001; Callaway 2001). Finally, additional support for the hierarchical model came from another discipline that would become increasingly important over the years: computational neuroscience.

Computational Model. Most theoretical approaches to complex receptive-field generation were open up by the elegant "two-bar interaction" experiments of Tony Movshon and colleagues (1978a). They argued that if complex cells respond to stationary or moving forms in a way that cannot be predicted from their first-order responses (e.g., responses to single bars or small spots), it must be because complex-cell responses depend on nonlinear interactions between at least two positions in space and time. Movshon and colleagues showed that when sets of two bars were applied to complex cells, their responses displayed on and off linear subunits resembling simple-cell subregions (see also Baker and Cynader 1986; Gaska and others 1994; Szulborski and Palmer 1990; Heggelund 1981). Emerson and others (1992) extended this finding by demonstrating that the subunits of directionally selective complex-cells were also directional selective. Similarly, Ohzawa and Freeman (1986) demonstrated that the subunits of binocular complex cells were also binocular and had the same optimal disparity as the complex cell (see also Ohzawa and others 1990 1997; Anzai and others 1999).

Based on these findings, many authors modeled complex cells' responses as a square sum of simple cells with similar orientation and spatial frequency but with phases that differed by 90 degrees (e.g., Emerson and others 1992; Fleet and others 1996; Ohzawa and others 1990, 1997; Pollen and others 1989; Qian and Zhu 1997; Sakai and Tanaka 2000; Okajima and Imaoka 2001; Shams and Malsburg 2002a, 2002b). Mathematically, this can be described as a square sum of two linear operators, each characterized by a Gabor function of the same frequency but with phases 90 degrees apart from each other. Because a linear filter followed by a squaring device and then an integrator is considered an energy detector (Green and Swets 1966), these models are collectively known as energy models (Adelson and Bergen 1985). Recently, Okajima and Imaoka (2001) demonstrated that energy models render complex cells that are optimally designed from an information-theory point of view. However, as an interesting alternative, Lampl and others (2001b) suggested that the pooling of simple cell inputs may be better described by a MAX operation. That is, the strongest input determines the response of the complex cell (Riesenhuber and Poggio 1999, 2002) in which the strongest input determines the response of the complex cell. Linear or nonlinear, both proposals rest on the assumption that simple and complex receptive fields represent two successive stages of cortical processing. A cartoon version of the core circuitry predicted by energy models is shown in Figure 4*B*.

Although energy models seem to be good news for the hierarchical hypothesis, a key question remains: how is this precise synaptic connectivity achieved during development? Recently, it has been shown that a two-layer network fed with natural images can learn the phase-invariance characteristic of complex cells (Einhauser and others 2002). The model uses layers of neurons that learn their properties from competitive Hebbian algorithms (based on the relative timing of pre- and postsynaptic spikes). Interestingly, neurons in the first layer learn to respond like simple cells, and then simple cell responses are used by neurons in the second layer to learn to respond as complex cells. Similar approaches have been used in the past to model the emergence of ocular dominance and orientation columns (e.g., Miller 1996; Miller and others 1999). Hyvarinen and Hoyer (2001) also used natural images to make neurons learn to respond as complex cells in a network organization that is very similar to cat area 17 (cells clustered according to retinotopy, spatial frequency, and orientation but independently of spatial phase; see DeAngelis and others 1999). Again, in this model, simple-cell responses emerge in the first layer (see also Olshausen and Field 1996) and complex cells in the second layer.

The hierarchical model has been very successful in great part because of its appealing simplicity. The essential idea that simple cells and complex cells represent two stages in receptive-field construction is likely to remain correct even if newer models incorporate increasingly more realistic circuits (see below). However, the hierarchical has model also been justly critized. One of these criticisms is based on an experimental finding that is now unanimously accepted: Some complex cells do receive direct geniculate input. This criticism makes it necessary for the hierarchical model to embrace other ideas and evolve.

Parallel Models

Conceptual Model. Historically, the first strong evidence against the hierarchical model was the discovery that some complex cells, like simple cells, receive monosynaptic input from the thalamus (Hoffmann and Stone 1971). Based on this discovery, Hoffman and Stone proposed that both cell types, simple and complex, were generated in parallel by separate thalamocortical pathways (Hoffman and Stone 1971; Stone and others 1979; see Fig. 5*A*). But how could complex cells become nonlinear without pooling simple cell inputs? It was already

known that there were linear and nonlinear cells in the retina and the LGN (X and Y cells, respectively [Shapley and Hochstein 1975]) and that the two cell types formed separate parallel channels: X cells in the retina projected to X cells in LGN and Y retinal cells projected to Y geniculate cells (see Stone and others 1979 for review). Hoffman and Stone used this evidence to propose that linear simple cells were built from the convergence of linear X-cell inputs and nonlinear complex cells from the convergence of nonlinear Y-cell inputs.

A striking experimental result soon came in support of the new parallel model: some complex cells seemed to respond to visual stimuli that were not effective in driving simple cells (Hammond and Mackay 1975, 1977). This result was reinforced by a series of remarkable studies done by Malpeli and colleagues (Malpeli 1983; Malpeli and others 1986; Mignard and Malpeli 1991). Malpeli and others (1986) found that injections of cobalt chloride in layer A of LGN inactivated layer 4 simple cells but not layer 2+3 complex cells (Malpeli 1983); responses at layers 2+3 cells were only affected when the inactivation of LGN (layer A) was combined with large lesions in area 18 (Mignard and Malpeli 1991).

The results of Malpeli and colleagues fitted very nicely with the idea that simple and complex receptive fields were built in parallel and independently. However, parallel models soon found serious criticisms also. First, the idea of two cleanly segregated parallel channels (X $\rightarrow$ simple cell, Y $\rightarrow$ complex cell) was discarded by a large number of studies (Singer and others 1975; Bullier and Henry 1979; Tanaka 1983; Ferster and Lindstrom 1983; Martin and Whitteridge 1984). Second, some authors showed that the impact of the entire Y pathway in cat area 17 was weak (Spitzer and Hochstein 1987; Ferster 1990a, 1990b; Burke and others 1992). Third, Skottun and colleagues (1988, 1991b) presented evidence against the Hammond and Mackay (1975, 1977) findings showing that most simple cells do respond to the stimuli that drive complex cells (but see Hammond 1991). Finally, with the availability of new techniques, Malpeli's results were found to be correct only for a minority of cells in the superficial layers (Martinez and Alonso 2001; Callaway 2001)—most layer 2+3 complex cells cannot be driven from within the classical receptive field when the main thalamic input to layer 4 is inactivated (see also Rivadulla and Sur 2000).

Regardless of the specifics, the main idea of parallel models remained valid: at least some complex receptive fields could be constructed from direct geniculate inputs. However, an important question was still without an answer. How can a complex cell be orientation selective if its main inputs are nonoriented and its receptive field does not have elongated subregions? Once again, computational neuroscience came to the rescue (Mel and others 1998).

Computational Model. Mel and colleagues (1998) clearly showed that a cortical neuron could receive input from geniculate cells with overlapping on and off centers and still produce phase-invariant orientation tuning. In this model, the arrangement of geniculate inputs, originated by a Hebbian developmental rule, produces functional subunits scattered across the dendrites. These functional subunits respond more efficiently to specific line orientations by activating local voltage-dependent excitatory currents (see also Spitzer and Hochstein 1985, 1988; see Fig. 5*B*, *left*).

In a similar line of thinking, a more recent parallel model combined thalamic and cortical inputs to generate linear and nonlinear visual responses (Tao and others 2001, forthcoming; see also Wielaard and others 2001). In Tao and others' (2001, forthcoming) model, nonlinear responses originate in cells that receive weak geniculate input and linear responses in cells that receive strong geniculate input (Fig. 5*B*, *right*). In agreement with Tao and others model, complex cells (that generate nonlinear responses) are abundant in cortical layers that receive the weakest thalamic input, and the converse is true for simple cells that generate linear responses. Also consistent with this model, a large number of complex cells in cat primary visual cortex do not receive measurable direct geniculate input (e.g., Ferster and Lindstrom 1983; Martin and Whitteridge 1984; Alonso and Martinez 1998).

Simple cells and complex cells are far from being two parallel cortical pathways in the same way that X and Y cells are parallel thalamic pathways. However, the idea that some complex receptive fields can be generated at least in part by direct thalamic inputs is likely to be correct. In support of this idea, recent computational models have provided plausible mechanisms to generate complex receptive fields directly from geniculate inputs (Spitzer and Hochstein 1988; Mel and others 1998; Tao and others 2001). Some of these models (Tao and others 2001, forthcoming) also emphasize another possible key player in the construction of complex receptive fields: the intracortical circuitry.

Recurrent Models

Conceptual Model. Perhaps the most widely used argument against any thalamocortical model (hierarchical or parallel) is the nature of the cortical circuit itself. The number of geniculate synapses is only a small fraction of the total excitatory synapses made onto cortical cells (LeVay and Gilbert 1976; Kisvarday and others 1986; Peters and Payne 1993; Ahmed and others 1994). Therefore, cortical responses should be determined mostly by cortical inputs and not by thalamic inputs (Martin 2002).

Douglas and Martin (Douglas and others 1989; Douglas and Martin 1991; see also Douglas and others 1995) developed the conceptual frame for a new type of model that is known as a recurrent model (Fig. 6*A*). Their proposal rests on three key assumptions:

1. Thalamic excitatory inputs are weak because geniculate synapses account for less than 10%

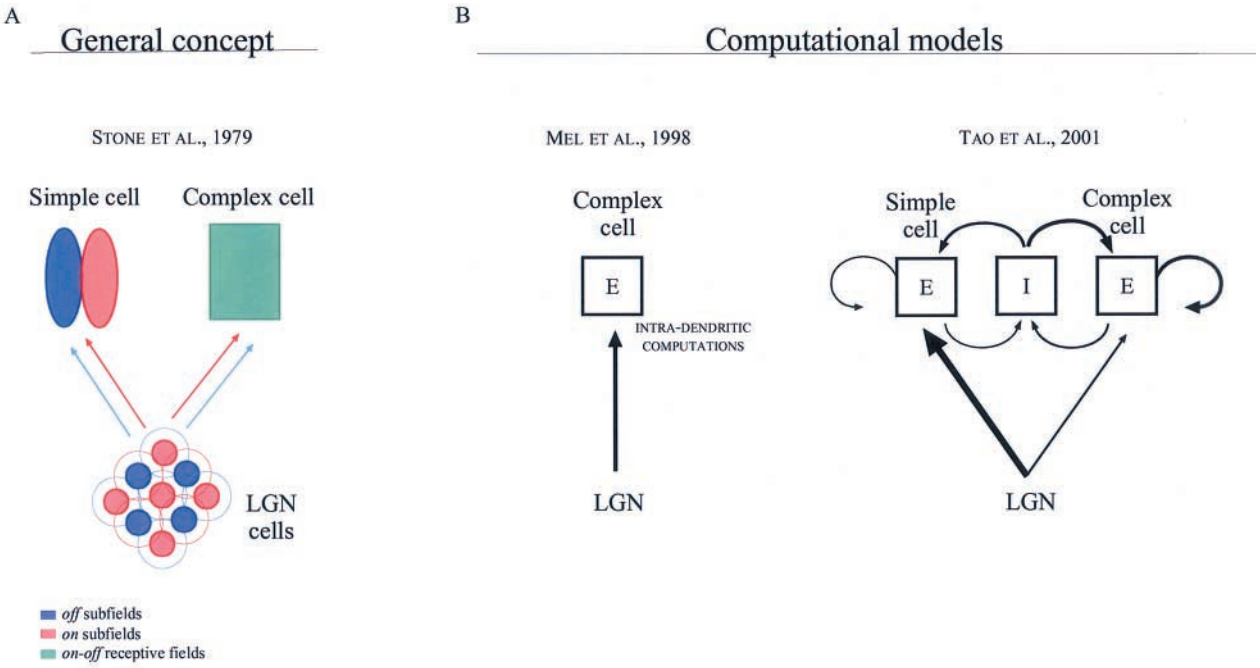

Fig. 5. *A*, Parallel models advocate that simple cells and complex cells are both created in parallel from different thalamocortical pathways. *B*, The most recent versions of parallel models come in two sorts that exploit the preponderance of the single cells over the circuit (Mel and others 1998) or vice versa (Tao and others 2001, forthcoming). Mel and others' (1998) proposal generates complex-cell responses that are orientation selective through specific intradendritic computations. Tao and others' scheme obtains simple cells and complex cells by modulating the gain of the thalamocortical and corticocortical inputs. The line thickness represents the strength of the connection. E and I stand for excitatory and inhibitory neurons, respectively. See the text for details.

of the total excitatory synapses onto layer 4 cells (Kisvarday and others 1986; Peters and Payne 1993; Ahmed and others 1994).
2. Cortical excitation from neighboring neurons is very strong and serves to amplify the weak thalamic input.
3. Weak cortical inhibition controls the gain of the "cortical amplifier" preventing runaway excitation and adjusting the network performance to changes in visual stimulation.

In initial versions of recurrent models, the predominance of intracortical excitation over intracortical inhibition was emphasized because 80% of the synapses on cortical cells are excitatory (Braitenberg and Schuz 1991) and because intracellular measurements failed to find evidence for strong inhibition in response to visual stimuli (Berman and others 1991; Douglas and others 1991; Ferster 1988). However, our view of cortical inhibition has changed over the years (Sillito 1975, 1977, 1979; Pei and others 1994; Allison and others 1996; Crook and others 1998; Borg-Graham and others 1998; Hirsch and others 1998; Murthy and Humphrey 1999; Anderson and others 2000; Martinez and others 2002), and, accordingly, new recurrent models are incorporating stronger inhibitory inputs (Somers and others 1995; Ben-Yishai and others 1995; reviewed in Sompolinski and Shapley 1997).

Recurrent models claim to be more faithful to the structure of the cortical network than hierarchical models, and, to some extent, they are. Cortical neurons (simple or complex) receive abundant input from other cortical neurons both inhibitory and excitatory (e.g., Ahmed and others 1994; Callaway 1998; Fitzpatrick 1996; Braitenberg and Schuz 1998). Therefore, any model that incorporates more elaborate intracortical circuitry is in some way closer to the reality than are models that use primarily one type of input. That being said, the idea that geniculate inputs are much weaker than cortical inputs is unlikely to be correct. Although the number of geniculate synapses is a small percentage of the total excitatory synapses made on cortical cells (5% to 25% depending on which study is cited; e.g., Peters and Payne 1993; Levay and Gilbert 1976), geniculocortical connections have many other features that make them strong.

1. Geniculate synapses are bigger than cortical synapses (e.g., Ahmed and others 1994).
2. Geniculate synapses are located proximally in the dendrites, whereas excitatory cortical synapses tend to be located more distally (e.g., Ahmed and others 1994).
3. Geniculate synapses have more release sites than cortical synapses do (Gil and others 1999).
4. Thalamocortical excitatory postsynaptic potentials (EPSPs) are likely to be larger than cortico-

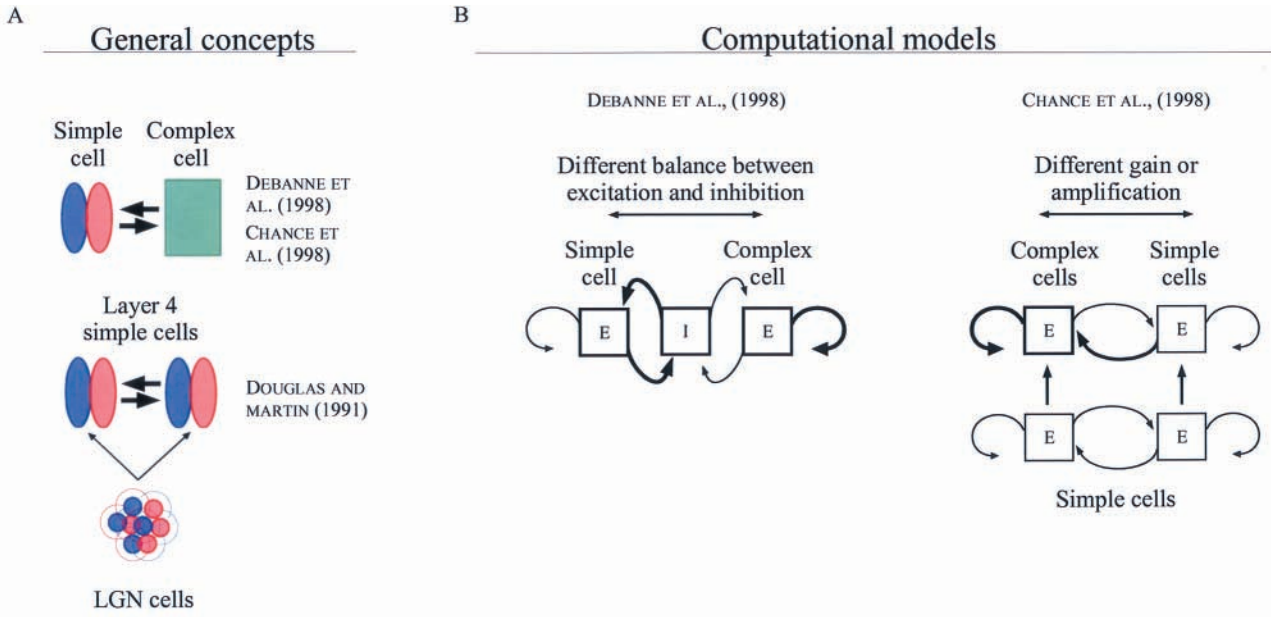

Fig. 6. Recurrent models changed the focus of attention from single cells to networks of cortical connections. *A*, Most models are inspired by Douglas and Martin's (1991) canonical microcircuit that emphasized the predominance of the input from intracortical connections over the thalamocortical input (*bottom*). The most recent addenda to the family of recurrent models are the so-called state-dependent models. State-dependent models propose that simple and complex cells originate from the same cortical circuit operating at different amplification gains (*top*). *B*, Debanne and others' (1998) model (*left*) emphasizes the balance between excitatory and inhibitory inputs as the origin of different cortical receptive fields. Chance and others' (1999) model (*right*) emphasizes the strength of local excitatory connections within the superficial layers of the cortex. The line thickness represents the strength of the connection. E and I stand for excitatory and inhibitory neurons, respectively. A more detailed explanation is given in the text.

cortical EPSPs (Gil and others 1999; Stratford and others 1996).

5. Geniculate cells have generally higher firing rates than cortical cells do (e.g., Bullier and others 1982).
6. Many of the geniculate cells that converge on the same cortical cell generate precise and strong synchronous firing (Alonso and others 1996; such precise *and* strong synchrony is not found within the cortex).
7. The inactivation of a tiny region of LGN is enough to silence the activity of most cortical cells in layers 4 and 2+3 (Martinez and Alonso 2001; see also Malpeli 1983). The inactivation of a similarly tiny region in the cortex has only a subtle effect on the response of a cortical cell (Bolz and Gilbert 1986; Grieve and Sillito 1995).

In general, it is risky to estimate the strength of a given pathway based solely on the number of synaptic contacts. Indeed, if this type of assumption were correct, the geniculate receptive fields should resemble more cortical receptive fields than retinal receptive fields, and this is clearly not the case (Hubel and Wiesel 1961; Cleland and others 1971; Usrey and others 1998; geniculate cells receive 7% of excitatory synapses from the retina and about 40% from the cortex; Van Horn and others 2000).

Although the relative contribution of thalamic and cortical inputs to cortical receptive-field generation is still a matter of debate (Ferster and Miller 2000; Martin 2002), recent models have chosen to focus on a different question: how do recurrent networks generate simple and complex receptive fields? This focus is the seed of a new bold idea: Simple cells and complex cells may not be different cell types after all (e.g., Mechler and Ringach 2002; Abbott and Chance 2002). Supporting this proposal, the work of Debanne and others (1998) showed that the relative strength of cortical *on-off* responses could be modified by pairing visual stimuli with current injections. In theory, if we can modify the strength of *on-off* responses, we should also be able to transform a simple cell into a complex cell and vice versa. In reality, changes in the strength of *on-off* responses are rare in the adult (four cases in Debanne and others 1998), and they are usually subtle. More pronounced changes are generated when the precise balance between cortical excitation and inhibition is manipulated (Sillito 1975; Nelson and others 1994; Rivadulla and others 2001). However, these changes are not inconsistent with a hierarchical model. In a hierarchical model, a stimulus presented in an *on* subregion of a simple cell should generate on responses because it activates mostly the receptive-field centers from on-center geniculate cells. In the absence of cortical inhibition, the same stimulus should also activate *off* responses that originate from two main different sources:

1. the *off* surrounds from on-center geniculate cells that overlap the *on* subregion and
2. the off-center borders from off-center geniculate cells that overlap the adjacent *off* subregion (see Alonso and others 2001 for multiple examples).

The fact that *on-off* responses are observed in simple cells after blocking inhibition is exactly what we would expect from a hierarchical model and the properties of thalamocortical connections (Reid and Alonso 1995; Alonso and others 2001). Also as expected from a hierarchical model, when *on-off* responses are quantitatively mapped and averaged, simple receptive fields still look "very simple" even if cortical inhibition is blocked (Murthy and Humphrey 1999; see also Rivadulla and others 2001).

The idea that simple cells and complex cells are the same cell type (or generated by the same basic circuit) seems hard to accept based on a large body of literature in cat visual cortex (see the introduction of this review). Recent anatomical and physiological data also suggest that thalamic inputs are not weaker than cortical inputs (see above). In spite of these criticisms, the "intracortical emphasis" that emerged from recurrent models is important and is leading to powerful computational models.

Computational Model. Recurrent models moved the focus of attention from thalamo-cortical connections to networks of cortical neurons that are reciprocally connected (Martin 2002; Nelson 2002). With differences regarding mainly the role of inhibitory inputs, recurrent models used networks of cortical neurons to explain the emergence of orientation and direction selectivity in the cortex (Somers and others 1995; Ben-Yishai and others 1995; reviewed in Sompolinski and Shapley 1997). Somers and others (1995) developed a recurrent model that was especially successful at explaining a large body of experimental data. In this model, cortical excitation links cells with similar orientation preferences, whereas cortical inhibition links cells with a broader range of orientations. The model was challenged by recent data demonstrating that the tuning for excitatory and inhibitory inputs in layers 4 and 2+3 is similar (Nelson and others 1994; Anderson and others 2000; Martinez and others 2002). Also, against the model predictions, local excitatory connections have been found to extend farther laterally than inhibitory connections do (Roerig and Chen 2001; Yousef and others 1999, 2001; Buzas and others 2001; see Carandini and Ringach 1997 for other criticisms). Another recurrent model was proposed by Ben Yishai and others (1995) to generate contrast invariant orientation tuning. In this model, feedback connections are so strong that the cortical network becomes an orientation attractor (i.e., the output of the network is determined simply by the balance between excitation and inhibition independently of the properties of the input). This type of orientation attractors (see also Tsodyks 1999) has been challenged by data showing that cortical responses to oriented gratings do depend on the spatial frequency of the input (Vidyasagar and Siguenza 1985; Webster and De Valois 1985; Jones and others 1987; Hammond and Pomfrett 1990).

The models of Somers and others (1995) and Ben-Yishai and others (1995) did not address specifically the generation of complex receptive fields; however, they were very influential in setting the basis for future recurrent models that did address the issue. Debanne and others (1998) used recurrent circuits to generate simple and complex cells. In their model, both cell types receive input from geniculate cells with overlapping on- and off-receptive-field centers (and other cortical cells). Simple cells are generated when the cortical inhibitory inputs are strong enough to impose a bias toward either *on* or *off* responses. Complex cells are generated when inhibition is reduced to unmask *on-off* responses. Debanne and others' model explains why simple cells generate *on-off* responses when cortical inhibition is pharmacologically blocked (Sillito 1975; Ramoa and others 1988; Nelson and others 1994; Shulz and others 1993). However, as discussed earlier, these findings can also be explained with a hierarchical model (see above). Moreover, some of the details of Debanne and others' model are at odds with experimental data. For example, not all smooth inhibitory neurons in layer 4 have S1 receptive fields (e.g., Gilbert and Wiesel 1979; Martin and others 1983; Kisvarday and others 1985, 1987; Azouz and others 1997; Hirsch and others 2000), and the model fails to explain why many layer 4 cells have receptive fields with push-pull organization (Ferster 1988; Hirsch and others 1998; Martinez and others 1999; Anderson and others 2000).

Although Debanne and others' (1998) model made a bridge between parallel and recurrent models (Tao and others 2001, forthcoming are also in this category), the model of Chance and others (1999) abandoned this bridge to adopt elements from a hierarchical organization. As in a hierarchical model, Chance and others use a first layer of simple cells (equivalent to layer 4) that feeds into another layer of cells with similar orientation preferences (equivalent to layers 2+3). Chance and others model departs from a hierarchical organization by making the connections from layer 4 to layers 2+3 weaker than the connections within layers 2+3 and by making the vertical connections link cells with similar spatial phases, whereas strong recurrent connections link cells with different spatial phase (Fig. 6*B*, *right*). Although previous recurrent models used cortical amplification to generate orientation tuning and direction selectivity (Douglas and others 1995; Ben-Yishai and others 1995; Somers and others 1995), Chance et al. used cortical amplification to generate nonlinear responses. In support of Chance et al.'s model, it has been suggested that simple cells and complex cells cannot be separated into two different populations (Mechler and Ringach 2002; Abbott and Chance 2002) and that complex cells can behave like simple cells when the balance between excitation and inhibition changes (Sillito 1975; Nelson and

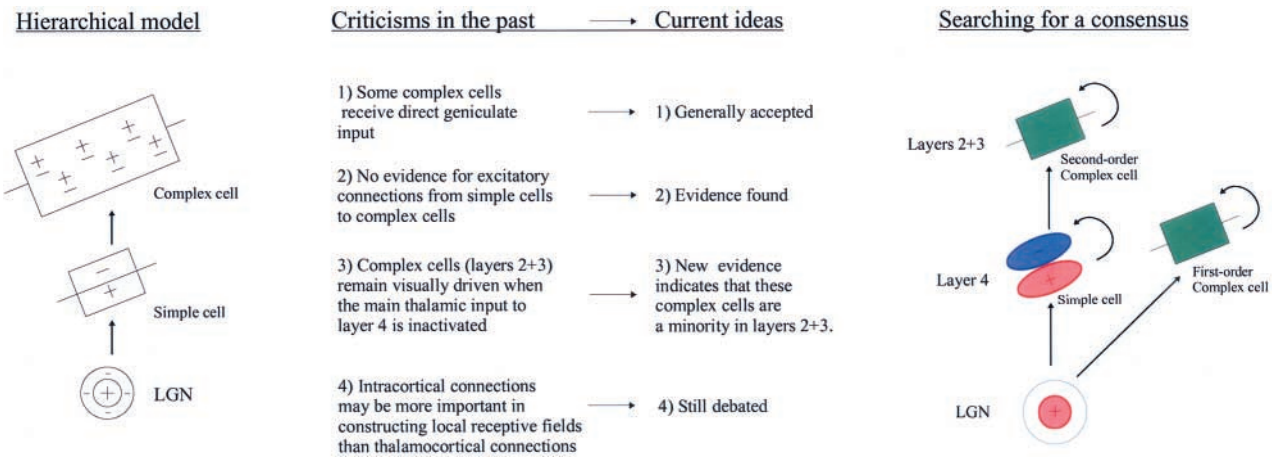

Fig. 7. Hubel and Wiesel's (1962) hierarchical model (*left*) has been extensively challenged over the years. Two main criticisms (*middle*) have propelled the emergence of alternative frameworks that accounted for the new experimental findings. Here, we propose a new circuit diagram for cat primary visual cortex (*right*) that embraces all current ideas of hierarchical, parallel, and recurrent models. Layer 4 simple cells and superficial complex cells form two successive stages in receptive-field construction. In addition, some cells in layer 4 have complex receptive fields derived, in parallel, from direct geniculate inputs. At each layer, local intracortical circuits (both excitatory and inhibitory) modulate the gain of the network. See the text for a detailed explanation.

others 1994; Shulz and others 1993; Rivadulla and others 2001). We have already criticized these two suggestions in detail (see above). Here, we could also add that the specificity of vertical connections (layer 4 → layers 2+3) for spatial phase is difficult to reconcile with the lack of clustering for this property (DeAngelis and others 1999) and with the finding that some layer 4 simple cells with different spatial phases do converge on the same superficial complex cells (Alonso and Martinez, unpublished observations). Furthermore, it has been recently shown that the synaptic connections between layer 4 and layers 2+3 are among the strongest connections in the cortical network (stronger than local horizontal connections between cells in the superficial layers; Feldmeyer and others 2002).

As with any model, the circuitry predicted by recurrent models will have to pass the test of time and experimental data. In any case, the idea that response linearity could be modulated by the gain of the cortical network is interesting and may prove to be correct in some extent. New experimental data will tell us whether this modulation exists, how strong it is, and which cortical layers it involves (see Rivadulla and others 2001).

Searching for a Consensus

Of all the criticisms that the hierarchical model has received over the years, two have remained sufficiently strong as to promote the birth of new conceptual frameworks (Fig. 7). First, as suggested by parallel models, some complex receptive fields can be generated at least in part from direct geniculate inputs. Second, as suggested by recurrent models, cortical cells are heavily interconnected, and their responses are likely to be modulated by recurrent connections. In spite of these criticisms, the idea that simple cells and complex cells represent two stages in receptive-field construction is likely to remain correct even if models evolve and become more precise. Eventually, all models will mix to adopt elements from each other and finally converge into one. For example, Troyer and others (1998) introduced strong inhibitory inputs and more elaborate local connectivity in their hierarchical model of orientation selectivity in cat layer 4 (see also Miller and others 2001 for review). Chance and others (1998) adopted a hierarchical organization by using a first layer of simple cells that feeds into a second layer of cells with more elaborated responses (simple or complex depending on network gain). Also, Martin (2002) has recently described his recurrent model as an "alternative hierarchy" (the word *hierarchy* was not used in the original description of recurrent or canonical microcircuits [e.g., Douglas and Martin 1991]). Finally, Tao and others (2001, forthcoming) used strong/weak geniculate inputs to model simple cells/complex cells, making simple cells "closer" to their thalamic inputs than complex cells are (in previous parallel models, complex cells received strong geniculate inputs, e.g., Mel and others 1998).

It seems clear that each line of models—hierarchical, parallel, or recurrent—can no longer be considered in isolation. Here, we propose a circuit diagram to integrate the main ideas/models discussed above (Fig. 7). The diagram cannot be easily ascribed to any of the main models, although some could see it as clearly hierarchical, others as clearly parallel, and still others as clearly recurrent. The diagram gives a general framework of the main connections as a function of cortical layers (layer 4, 2+3) and receptive-field properties (simple/complex). Layer 4 simple cells and layers 2+3 complex cells are two stages in receptive-field construction as proposed by the hierarchical model (Hubel and Wiesel 1962; Adelson and Bergen 1985) and some recurrent models (Chance and

others 1998). At the same time, a subpopulation of complex cells has their receptive fields constructed from direct geniculate inputs (Spitzer and Hochstein 1988; Mel and others 1998; Tao and others 2001, forthcoming). Finally, at each layer, local intracortical circuits (both excitatory and inhibitory) modulate the gain of the cortical network (Debanne and others 1998; Chance and others 1998; Tao and others 2001, forthcoming) generating different results depending on the layer. Within layers 2+3, response linearity would be modulated in some extent by local connections as proposed by recurrent models; within layer 4 (simple cells with push-pull receptive fields), response linearity would not be so easily modulated. Forty years had gone by since Hubel and Wiesel proposed the hierarchical model; one wonders how many times the pendulum will swing back and forth before it finally sets at a standstill and we reach a consensus on how complex receptive fields are generated.

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