LINKING VISUAL CORTICAL DEVELOPMENT TO VISUAL PERCEPTION

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Abstract

A central question in cognitive science and neuroscience concerns how the visual cortex autonomously develops, stabilizes its own development, and then gives rise to visual perception in the adult. Much evidence suggests that the visual cortex generates representations of perceptual boundaries and surfaces. The present article focuses on how the visual cortex develops the circuitry that generates perceptual boundaries. Boundary formation is also known as perceptual grouping, or the binding problem. Developing cortical circuits may be refined by visual experience. The model clarifies how developing circuits protect themselves against being catastrophically eroded by fluctuations in visual inputs. Remarkably, the processes which stabilize development in the infant lead to properties in the adult of perceptual grouping, attention, and learning. Thus, the laws of adult perception seem to be strongly constrained by stability constraints on infant development. This modeling perspective opens a path towards unifying three fields: infant cortical development, adult cortical neurophysiology and anatomy, and adult visual psychophysics. The model further clarifies why visual cortex, indeed all neocortex, is organized into layered circuits. It hereby contributes to an understanding of how the laminar organization of neocortex supports biological intelligence.

Introduction

A central question in cognitive science and neuroscience concerns how the visual cortex autonomously develops, stabilizes its own development, and then gives rise to visual perception in the adult. What is the link between processes of development in the infant and processes of perception and learning in the adult? What are the functional units that determine perception in the adult, and how do developmental processes give rise to these units?

During the past twenty years, a large body of experimental and theoretical evidence has lent accumulating support to the idea that the visual cortex devotes substantial processing resources to generating three-dimensional representations of perceptual boundaries and surfaces, notably representations that can separate figures from their backgrounds and complete the representations of partially occluded objects. It has been proposed that these boundary and surface representations are formed in the interblob and blob streams, respectively, that project between cortical areas V1 to V4 (see Grossberg (1994) for a review). These representations, in turn, then project to higher levels of the brain, notably inferotemporal cortex, where they are categorized, or unitized, into object representations. All of these cortical areas and their representations are, moreover, linked with each other through feedback pathways.

Perhaps the earliest modeling studies to propose that and how boundaries and surfaces are computed in these cortical streams were provided by the author and his colleagues; e.g., Cohen and Grossberg (1984), Grossberg (1984, 1987a, 1987b), Grossberg and Mingolla (1985a, 1985b), Grossberg and Todorovic (1988). Since that time many experiments have lent support to this hypothesis (see Grossberg (1994, 1997) for reviews). A great deal of theoretical progress has also been made towards further characterizing these boundary and surface processes; e.g., Douglas *et al*. (1995), Finkel and Edelman (1989), Grossberg (1994, 1997), Grossberg and Kelly (1999), Grossberg and McLoughlin (1997), Grossberg, Mingolla, and Hwang (2001), Grossberg and Pessoa (1998), Heitger *et al*. (1998), Kelly and Grossberg (2000), Li (1998), McLoughlin and Grossberg (1998), Mumford (1992), Pessoa, Mingolla, and Neumann (1998), Somers *et al*. (1998), Stemmler, Usher and Niebur (1995), and Ullman (1995). Another parallel processing

stream, through cortical area MT, helps to compute object motion and cues useful for visual navigation. Motion processing will not be further discussed in this article. Relevant theoretical progress towards theoretically characterizing object motion and navigational processes can be found in Grossberg, Mingolla, and Viswanathan (2001) and Grossberg, Mingolla, and Pack (1999).

The present article will focus on aspects of how the visual cortex generates perceptual boundaries. This process is also known as *perceptual grouping*, or the *binding problem*. The present summary will not discuss three-dimensional boundary formation or the figure-ground problem. It will, instead, focus on some of the fundamental perceptual grouping mechanisms on which these three-dimensional processes are based. These perceptual grouping processes are known to play an important role in how infants perceive the world. For example, neonates appear to perceive a partly occluded object as disjoint parts. The ability to process these fragments as coherent objects via perceptual grouping develops rapidly within the first two to four months of life (Kellman and Spelke, 1983; Johnson and Aslin, 1996; Johnson, 2001).

A neural model is here reviewed of how such perceptual grouping circuits develop in the visual cortex. Many experiments over the past thirty years have illustrated how properties of cortical circuits may be influenced by visual experience. Whenever developing circuits may be "taught" by environmental inputs, a key concern is how these circuits protect themselves against being washed away by fluctuations in these inputs. The same problem arises during adult learning. This is often called the problem of "catastrophic forgetting." Catastrophic forgetting does not refer to the desirable refinement and adjustment of circuits in response to environmental fluctuations. Rather, it acknowledges that such fluctuations can cause an undesirable collapse in useful circuit properties in incompletely realized neural models. Most neural models, such as the popular back propagation model (see Grossberg (1988) for a review) do experience catastrophic forgetting because their mechanisms include biologically unrealistic elements.

The present model proposes neural mechanisms that enable developing cortical circuits to stabilize themselves using properties of their self-organized circuit interactions. Remarkably, the same processes which help to stabilize development in the infant lead to properties in the adult of perceptual grouping, attention, and learning. Many useful implications follow from this observation. One is that the laws of adult perception are strongly constrained by stability constraints on infant development. Because of this link between infant development and adult perception, the article discusses adult perceptual properties in some detail, since these are the targets to which infant development is aimed. Another implication of the model is that the visual cortex is not merely a bottom-up filtering device, as was suggested in the classical Nobel-prize winning work of Hubel and Weisel (1977). Rather, even early stages of visual cortical processing actively carry out perceptual grouping, attentional selection, and adaptive reorganization of circuitry in response to changing environmental conditions.

The model further clarifies why visual cortex, indeed all neocortex, is organized into layered circuits. It hereby contributes to an understanding of how the laminar organization of neocortex supports biological intelligence. This laminar organization is shown to realize at least three interacting processes: (1) the developmental and learning processes whereby the cortex shapes its circuits to match environmental constraints in a *stable* way through time; (2) the binding process whereby cortex groups distributed data into coherent object representations that remain sensitive to analog properties of the environment; (3) the attentional process whereby cortex selectively processes important events. As noted above, the model proposes that the mechanisms which achieve property (1) imply properties of (2) and (3). The model also opens a

path towards understanding how variations and specializations of these processes operate in other types of neocortex. This modeling perspective opens a path towards unifying three fields: infant cortical development, adult cortical neurophysiology and anatomy, and adult visual psychophysics.

The model is called a LAMINART model because it clarifies how mechanisms of Adaptive Resonance Theory, or ART, can be realized within identified laminar cortical circuits. Earlier ART models were devoted to understanding how bottom-up and top-down cortical interactions work together for the control of cortical development, learning, perception, and cognition. Although these studies successfully explained and predicted a variety of behavioral and brain data, they did not show how these processes are realized within laminar cortical circuits. Grossberg (1999b) reviews some of these ART concepts and some of the data that they explain. The LAMINART model extends these results by proposing how bottom-up, top-down, and *horizontal* cortical circuits work together in *laminar* circuits, and how they realize processes of development, learning, grouping, and attention. LAMINART hereby unifies concepts about ART learning and attention with concepts about perceptual grouping. This innovation was introduced in Grossberg, Mingolla, and Ross (1997) and Grossberg (1999a).

Subsequent work on the LAMINART model has clarifies how excitatory and inhibitory connections in the cortex can develop stably by maintaining a balance between excitation and inhibition in multiple cortical circuits (Grossberg and Williamson, 2001). It is known, for example (see below for references), that long-range excitatory horizontal connections between pyramidal cells in layer 2/3 of visual cortical areas play an important role in perceptual grouping. The model proposes how development enables the strength of these long-range excitatory horizontal connections to be (approximately) balanced against the strength of short-range disynaptic inhibitory interneurons which input to the same target pyramidal cells. These balanced connections are proposed to realize properties of perceptual grouping in the adult. Figure 1 summarizes how these balanced connections enable perceptual groupings to form between pairs, or greater numbers, of inducers in an image (the case of a Kanizsa square is here illustrated), but not outwardly from a single inducer, which would fill the percept with spurious boundaries.

The model also proposes that development enables the strength of excitatory connections from layer 6-to-4 is be balanced against those of inhibitory interneuronal connections to the same layer 4 cells; see Figure 2. Due to this balance, the net excitatory effect of layer 6 on layer 4 is proposed to be modulatory. These (approximately) balanced excitatory and inhibitory connections exist within the on-center of an on-center off-surround network from layer 6-to-4. The off-surround cells can strongly inhibit their target cells, even though the on-center cells can only provide excitatory modulation to their target cells.

The model proposes how this layer 6-to-4 circuit functions as a "selection circuit" because it can help to select the groupings that enter conscious attention. Grouping cells in layer 2/3 can activate the layer 6-to-4 selection circuit via excitatory connections from layer 2/3 to layer 6; see Figure 3a. When ambiguous and complex scenes are being processed, many possible groupings can start to form using the horizontal connections within layer 2/3. The selection circuit enables the strongest groupings to inhibit weaker groupings via the 6-to-4 off-surrounds of the strongest groupings.

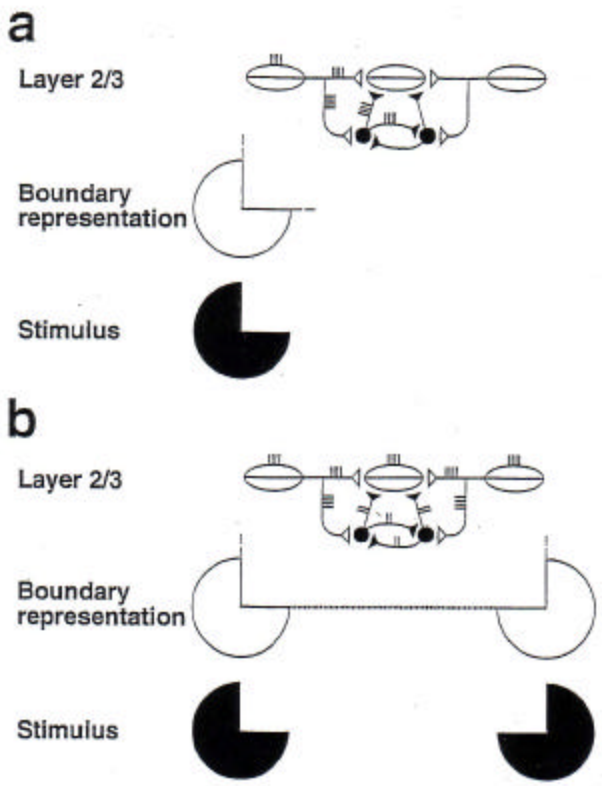

Figure 1. Schematic of the boundary grouping circuit in layer 2/3. Pyramidal cells with collinear, coaxial receptive fields (shown as ovals) excite each other via long-range horizontal axons (Bosking, *et al.* 1997; Schmidt, *et al.* 1997a), which also give rise to short-range, disynaptic inhibition via pools of interneurons, shown filled-in black (McGuire, *et al*. 1991). This balance of excitation and inhibition helps to implement what we call the *bipole property.* **(a)** Illustration of how horizontal input coming in from just one side is insufficient ot cause above-threshold excitation in a pyramidal cell (henceforth referred to as the target) whose receptive field does not itself receive any bottom-up input. The inducing stimulus (e.g. a Kanizsa 'pacman', shown here) excites the oriented receptive fields of layer 2/3 cells, which send out long-range horizontal excitation onto the target pyramidal. However, this excitation brings with it a commensurate amount of disynaptic inhibition. This creates a case of 'one against one', and the target pyramidal is not excited above-threshold. The boundary representation of the solitary pacman inducer produces only weak, sub-threshold collinear extensions (thin dashed lines). **(b)** When two collinearly aligned induced stimuli are present, one on each side of the target pyramidal's receptive field, a boundary grouping can form. Long-range excitatory inputs fall onto the cell from both sides, and summate. However, these inputs fall onto a shared pool of inhibitory interneurons, which, as well as inhibiting the target pyramidal, also inhibit each other (Tamas, *et al.* 1998), thus normalizing the total amount of inhibition emanating from the interneuron pool, without any individual interneuron saturating. This summating excitation and normalizing inhibition together create a case of 'two-against-one', and the target pyramidal is excited above-threshold. This process occurs along the whole boundary grouping, which thereby becomes represented by a line of suprathreshold layer 2/3 cells (thick dotted line). Boundary strength scales in a graded analog manner with the strength of the inducing signals. [Reproduced with permission from Grossberg and Raizada (2000)].

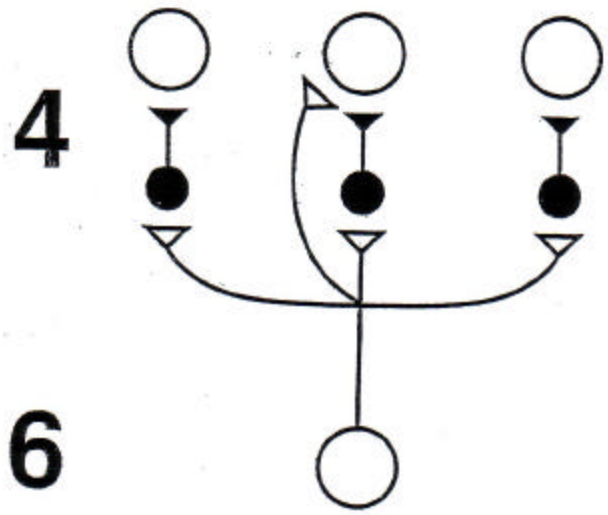

Figure 2. Schematic of the modulatory layer 6? layer 4 on-center off-surround path. Pyramidal cells in layer 6 give on-center excitation to layer 4 spiny stellates in the column above them, but also make medium-range connections onto layer 4 inhibitory interneurons, shown filled-in black (McGuire, *et al.* 1984; Ahmed, *et al.* 1997). These interneurons synapse onto the spiny stellates, creating a 6? 4 off-surround, and also onto each other (connection not illustrated), thereby helping to normalize the total amount of inhibition (Ahmed, *et al.* 1997). Note that the 6? 4 off-surround inhibition spatially overlaps with the excitatory on-center, with the consequence that the 6? 4 excitation is inhibited down into being modulatory, *i.e.* priming or subthreshold (Stratford, *et al.* 1996: Callaway, 1998b). [Reproduced with permission from Grossberg and Raizada (2000)].

The model clarifies how top-down attention can bias this selection process, and thereby influence which groupings will enter conscious perception. In particular, it is proposed that top-town attentional signals from higher cortical areas, such as area V2, can also activate the layer 6-to-4 on-center off-surround network; see Figure 3b. Because both grouping and attention share the same selection circuit, this anatomical arrangement enables attention to influence which groupings are perceived. Attention hereby can modulate, or sensitize, cells in the attentional on-center, without fully activating them, because the excitatory and inhibitory signals in the on-center are balanced. Attention can also inhibit cells in the off-surround. In this way, attention can shift the excitatory/inhibitory balance that determines which groupings will enter consciousness. A dramatic example of this influence occurs when attention that is caste on one part of an object can flow selectively along the perceptual groupings that define the entire object. Roelfsema *et al.* (1998) have discovered such a flow of attention along a perceptual grouping during their neurophysiological recordings in macaque area V1. Because of this property, both infants and adults can focus their attention selectively upon whole objects, rather than just random subsets of visual features.

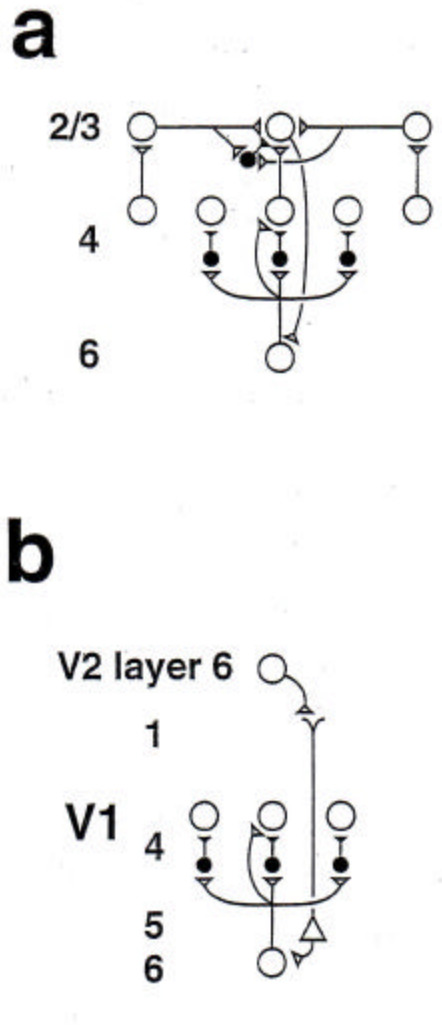

Figure 3.
(a) Connecting the 6? 4 on-center off-surround to the layer 2/3 grouping circuit: like-oriented layer 4 simple cells with opposite contrast polarities compete (not shown) before generating half-wave rectified outputs that converge onto layer 2/3 complex cells in the column above them. Like attentional signals from higher cortex, groupings which form within layer 2/3 also send activation into the *folded feedback* path, to enhance their own positions in layer 4 beneath them via the 6? 4 on-center, and to suppress input to other groupings via the 6? 4 off-surround. There exist direct layer 2/3? 6 connections in macaque V1, as well as indirect routes via layer 5. (b) *Folded feedback* carries attentional signal from higher cortex into layer 4 of V1, via the modulatory 6? 4 path. Corticocotrical feedback axons tend preferentially to originate in layer 6 of the higher area and to terminate in the lower cortex's layer 1 (Salin and Bullier, 1995, p. 110), where they can excite the apical dendrites of layer 5 pyramidal cells whose axons send collaterals into layer 6. Several other routes through which feedback can pass into V1 layer exist. Having arrived in layer 6, the feedback is then 'folded' back up into the feedforward stream by passing through the 6? 4 on-center off-surround path (Bullier *et al*., 1996). [Reproduced with permission from Grossberg and Raizada (2000)].

The feedback circuits that govern the grouping and attentional selection processes are predicted to play a key role in helping to stabilize both development and adult learning within multiple cortical areas, including cortical areas V1 and V2. During development, the selection circuit (which itself is developing) helps to prevent the wrong combinations of cells from being co-activated, and thus from being associated, or wired, together.

Balanced excitatory and inhibitory connections have other effects as well on cortical processing. Balanced connections help to explain the observed variability in the number and temporal distribution of spikes emitted by cortical neurons. Modeling studies have shown how balanced excitation and inhibition can produce the highly variable interspike intervals that are found in cortical data (Shadlen and Newsome, 1998; van Vreeswijk and Sompolinsky, 1998). The present model suggests that such variability may reflect mechanisms that are needed to ensure stable development and learning by cortical circuits. Given that "stability implies variability," the cortex is faced with the difficult problem that variable spikes are quite inefficient in driving responses from cortical neurons. On the other hand, when one analyses how these balanced excitatory and inhibitory connections actually work to generate perceptual groupings, it becomes clear that the grouping circuits automatically have the property of preferentially responding to synchronized inputs. Figure 1 illustrates why synchronously activated cells will have a difficult time generating a perceptual grouping, whereas synchronously activated cells

will not. According to Figure 1a, an asynchronous volley of horizontal signals from a single population of layer 2/3 cells will kill itself off due to balanced excitation and inhibition. According to Figure 1b, a synchronous volley from pairs of appropriately positioned cells will initiate grouping. Earlier studies have also shown how both perceptual grouping and attentional circuits can, in fact, actively resynchronize signals that have become partially desychronized (Grossberg and Somers, 1991; Grossberg and Grunewald, 1998). The model hereby discloses a previously unsuspected link between properties of stable development, adult learning, grouping, attention, and synchronous cortical processing.

The article will focus primarily on how the types of horizontal connections and interlaminar connections develop within cortical layers 2/3, 4, and 6 that are mentioned above, primarily in cortical area V1, but by extension to V2 and higher cortical regions. These interactions are often cited as the basis of "non-classical" receptive fields that are sensitive to the context in which individual features are found (von der Heydt, Peterhans, and Baumgartner, 1984; Peterhans and von der Heydt, 1989; Born and Tootell, 1991; Knierim and van Essen, 1992; Sillito *et al.*, 1995). In this first modeling study, it is assumed that receptive fields of individual simple and complex cells in layers 4 and 2/3, respectively. have already substantially developed. Neurophysiologists classify simple and complex cells using a number of cell response properties. One of the most basic properties is that simple cells tend to respond to an oriented set of image features (oriented edge, shading, or texture gradient) with a prescribed contrast polarity (either dark-to-light or light-to-dark, but not both). Simple cells are the first stage of processing for bottom-up inputs to the cortex from the Lateral Geniculate Nucleus, or LGN; see Figures 3a and 4a. Complex cells tend to respond to an oriented set of image features that may have either contrast polarity (either dark-to-light or light-to-dark). In the classical Hubel and Wiesel (1977) model of cortical organization, these properties were assumed to arise due to converging inputs from two or more simple cells that respond to either contrast polarity onto a shared population of complex cells. This processing step occurs between layer 4 and 2/3 in the model (Figures 3a and 4a). The Hubel and Wiesel picture cannot, however, be fully supported because there are known feedback interactions that link cells in layers 2/3, 4, and 6 together, as illustrated in Figure 3a. These feedback signals cause the involved cells to share some of their response properties.

Two other models will be briefly reviewed in order to clarify how the simple and complex cell receptive fields themselves develop. Taken together, these three modeling studies provide a foundation for ongoing modeling work that is attempting to show how all the cortical layers develop; e.g., Seitz and Grossberg (2001). The second model considers the basic question of how cortical area V1 manages to pack in all the simple cells that respond to different eyes or different orientations at different positions on the retina. Cells in cortical area V1 are arranged into columns that run vertically through the cortical layers. Local circuits link together cells within a column in the different cortical layers, as illustrated in Figures 3a and 4b. Cells in each column have similar orientational tuning and sensitivity to eye of origin, or ocular dominance. The cortex packs these columns together in an efficient way by using a two-dimensional *map* of orientation and ocular dominance (Hubel and Wiesel, 1962, 1963, 1968). This organization is called a map because cell tuning to orientation and ocular dominance varies in a systematic way as the cortex is traversed in a horizontal direction. Such maps exhibit properties that are called singularities, fractures, and linear zones (Blasdel, 1992a, 1992b; Obeymeyer and Blasdel, 1993). The model shows how these features of cortical maps develop; see Figure 14 below.

Because of the critical importance of simple cells and of cortical maps in understanding cortical function, a number of models have studied how simple cells develop their orientationally tuned receptive fields within maps of orientation and ocular dominance (e.g., von der Malsburg, 1973; Grossberg, 1976a; Willshaw and von der Malsburg, 1976; Swindale, 1980, 1982, 1992; Linsker, 1986a, 1986b; Rojer and Schwartz, 1989, 1990; Durbin and Mitchison, 1990; Obermayer *et al.*, 1990, 1992; Miller, 1992, 1994; Grossberg and Olson, 1994; Sirosh and Miikkulainen, 1994; Olson and Grossberg, 1998). The model described herein will show, in addition, how nearby pairs of simple cells develop that are sensitive to the same orientation but opposite contrast polarities (Liu *et al.*, 1992). Such a model is called a Triple-O model because it shows how Orientation, Ocular Dominance, and Opposite Contrast Polarities all develop together (Olson and Grossberg, 1998). Earlier models were either Single-O or Double-O models, and many did not represent the dynamics of the cells whose connections were undergoing development. The Triple-O model clarifies how nearby simple cells that are sensitive to opposite contrast polarities could, in principle, cooperate to activate a shared complex cell.

The third model suggests how nearby pairs of simple cells that are sensitive to opposite contrast polarities actually develop connections to shared complex cells (Grunewald and Grossberg, 1998). In addition to being tuned to position, size, orientation, and pooled contrast polarities, the complex cells in the model, and *in vivo*, are also tuned to binocular disparity, which is a well-known cue to object depth (Julesz, 1971). These complex cell properties help to explain how depth-sensitive perceptual groupings can form over objects that are seen in front of textured backgrounds, and also how figure-ground properties emerge; see Grossberg (1994) for further discussion of how this happens. A key question for present purposes concerns how oppositely polarized simple cells, whose activations are *anti-correlated* in time (if a contrast at a given position is dark-to-light, it cannot also be light-to-dark, and conversely), can nonetheless develop connections to a shared complex cell, and thereby become *correlated*.

Several mechanisms are proposed that work together to achieve this end. One mechanism causes *antagonist rebounds* to occur between simple cells that are sensitive to opposite contrast polarities but the same positions and orientations. For example, when a simple cell that has been on for awhile in response to a dark-to-light contrast shuts off, an opponent simple cell, that is sensitive to a light-to-dark contrast, briefly turns on. Such rebounds are proposed to be due to the chemical transmitters that carry signals between model cells. Certain of these transmitters are proposed to habituate, or inactivate, when they are released by signals in their pathways, or axons. The same habituative transmitters also play a role in the models of simple cell maps develop. It is also suggested how learned feedback from cortical area V1 to the LGN may carry out a matching process that helps to stabilize the development of disparity tuning in cortical complex cells and, by extension, the cortical map itself; see Figure 4d. This V1-to-LGN feedback is homologous to the attentional feedback that is proposed to occur from cortical area V2 to V1 (Figure 3b), and by extension other cortical areas as well. These various interactions clarify how complex cells can binocularly match left and right eye image features with the same contrast polarity, yet can also pool signals with opposite contrast polarities, consistent with psychophysical and neurobiological data about adult 3-D vision; see Grossberg and McLoughlin (1998) and McLoughlin and Grossberg (1997) for an explanation of such data using these mechanisms. With this extended Introduction in hand, the relevant modeling concepts will now be described in greater detail.

Linking Cortical Development to Adult Perception

Perceptual grouping is the process whereby the brain organizes image contrasts into emergent boundary structures that segregate objects and their backgrounds in response to texture, shading, and depth cues in scenes and images (Julesz, 1971; Ramachandran and Nelson, 1976; Beck, Prazdny, and Rosenfeld, 1983; Polat and Sagi, 1994). Perceptual grouping is a basic step in solving the "binding problem", whereby spatially distributed features are bound into representations of objects and events in the world. Illusory contours are a particularly vivid form of perceptual grouping, since they illustrate how perceptual groupings can be completed over image locations that contain no contrastive scenic elements. Illusory contours are thus a popular and useful probe of how perceptual grouping occurs. Illusory contours are not just perceptual curiosities, however, since they also help to complete boundary representations over the retinal blind spot and veins, over missing pixels in textured images, and the like.

The first model to be reviewed (Grossberg and Williamson, 2001) suggests that many aspects of cortical design have evolved to carry out perceptual grouping. In particular, the model proposes how the laminar circuits of visual cortex enable it to develop connections capable of actively selecting and completing the perceptual grouping which best represents a visual scene, and suppressing the weaker groupings which represent the scene less well. The winning grouping has the property of *coherence* in the sense that its constituent features are actively bound together, indeed even synchronized, by feedback interactions. The winning grouping that is chosen in this way can also represent *analog* properties of the world, such as the relative contrasts and spatial positions of objects in the scene. I have called this combination of properties *analog coherence*. Analog coherence is not an easy combination of properties to achieve computationally in a robust way, since an active selection process that leads to coherent binding can all too easily sharpen feature values so much that their analog properties are lost. The LAMINART model shows how the laminar circuits of neocortex can robustly achieve analog coherence, and thereby solve the binding problem.

Model such as LAMINART which link brain to behavior need to show how interactions among many model cells give rise to emergent properties that match behavioral data. Several types of emergent properties are simulated by the model. As noted above, the model assumes that the classical receptive fields of simple and complex cells have already developed. This hypothesis is consistent with data showing that the oriented pattern of LGN-to-V1 connections develops prior to eye opening and structured visual input (e.g., Chapman *et al.*, 1991; Stryker and Harris, 1986; Antonini and Stryker, 1993a; Chapman and Stryker, 1993). The model focuses upon how the longer-range non-classical connections between cortical columns develop both prior to eye opening and after structured visual inputs occur. It proposes rules whereby such cortical development is controlled. Several such rules work together to control stable growth of model connections by ensuring that balanced excitatory and inhibitory connections develop. The emergent properties of this developmental process are the adult anatomical and neurophysiological circuits into which the model develops. After model development stabilizes, visual inputs activate cells within the developed anatomy, thereby leading to a second type of emergent properties, namely, the cell activity patterns that match data about adult visual perception. These two types of emergent properties show how a single model can explain data about cortical development, anatomy, neurophysiology, and perception.

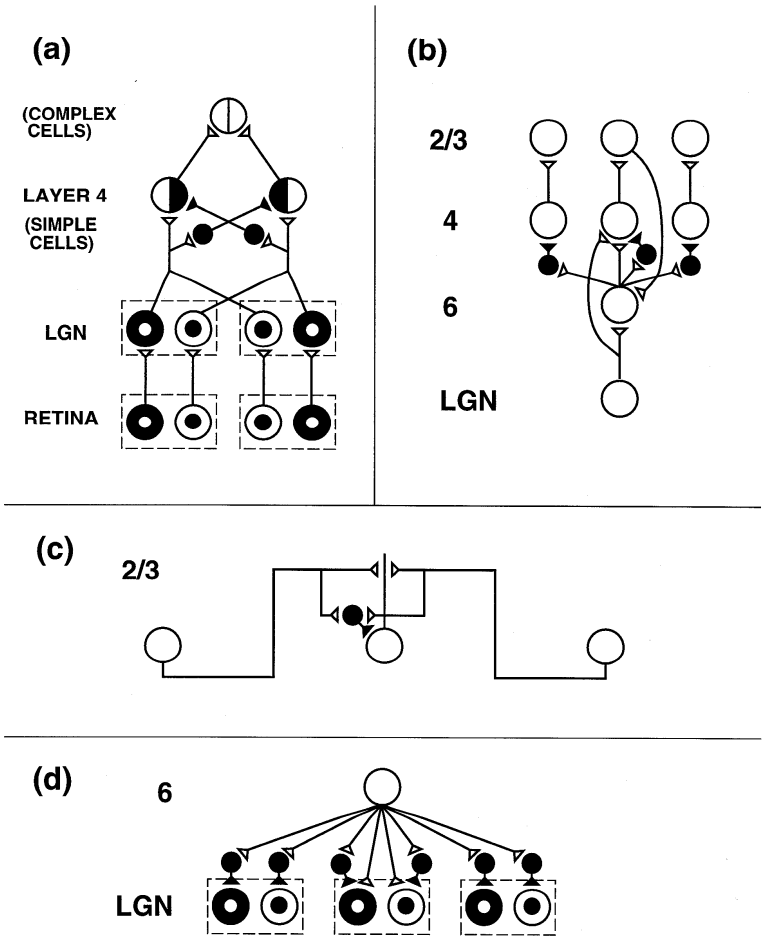

Figure 4. The adult network of retinal, V1, and lateral geniculate nucleus (LGN) neurons to which the developmental model converges: **(a)** Feedforward circuit from retina to LGN to cortical layer 4. **Retina:** Retinal ON cells have on-center off-surround organization (white disk surrounded by black annulus). Retinal OFF cells have an off-center on-surround organization (black disk surrounded by white annulus). **LGN:** The LGN ON and OFF cells receive feedforward ON and OFF cell inputs from the retina. **Layer 4:** LGN ON and OFF cell excitatory inputs to layer 4 establish oriented simple cell receptive fields. Like-oriented layer 4 simple cells with opposite contrast polarities compete before generating half-wave rectified outputs. Pooled simple cell outputs enable complex cells to respond to both polarities. They hereby full-wave rectify the image. See text for details. **(b)** Cortical feedback loop between layers 4, 2/3, and 6: LGN activates layer 6 as well as layer 4. Layer 6 cells excite layer 4 cells with a narrow on-center and inhibit them using layer 4 inhibitory interneurons that span a broader off-surround. Layer 4 cells excite layer 2/3 cells, which send excitatory feedback signals back to layer 6 cells via layer 5 (not shown). Layer 2/3 can hereby activate the feedforward layer 6-to-4 on-center off-surround network. (c) The horizontal interactions in layer 2/3 that initiate perceptual grouping: Layer 2/3 complex pyramidal cells monosynaptically excite one another via horizontal connections, primarily on their apical dendrites. They also inhibit one another via disynaptic inhibition that is mediated by model smooth stellate cells. Together these interactions can realize the "bipole property" which enables groupings to form inwardly across the space between two or more inducers, but not awardly from a single inducer. (d) Top-down corticogeniculate feedback from layer 6: LGN ON and OFF cells receive topographic excitatory feedback from layer 6,and more broadly distributed inhibitory feedback via LGN inhibitory interneurons that are excited by layer 6 signals. The feedback signals pool outputs over all cortical orientations and are delivered equally to ON and OFF cells. See the text for further details. [Reproduced with permission from Grossberg and Williamson (2001)].

Classical Receptive Fields: The model assumes that three types of circuits with (primarily) classical receptive field properties develop, at least in part, before the circuits that subserve non-classical receptive fields. We call the circuits that have already developed "pre-

developed" circuits. The circuits that develop through model dynamics are called "self-organized" circuits. The model analyzes one important combination of intracortical and intercortical pathways. It does not attempt to model all cortical connections, or the variations that exist across species. It also models the pre-developed circuits in the simplest possible way, since they are not the focus of this study (but see below for models of how the pre-developed circuits themselves develop), and the computational demands of the simulations are great even with these simplifications. Preliminary studies indicate, however, that the computational principles modeled herein can be elaborated and adapted to handle these variations.

Model analyses will be restricted to cortical area V1, and more particularly to the interblobs within V1 within which are suggested to carry out early stages of perceptual grouping. Converging evidence suggests that area V2 replicates the structure of area V1, but at a larger spatial scale, notably with longer horizontal connections to carry out grouping (van Essen and Maunsell, 1983; von der Heydt, Peterhans, and Baumgartner, 1984; Felleman and van Essen, 1991; Grosof, Shapley, and Hawken, 1993; Kisvarday *et al.*, 1995). These anatomical similarities make it plausible to assume that similar developmental processes may be operative in both V1 and V2. Due to the larger extent of horizontal connections in area V2, V2, rather than V1, may be carrying out the longer-range perceptual groupings that cross the blind spot, make illusory contours, and group discrete texture elements. In addition, V2 seems to be specialized for some of the processes that initiate depthful separation of figures from their backgrounds; see Grossberg (1994, 1997) for further analyses of figure-ground separation. The model's pre-developed and self-organized properties are intuitively described below. For a mathematical description, see Grossberg and Williamson (2001). Figure 4 schematize the model's connections.

Direct LGN Inputs to Layer 4: In both the brain and the model, the retina activates the Lateral Geniculate Nucleus (LGN) which, in turn, inputs to cortical area V1. LGN inputs directly excite layer 4 (Hubel and Wiesel, 1962; Chapman *et al.*, 1991; Reid and Alonso, 1995), as in Figures 4a and 4b. In modeling these data, a single, generic, layer 4 is used for simplicity; see the pathways with open triangles in Figure 4a. These inputs play a key role in establishing the orientational tuning of V1 simple cells.

Model simple cells have pre-developed connections that respond to a given orientation and contrast polarity; that is, they respond best to visual inputs that have a prescribed orientation and whose luminance preference, across this oriented axis, goes either from dark-to-light, or from light-to-dark, but not both. Simple cells are represented by circular symbols with half-white and half-black hemidisks in Figure 4a. These properties arise as follows from model LGN inputs and intracortical interactions: LGN ON cells (cells that are turned on by input onset; see symbols with white disks and black annuli in Figure 4a) and LGN OFF cells (cells that are turned off by input onset; see symbols with black disks and white annuli in Figure 4a) both input to layer 4. They are organized into spatially offset arrays, with the ON cell inputs spatially displaced with respect to the OFF cell inputs, as in Figure 4a. Due to this input array, layer 4 simple cells can respond to an oriented input whose luminant area excites the ON cells, and whose dark area excites the OFF cells.

Selectivity of simple cell responses to oriented contrasts is improved by mutually inhibitory interactions between cells that are sensitive to the same orientation but opposite contrast polarities (Palmer and Davis, 1981; Pollen and Ronner, 1981; Ferster, 1988; Liu *et al.*, 1992; Gove, Grossberg, and Mingolla, 1995); see the pathways with black triangles in Figure 4a. Then when cells that code opposite contrast polarities are equally activated by a uniform input, they shut each other off by mutual inhibition. On the other hand, when an oriented input is

presented, the simple cells that best match its position, orientation, and contrast polarity will be most activated. I review below how mutually inhibitory simple cells may develop which are sensitive to the same orientation and opposite contrast polarities, at the same time that a cortical map develops whose orientation and ocular dominance columns exhibit the fractures, singularities, and linear zones reported by Blasdel (1992a, 1992b) and Obermayer and Blasdel (1993).

Balanced LGN Inputs to Layer 4 via Layer 6: In both brain and model, LGN inputs also directly excite layer 6 (Ferster and Lindström, 1985), which then indirectly influences layer 4 via an on-center off-surround network of cells (Grieve and Sillito, 1991a, 1991b, 1995; Ahmed *et al.*, 1994, 1997). Cells in the on-center receive excitatory inputs from layer 6, whereas those in the spatially broader off-surround (that includes on-center cells) receive inhibitory inputs from layer 6 via inhibitory interneurons in layer 4. In Figure 4b, open triangles designate excitatory connections and black triangles designate inhibitory connections.

The model explains why layer 4 receives both direct LGN inputs and indirect inputs via layer 6. The indirect inputs to layer 4 from layer 6 cannot activate layer 4 cells. Because of the (approximate) balance between excitation and inhibition in the on-center, layer 6 can at best weakly activate, or modulate, cells in layer 4. This modulatory property helps to ensure stable cortical development and adult learning within the model. Such a dual input to layer 4 is found in many neocortical areas (van Essen and Maunsell, 1983; Felleman and van Essen, 1991). The model suggests that the combination of direct and indirect inputs to layer 4 from LGN helps to preserve stable development and learning in all these areas, while also allowing them to be activated by bottom-up inputs. In particular, the model predicts that the balance between the on-center and the off-surround inputs from layer 6 to 4 has the consequence that direct activation of layer 6 can modulate, prime, or subliminally activate, cells in layer 4, but cannot fire them vigorously. Although this prediction has not been directly tested, compatible data have been reported by Callaway (1998b), Hupé *et al.* (1997), and Wittmer, Dalva, and Katz (1997). The need to maintain this balance also predicts why direct inputs to layer 4 are needed, in addition to the indirect on-center inputs via layer 6, in many cortical areas. Since the indirect LGN-to-6-to-4 inputs cannot activate layer 4 cells without destabilizing cortical development and learning, the direct LGN-to-4 inputs are needed to initiate cortical firing. Model simulations support the prediction that, if the excitatory on-center inputs from layer 6 get too strong relative to the off-surround inputs from layer 6 to 4, then development does not self-stabilize. Instead, the non-classical receptive fields of the model proliferate uncontrollably. On the other hand, if the inhibition gets too strong, then it can inhibit the inputs arriving at layer 4 too much, thereby preventing the model cortex from becoming activated at all. Of course, one might argue that the direct LGN to layer 4 connections develop earlier in any case, but this explanation does not explain why this connection did not also develop the on-center off-surround network as a single input pathway.

Given that strong direct inputs from LGN to layer 4 do exist, the combined effect of both the direct and indirect pathways from LGN to layer 4 is to form an on-center off-surround network. When cells in such a network obey the membrane equations of neurophysiology, then they can maintain their sensitivity to input intensities that may vary over a large dynamic range (Grossberg, 1973, 1980b; Heeger, 1993; Douglas *et al.*,1995). This means that the *relative* input sizes can be detected by the target cells in layer 4, without saturation, over a wide dynamical range. This is because the membrane equations of neurophysiology which govern cell activation contain "shunting", or automatic gain control terms, that respond to properly balanced on-center

and off-surround inputs by normalizing the activities of target cells without destroying their sensitivity to the relative sizes of the inputs; see Grossberg (1988) for a review of this property. In the present instance, such a network maintains the sensitivity of cells in layer 4 to inputs from the prior processing level, whether it be cells in V1 responding to LGN inputs, cells in V2 responding to inputs from V1, or any other combination of inputs.

In summary, the LAMINART model predicts that the mechanism whereby the balance between excitation and inhibition is maintained in the layer 6-to-4 circuit is of great importance for achieving stable cortical development and later visual perception. This issue has been hardly explored experimentally. This prediction implies that a key cortical design problem is the following: As more and more cells in the off-surround become activated by increasingly dense patterns of inputs, what prevents the total inhibition that is converging on a layer 4 cell from growing linearly? If there was just enough inhibition to balance the excitation when just a few inputs were active, then why would not the inhibition become much too strong when many inputs were active, thereby shutting down the network? On the other hand, if the inhibition is well balanced when many inputs are active, then why does not runaway excitation occur when just a few inputs are active?

Development of Self-Normalizing Inhibitory Interneurons in Layer 4: The model solves these problems by assuming that the inhibitory interneurons in layer 4 inhibit one another, as well as target cells in layer 4; see Ahmed *et al.* (1994, 1997) for consistent data. In particular, the model suggests how layer 4 inhibitory interneurons learn connections to layer 4 spiny stellate excitatory cells as well as to other nearby layer 4 inhibitory interneurons. These connections start out with synaptic weights of zero magnitude, which are updated to learn the activity patterns of their target cells. The recurrent inhibition that develops between the inhibitory interneurons converts the network of inhibitory interneurons into a recurrent feedback network. Because the cells of this network obey membrane equations, this network of recurrent inhibitory interneurons tends to normalize the total activity across the interneuron population (Grossberg, 1973, 1980b). The total inhibition that converges on a layer 4 simple cell thus tends to be conserved as the total number of inputs varies, thereby maintaining the balance between excitation and inhibition, and avoiding the problems stated above.

If this self-normalization property within the inhibitory interneuronal population is experimentally confirmed, then it will be an interesting example of how less order on one level of biological organization generates more order on a higher level. In particular, the crucial self-normalization property can be achieved simply by allowing the inhibitory interneurons to randomly develop to inhibit all cells within their range, rather than restricting the developing inhibitory pathways to reach only excitatory target cells. As a result of this less ordered growth of inhibitory connections, the stability of the total network is facilitated.

Maintaining the balance between excitation and inhibition within the layer 6-to-4 on center does not imply that inhibition is weak. In fact, layer 4 cells that receive only off-surround inputs can be strongly inhibited. The model suggests how the on-center off-surround network from layer 6-to-4 can act as a *selection network* which selectively amplifies the strongest perceptual groupings in layer 2/3 via a 2/3-to-6-to-4 feedback loop, while actively suppressing LGN inputs to layer 4 that correspond to weaker groupings via the 6-to-4 off-surround; see Figure 1a. The inputs that have been supporting the weaker groupings are hereby inhibited, and thus the groupings themselves. This is proposed to happen as follows.

Columnar Organization via Folded Feedback: Active model layer 4 cells are assumed to generate inputs to pyramidal cells in layer 2/3 via pre-developed pathways. These layer 2/3

cells initiate the formation of perceptual groupings via horizontal connections that self-organize during model development; see Figures 1a and 4. How these horizontal connections develop is described below. Before describing this, we first note what happens when layer 2/3 cells are activated. Throughout the developmental process, all cells that are activated in layer 2/3, whether by bottom-up or horizontal inputs, send excitatory feedback signals to layer 6 via layer 5 (Gilbert and Wiesel, 1979; Ferster and Lindström, 1985), as in Figure 1b. Layer 6, in turn, once again activates the on-center off-surround network from layer 6 to 4. This process is called *folded feedback* (Grossberg, 1999a), because feedback signals from layer 2/3 get transmitted in a feedforward fashion back to layer 4. The feedback is hereby "folded" back into the feedforward flow of bottom-up information within the laminar cortical circuits.

Folded feedback is predicted to be a mechanism that binds the cells throughout layers 2/3, 4, 5 and 6 into functional columns (Mountcastle, 1957; Hubel and Wiesel, 1962, 1977). The on-center off-surround network from layer 6 to 4 responds to its layer 2/3 inputs by helping to control which combinations of cells remain simultaneously active during development, and thus which cells will wire together, because "cells that fire together wire together".

In particular, early during the development of model horizontal connections in layer 2/3, these connections are relatively unselective for colinear position and orientation, as in the data of Galuske and Singer (1996) and Ruthazer and Stryker (1996). Without further selection among the possible connection patterns, cortical interactions could remain both spatially and orientationally dispersed. This is corrected in the model via the intracortical folded feedback loop. In particular, suppose that a combination of bottom-up inputs and horizontal connections activates one subset of layer 2/3 cells a little more than a nearby subset of cells. Then, other things being equal, the favored layer 2/3 cells more vigorously activate their layer 2/3-to-5-to-6 pathway, and then their on-center off-surround layer 6-to-4 circuit. As a result, the cells whose activities form the strongest layer 2/3 grouping will suppress the activities of other cells via the layer 6-to-4 off-surround. The winning cells then get connected together via development, leading to a progressive increase in the projection range and orientational selectivity of these cells; see the simulations below. Such an increase in the projection range and orientational selectivity may explain why neonates appear to perceive a partly occluded object as disjoint parts, but can process these fragments as coherent objects within the first two to four months of life (Kellman and Spelke, 1983; Johnson and Aslin, 1996; Johnson, 2001).

This refinement process exploits the fact that orientationally tuned simple cells can bias development to favor long-range horizontal connections that are colinear with the preferred orientations of spatially aligned simple cells (Fitzpatrick, 1996; Schmidt *et al.*, 1997a). It is shown below how such oriented and colinear horizontal connections develop from an initial state in which no horizontal connections exist at all. It is also shown that, after development self-stabilizes, the same properties play a key role in generating perceptual groupings which exhibit properties of adult neurophysiological and psychophysical data. More recent modeling work simulates how the subplate can help to set up consistent initial orientational biases across layers even before layer 4 gets connected to layer 2/3 (Seitz and Grossberg, 2001).

Horizontal Connections and Perceptual Grouping: How these developing horizontal connections are prevented from generating run-away excitation and uncontrollable growth is one of the key properties of the model. A clue may be derived from properties of adult horizontal connections. In areas V1 and V2 of the adult, layer 2/3 pyramidal cells excite each other using monosynaptic long-range horizontal connections. They also inhibit each other using short-range disynaptic inhibitory connections that are activated by the excitatory horizontal connections

(Hirsch and Gilbert, 1991; McGuire *et al.*, 1991); see Figures 1a and 4c. The excitatory connections are hereby balanced by inhibitory connections. We show below how both types of connections can develop to generate perceptual groupings "inwardly" between two or more image contrasts that are aligned colinearly across space (Grosof, Shapley, and Hawken, 1993; Peterhans and von der Heydt, 1989; Redies, Crook, and Creutzfeldt, 1986; von der Heydt, Peterhans, and Baumgartner, 1984), but not "outwardly" from a single image contrast (Cannon and Fullenkamp, 1993; Hirsch and Gilbert, 1991; Knierim and van Essen, 1992; Somers, Nelson, and Sur, 1995; Stemmler, Usher, and Niebur, 1995). This is called the *bipole property* (Grossberg and Mingolla, 1985a, 1985b), and is illustrated in Figure 1. Illusory contours provide an excellent example of the bipole property: If a single image contrast could generate outward groupings, then our percepts would become crowded with webs of illusory contours spreading out from every feature in a scene. On the other hand, percepts of illusory contours between two or more colinear inducers are commonplace, as in the famous Kanizsa square (Kanizsa, 1979, 1985).

The model proposes how a balance between layer 2/3 excitation and inhibition develops that helps to stabilize cortical development and leads to the bipole property in the adult. For definiteness, call layer 2/3 pyramidal cells that receive bottom-up input from layer 4 "supported" cells, and those that do not "unsupported" cells. In the model, if an unsupported cell receives a sufficient amount of horizontal excitation, then it will be driven above its firing threshold. The cell will then output horizontal excitation to itself as well as to other pyramidal cells. Turning off input support from layer 4 causes all layer 2/3 activities to decay to zero. Therefore, boundaries can group across a gap provided the gap is small enough and the grouping signals from the supported cells on each end of the gap are sufficiently strong to drive the unsupported cells that lie between them above threshold.

The horizontal excitation from a single supported cell cannot cause runaway excitation and outward grouping among unsupported cells because it activates balanced disynaptic inhibition from smooth stellate cells. In this situation, the disynaptic inhibition is proportional to the horizontal excitation because both pyramidal and smooth stellate cells receive the same horizontal input signal. The inhibition from smooth stellate cells to pyramidal cells can lag behind the direct excitation between pyramidal cells due to the time it takes the smooth stellate cells to integrate their inputs. Therefore, synchronized inputs to layer 2/3 facilitate grouping because they allow the horizontal signals to summate at the target pyramidal cells before inhibition from local smooth stellate cells takes effect. This property is consistent with the finding of Usher and Donnelly (1998) that visual groupings are facilitated when inducers are presented synchronously.

As in the case of the layer 4 off-surround, the model disynaptic inhibitory interneurons are predicted to inhibit each other as well as the pyramidal cells. This model hypothesis is consistent with anatomical data showing that inhibitory interneurons synapse on both pyramidal cells and other interneurons (McGuire *et al.*, 1991; Kisvarday *et al.*, 1993). Hence the total activation within such a population of inhibitory interneurons is predicted to be normalized. As a result, it grows less quickly than summating activation of the pyramidal cells. The model hereby predicts that recurrent inhibition may be used to control the excitatory-inhibitory balance in both layer 2/3 and layer 4. In summary, net activation of the target pyramidal cells is possible, and grouping can occur inwardly but not outwardly, thereby realizing the bipole property (Grossberg and Mingolla, 1985b), which has been used to explain and predict many perceptual grouping data (e.g., Born and Tootell, 1991; Shipley and Kellman, 1992; Watanabe and Cavanagh, 1992;

Field, Hayes, and Hess, 1993; Grossberg, 1994, 1997; Polat and Sagi, 1994; Gove, Grossberg, and Mingolla, 1995; Dresp and Grossberg, 1997; Grossberg and Pessoa, 1998).

There is more neurophysiological evidence for the bipole property in cortical area V2 (e.g., von der Heydt, Peterhans, and Baumgartner, 1984; von der Heydt and Peterhans, 1989) than in V1. In V1, just a few unsupported cells have, to the present, been found that show full activation of unsupported cells by pairs of supporting cells. More V1 cells show a modulatory influence from neighboring pyramidal cells (e.g., Redies, Crook, and Creutzfeldt, 1986; von der Heydt and Peterhans, 1989; Grosof, Shapley, and Hawken, 1993; Kapadia, Ito, Gilbert, and Westheimer, 1995). These are challenging experiments to do in V1 because of the shorter horizontal connections there, and the existence of feedback from V2, which has longer horizontal connections. Unsupported V2 cells could be fully activated by stimuli that fall outside the V1 receptive fields, and could modulate V1 cells by top-down feedback. For simplicity, the present model assumes that the bipole property holds in both V1 and V2.

Developmental Rules: These properties of adult grouping arise in the model by specializing two well-known developmental rules. The first rule is that axons are attracted to cell targets when the source and target cells are both active (Gundersen and Barrett, 1979, 1980; Letourneau, 1978; Purves and Lichtman, 1980; Lichtman and Purves, 1981). The second rule is that axons compete intracellularly for growth resources (Purves and Lichtman, 1980; Lichtman and Purves, 1981). In the present instance, the first rule enables horizontal connections to form if activations in a source pyramidal cell and a target pyramidal cell are sufficiently correlated — in particular, if the target cell satisfies the bipole property — and removed if they are not (Callaway and Katz, 1990, 1991; Löwel and Singer, 1992; Dalva and Katz, 1994). This rule is realized by an activity-dependent morphogenetic gradient whose strength decreases with distance from the target cell that emits it. The gradient influences horizontal growth only in active source cells. The developing cells sense the correlation between the chemicals that define the morphogenetic gradient and those that are activity-dependent in the target cells. As contact between two cells is achieved, a synaptic learning law strengthens the synaptic contact by continuing to sense the correlation between presynaptic and postsynaptic activity.

The second rule prevents uncontrolled proliferation of horizontal connections by withdrawing connections from target cells that are receiving more poorly correlated signals than other target cells. The two rules work together to withdraw connections from cells that may be activated by weakly correlated image features or statistically insignificant noise.

Taken together, these model mechanisms for axonal growth and synaptic tuning dynamically stabilize cortical development as the developing cortical structure matches the statistics of its environmental inputs. If this match is disrupted later in life, then a new bout of development and/or learning can be triggered by the same mechanisms. Because of this property, the model can be used to clarify data about shared molecular substrates of neonatal development and adult learning (Bailey *et al.*, 1992; Kandel and O'Dell, 1992; Mayford *et al.*, 1992), plasticity of adult cortical representations after lesions (Merzenich *et al.*, 1988; Chino *et al.*, 1992; Gilbert and Wiesel, 1992; Darian-Smith and Gilbert, 1994; Kapadia *et al.*, 1994; Das and Gilbert, 1995; Schmidt *et al.*, 1996), dynamical reorganization of long-range connections in the visual cortex (Gilbert and Wiesel, 1992; Zohary *et al.*, 1994), and perceptual learning in the adult (Karni and Sagi, 1991; Poggio, Fahle, and Edelman, 1992). In fact, the model equations for activity-dependent controls of synaptic strength have already been used to explain properties of adult learning (e.g., Grossberg, 1980a; Carpenter and Grossberg, 1991).

Top-Down Feedback from V1 to LGN: Layer 6 of model area V1 sends top-down feedback to the LGN via an on-center off-surround network, as also occurs in vivo (Murphy and Sillito, 1987; Weber, Kalil, and Behan, 1989; Murphy and Sillito, 1996); see Figure 4d. The feedback on-center reinforces the activities of those LGN cells which have succeeded in activating V1 cells, notably V1 cells whose activations represent the strongest perceptual groupings. The feedback off-surround suppresses the activities of other LGN cells. As in the brain, this model feedback circuit increases the useful visual information that is transmitted from LGN to cortex by enhancing contextually significant differences between LGN responses (McClurkin *et al.*, 1994), and also influences the length tuning of LGN cells (Murphy and Sillito, 1987). The LGN to V1 circuit is also known to be modulatory (Sillito *et al.*, 1994).

Earlier modeling work predicted that this feedback pathway plays a role in stabilizing the development of bottom-up connections from LGN to V1, as well as the reciprocal top-down connections from V1 to LGN (Grossberg, 1976b, 1980a). Grunewald and Grossberg (1998) have modeled how the normal development of bottom-up disparity tuning can occur at V1 complex cells when such top-down feedback is operative, and have shown how this development may break down when it is not. Further experimental study of this question is needed. For simplicity, in the model of how horizontal and interlaminar cortical circuits develop, it was assumed that these top-down V1-to-LGN connections are pre-developed and are available to facilitate activation of the correct combinations of simple and complex cells.

Developmental Data and Simulations

This section illustrates how the model of Grossberg and Williamson (2001) simulates data about the development of long-range horizontal connections in area V1. After development self-stabilizes, the resultant network can, without further change, simulate adult neurophysiological and psychophysical data about perceptual grouping. One such simulation will be shown below after developmental data are simulated.

As in the brain, the model undergoes two stages of development (Figure 5). One occurs prior to eye opening, when endogenous random geniculate and cortical activity determine the initial specificity of horizontal connections (Ruthazer and Stryker, 1996). The other occurs after eye opening, when patterned visual inputs can strengthen and refine these connections (Galuske and Singer, 1996).

Several anatomical studies have investigated how horizontal projections develop in the superficial layers of visual cortex into adult connections that connect columns of similar orientation preference (Callaway and Katz, 1990; Durack and Katz, 1996; Galuske and Singer, 1996). Callaway and Katz (1990) used neuronal tracing and intracellular staining to investigate the development of clustered horizontal connections in cat striate cortex, or area 17 (the analog of monkey area V1). They found an even, unclustered distribution up to 2 mm from the injection site during the first postnatal week, followed by an increase in the range and clustering of the projections in the second postnatal week, when the eyes are opened, and finally a long, slow refinement of projections due to the elimination of some connections until an adult level of clustering was reached in the sixth postnatal week.

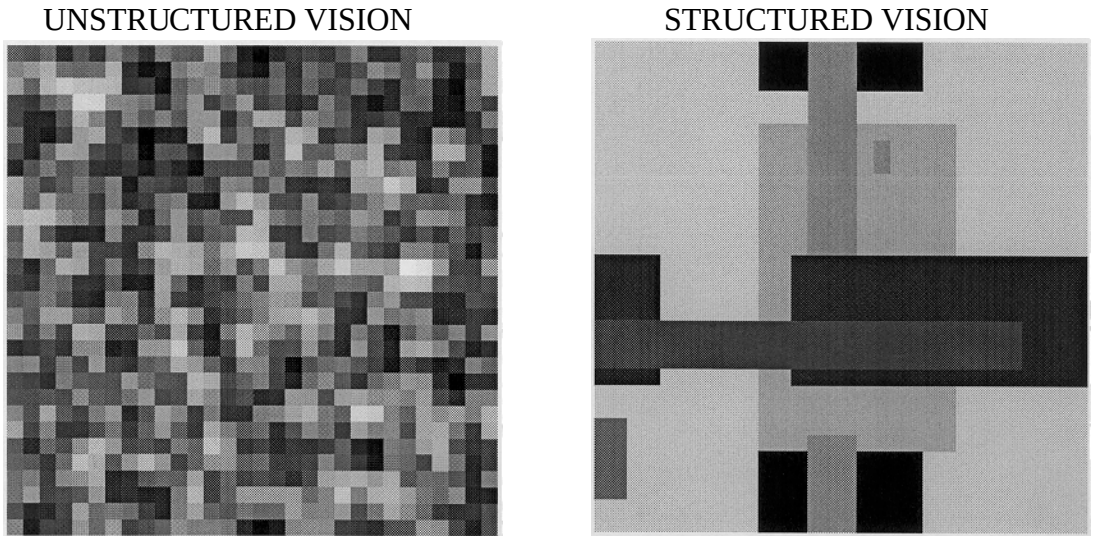

Figure 5. LEFT: Example training image, consisting of Gaussian filtered random noise, used to model unstructured vision prior to eye opening. **RIGHT**: Example training image, consisting of 7 randomly configured rectangles, with input values randomly distributed between 0 and 2, used to model structured vision after eye opening. [Reproduced with permission from Grossberg and Williamson (2001)].

Increase of Projection Range: The Galuske and Singer (1996) investigation of long-range projections in cat area 17 at different stages of postnatal development yielded a similar conclusion. Galuske and Singer (1996) also reported quantitative data about the projection range of pyramidal cells (Figure 6, top). Soon after eye opening, the projection range doubled over a period of twelve days (from P15- P26). Presumably, the increase in projection range is due to the greater correlations in activity over large spatial distances that occurs in natural, structured, images. Figure 6 (bottom) shows the simulated projection range in the model. Before eye opening, the short-range spatial correlations of the unstructured inputs are reflected in the relatively short-range extent of horizontal projections. Soon after eye opening, the long-range spatial correlations in the structured visual inputs cause the projection range to double, just as in the data of Galuske and Singer (1996). These results exploit the developmental rules described above by causing a larger projection range to grow when the statistics of visual imagery provided more long-range correlations. A similar effect in human infants, albeit delayed relative to the time scale of the cat, could explain how perceptual grouping takes hold between 2 and 4 months of age.

Increase of Orientational Selectivity: A similar pattern of exuberant growth followed by slow refinement of projections has also been found in the ferret. Because the ferret is born 3 weeks earlier in development than the cat, it has more stable orientation-selective cortical cell responses than the cat during the period in question (Durack and Katz, 1996; Ruthazer and Stryker, 1996). Ruthazer and Stryker (1996) reported quantitative data about the growing orientational selectivity of horizontal clustering over time, using a statistic called the Cluster Index (CI). The CI measures the log of the average nearest-neighbor distance between horizontal projections within a measurement window, divided by the average distance between a randomly selected point in the window and the nearest horizontal projection. Therefore, a uniform

distribution of horizontal projections would lead to a CI of log(1) = 0. As clustering becomes more refined, CI increases. Figure 7 (top) shows the CI obtained by Ruthazer and Stryker (1996) from 21 days postnatal up to adult age. Before eye opening, which is about 31 days postnatal, there is a positive CI, indicating a clustering bias, presumably favoring iso-orientation connections. After eye opening, the CI rapidly increases to reflect the strong, adult bias in favor of iso-orientation connections.

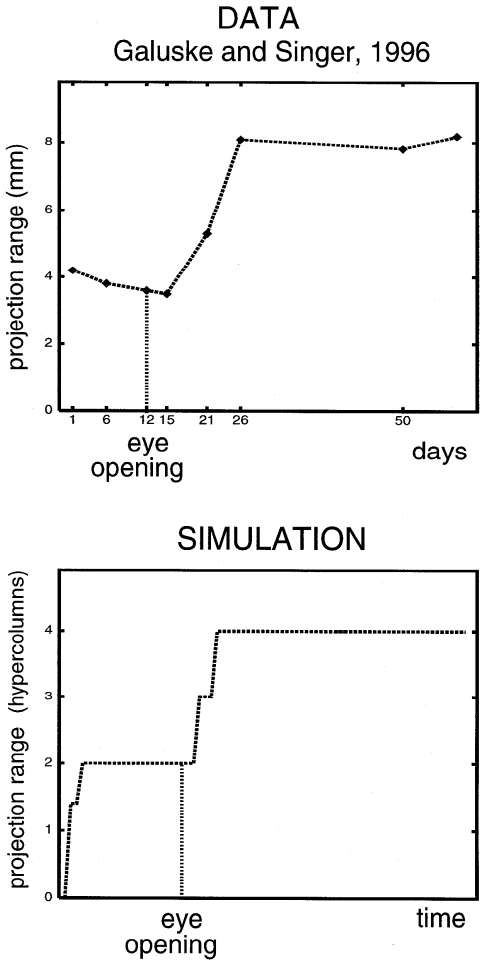

Figure 6. TOP: Projection range of pyramidal cells in cat visual cortex as a function of age. Projection range doubles after eye opening [Adapted from Galuske and Singer (1996)]. **BOTTOM**: Projection range of model pyramidal cells during development. Model projection range also doubles after ``eye opening''. [Reproduced with permission from Grossberg and Williamson (2001)].

The model does not represent individual horizontal projections, but rather the average strength of horizontal projections from an orientation column to other orientation columns. Therefore, the model's format is unsuitable for computing a CI index. An analogous measurement of orientation preference was computed by dividing the strength of a column's horizontal connections to nearby columns with the same orientation preference by the strength of all the column's horizontal connections. This statistic is shown in Figure 7 (bottom). Like the CI index, it shows an initial moderate bias in favor of iso-orientation connections that dramatically increases after eye opening. In order to make the computer simulations tractable, the model presently represents only two orientations (vertical and horizontal) so Figure 7 shows the bias in favor of one orientation over the perpendicular orientation. If the model represented intermediate orientations as well, then the relative iso-orientation bias would be smaller because the presence

of intermediate orientations would reduce the average orientation distance between iso- and non-iso-orientation columns.

After development, horizontal projections preferentially connect columns with similar orientation preferences that are aligned colinearly with their orientation preference (Fitzpatrick, 1996; Schmidt *et al.*, 1997a). Figure 8 (left) shows a polar plot from Fitzpatrick (1996) of the projection field from a site in layer 2/3 of tree shrew striate cortex. The distance of each point from the center of the projection field represents the number of labeled terminals at that angle (in 10° increments). The orientation of the projection field is aligned with the orientation preference of its source neuron. Figure 8 (right) shows the analogous projection field from a horizontally tuned column in layer 2/3 of the model after development has equilibrated. The size of each circle represents the strength of the connection to each iso-orientation column. The anisotropy of the model's projection field is qualitatively consistent with Fitzpatrick's data. These results derive from the fact that visual cues are, with high probability, locally-linear across space, so that the largest correlations would be generated by cells whose orientations match those of the input and are colinearly aligned across space. The developmental rules enable the network to sense these correlations and to selectively amplify the growth of those connections which best match them.

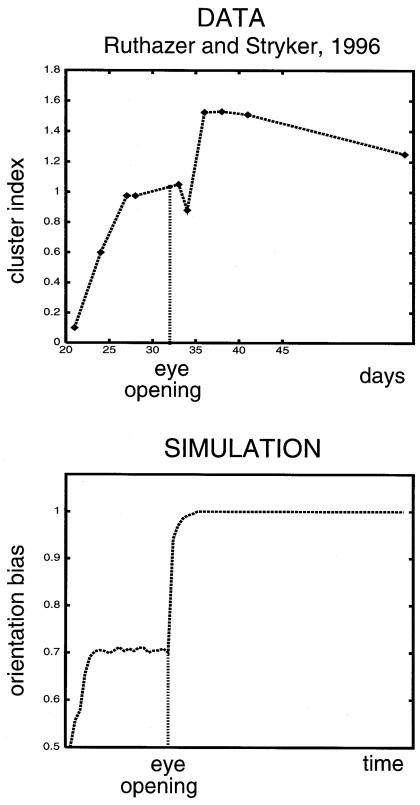

Figure 7. TOP: Mean Cluster Index (CI) in ferret area 17 as a function of age. From Ruthazer and Stryker (1996): "At P27 horizontal connections are significantly clustered, but single-unit recordings reveal poor orientation selectivity (25% of cells have orientation-selective responses), and optical imaging does not yet show an orientation map. Between P32 and P36, a secondary refinement of horizontal connections occurs along with the maturation of single-unit orientation selectivity and the emergence of the earliest optical orientation maps." Eye opening takes place at about P31. [Adapted from Ruthazer and Stryker (1996).] **BOTTOM**: Clustering bias in model during development. The strength of horizontal connections to iso-orientation columns divided by the net strength of horizontal connections is plotted as a function of age. Like the data of Ruthazer and Stryker, the clustering bias increases after eye opening. [Reproduced with permission from Grossberg and Williamson (2001)].

Projection Field vs. Receptive Field: Neurophysiological recordings confirm that the anatomy which develops in the model has the cellular properties similar to those that have been recorded from adult animals. A remarkable property of this kind provides additional support for the bipole property. In particular, the extent of a cell's total anatomical projection field is much greater than that of its neurophysiologically recorded receptive field (Fitzpatrick, 1996).

Fitzpatrick found that the projection fields in tree shrew extend for more than 2 mm. from the injection site, a distance that corresponds to 15 degrees eccentricity, whereas the dimensions of neurophysiologically characterized receptive fields at that eccentricity are less then 5 degrees. A smaller classical receptive field than projection field was also shown by Das and Gilbert (1995), who compared cortical point spread (PS) distributions, measured with optical recording, which reflect both spiking and subthreshold activity, with spiking distributions measured with extracellular electrodes. A small oriented visual stimulus produced a PS distribution 20 times larger than the spiking distribution. Moreover, the close match of the PS distribution with columns whose orientation preference agrees with the orientation of the visual stimulus suggests that the distribution arises from iso-oriented long-range horizontal projections.

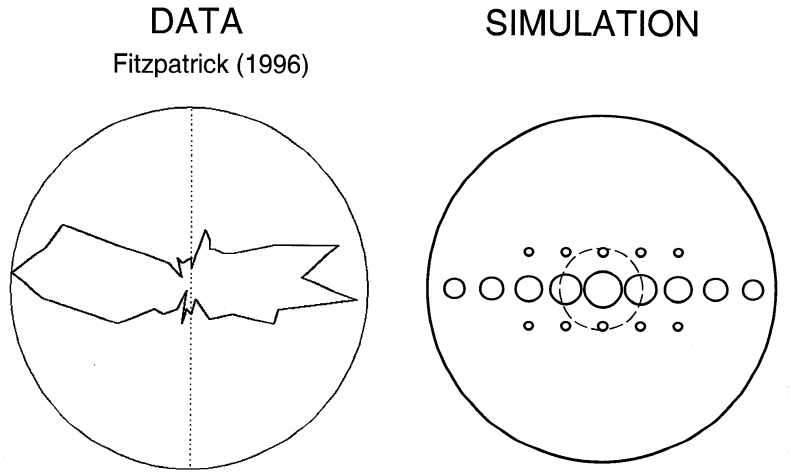

Figure 8: LEFT: Polar plot of the projection field from a site in layer 2/3 of tree shrew striate cortex. The orientation of the projection field is in agreement with the orientation preference of its source neuron. [Adapted from Figure 11 of Fitzpatrick, 1996.] **RIGHT**: The projection field from a horizontally tuned column in layer 2/3 of the model after learning has equilibrated. The size of each circle represents the strength of the connection to each iso-orientation column. The dashed circle in the middle shows a layer 2/3 cell's classical receptive field, which is the spatial extent within which a point input causes the cell to ``fire'' (i.e., go above its output threshold). [Reproduced with permission from Grossberg and Williamson (2001)].

A similar property holds in the model after development equilibrates: Figure 8 (right) shows the size of a layer 2/3 cell classical receptive field (dashed-line circle) with respect to its projection field in the model. This discrepancy between projection field and receptive field can be traced to the model's bipole property: The classical receptive field reflects mainly bottom-up properties of the cortical network in the model, whereas the subthreshold activations reflect the fact that the bipole requirement for firing the cells via long-range horizontal connections was not satisfied. The developed model also exhibits the type of cortical point spread functions that have been found through optically recorded signals are believed to arise from subthreshold dendritic activity in the superficial layers (Grinvald *et al.*, 1994). See Grossberg and Williamson (2001) for further details.

Psychophysical Data and Simulations

A crucial test of a model of visual cortical development concerns whether the developed model behaves perceptually like an adult cortex after development ends. This sort of test provides strong indirect evidence that the types of factors which influence how infants group image parts at different stages of development are actually being captured by the model. Without quantitative tests of such a linking hypothesis, it is difficult to feel any confidence in hypotheses about what brain mechanisms are responsible for observed changes in infant perception at different ages. In the present model, a key perceptual issue concerns whether the model can reproduce data about perceptual grouping. In particular, can the developed model generate illusory contours that are sensitive to changes in the strength and position of contour inducers in the same way that human observers are? This is a crucial test of perceptual grouping for at least two reasons: First, illusory contours require grouping to occur over positions that do not receive bottom-up inputs, so the model's *boundary completion* property is tested in this way. Second, analog changes in the emergent groupings as a function of input intensity and position — which I have called the property of *analog coherence* — is one of the key properties that laminar cortical circuitry has been predicted to generate. Two tests of these properties are summarized below.

Contour Sensitivity to Support Ratio: Figure 9 shows how the illusory contours formed by the model, either colinear to edges or perpendicular to line ends, vary in strength as the inducing features are parametrically varied. These simulations illustrate that the developed layer 2/3 connections do exhibit the property of analog coherence. Figure 9 (top) plots data of Shipley and Kellman (1992) which show the effect of increasing the length of the contour inducers while decreasing the gap between them, keeping the total length of inducers-plus-gap constant. Then, illusory contour clarity increases roughly linearly. In other words, "contour clarity increases with support ratio". Figure 9 (top) shows that the clarity of the model's illusory contours also increases linearly as the support ratio is increased. This result is due to the fact that, as the gap between two inducers is made smaller, the grouping signal becomes stronger, due to the monotonically increasing magnitude of the layer 2/3 grouping kernel towards its center (see Figure 8).

The model matches the psychophysical data well, with the caveat that the model cannot form illusory contours when the support ratio falls below 0.5. This is due to simplifications in the model that were made for computational tractability. These simplifications limit the extent of the groupings it can make. In particular, model parameters were chosen so that its developed horizontal projections extend only four hypercolumns away from the center. (A hypercolumn is a unit in the cortical map that contains the complete set of orientationally tuned and ocular dominance cells that represent a given position.) Another limitation is that the model only simulates grouping in V1 and does not take advantage of larger-scale processing in V2. Finally, the model does not include the retina-to-cortex cortical magnification factor (van Essen *et al.*, 1984), whereby scale expansion takes place as stimuli move into the periphery.

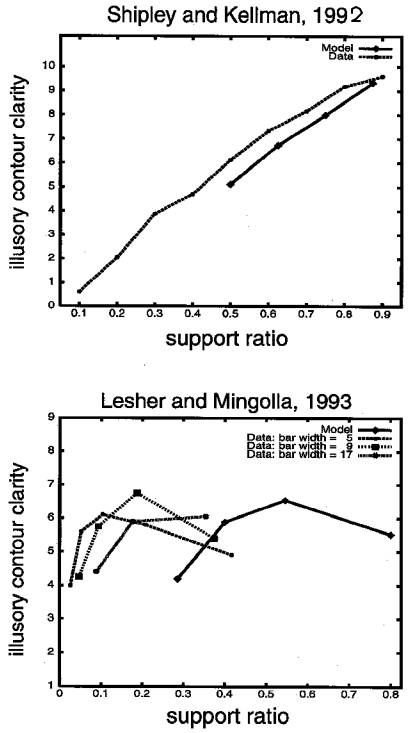

Figure 9. TOP: Shipley and Kellman (1992) obtained clarity ratings for illusory contours as a function of their support ratio. The stimulus was a 4 cm. illusory Kanizsa square, induced by four pacmen figures. As the support ratio increased (i.e., the size of the pacmen increases and the size of the gap decreases) the illusory contour clarity increased roughly linearly. [Adapted from Figure 5 of Shipley and Kellman (1992).] The model results were obtained by measuring the strength of vertical grouping between two aligned rectangles (3 pixels wide). The length of the rectangles plus gap was 8 pixels. As the size of the gap was decreased from 4 pixels to 1 pixel by increasing the length of the rectangles, the average grouping strength in the gap increased. See text for a description of how the grouping strength was mapped into a metric of perceived illusory contour clarity. **BOTTOM:** Lesher and Mingolla (1993) also obtained clarity ratings for illusory contours as a function of support ratio. However, they increased support ratio by increasing the number, and hence the density, of inducers in the following type of Kanizsa square display: The pacmen were built up from concentric rings of black contours whose number and density was increased across displays. As the number of inducing contours, and hence the support ratio, increases, the illusory contour clarity increases and then decreases. [Adapted from Figures 8a and 10c of Lesher and Mingolla (1993).] The model's illusory contour strength was measured along a 4-pixel gap. Inducers were 2-pixel-wide bars, with the spacing between bars varied to yield 1, 2, 3, and 4 bars on each side of the gap, with inter-bar spacing of 3, 2, 1, and 0 pixels, respectively. [Reproduced with permission from Grossberg and Williamson (2001)].

Figure 9 (bottom) summarizes psychophysical data obtained by Lesher and Mingolla (1993) showing that, if support ratio is increased in a different way, then an inverted-U in illusory contour clarity strength is obtained. In this study, parallel bars with aligned ends were

used to form four pacman figures with which to induce an illusory Kanizsa square percept. The square formed perpendicular to the bars through their aligned ends. Contour clarity of the illusory square was measured as the numbers of bars, and hence the support ratio, varied. The inducing pacmen had a circular radius of 128 pixels, and the gap between pacman pairs in which the Kanizsa square percept formed was 128 pixels. The support ratio was computed as the number of bar inducers (1, 2, 4, 8, 16 per pacman) times bar width, divided by the length of the side of the square (384 pixels). As the width of the bar inducers is increased, the number of possible inducers becomes limited, which is why there are only results for up to 16 inducers in the 9-pixel-width case, and up to 8 inducers in the 17-pixel-width case.

Figure 9 (bottom) shows that the model simulates the inverted-U in contour strength as a function of bar density. This inverted-U result is due to an interaction between the long-range excitatory horizontal connections in layer 2/3 and the medium-range inhibitory connections from layer 6-to-4. The Shipley and Kellman (1992) data, and our simulation thereof, show that decreasing the distance between inducers, up to a certain point, increases grouping strength as a result of layer 2/3 horizontal cooperation. As the inducers get even closer together, however, layer 6-to-4 inhibition increasingly inhibits the net excitation caused at layer 4 by each LGN input. Thus, although more inputs activate the cooperating layer 2/3 pyramidal cells, the net effect of each input on layer 2/3 gets smaller as the inducers get denser. This simulation shows that the self-organized connections preserve a good balance between layers 6, 4, and 2/3. As in the psychophysical data in Figure 9, the model's illusory contour strength is affected more strongly by variations in support ratio than in bar density.

Due to the implementational limitations of the model described above, the network simulated these data using bars that are relatively wide with respect to the length of the gap (2 pixel wide bars, 4 pixel long gap). Figure 9 (bottom) shows results obtained by the model with inter-bar gap size decreasing from 3 to 0, with the total length spanned by the inducers and gaps held roughly constant. The model's inverted-U curve is shifted to the right of the data curves, reflecting the fact that the model used inducers that were wider relative to the gap size. Note that, in the data as well, the curves shift to the right as the width of the inducers increases.

Interactions of Grouping, Attention, and Recognition

Up to this point, the article has summarized a LAMINART model of how horizontal and interlaminar cortical connections in cortical areas V1 (and by extension, area V2) develop in a stable fashion. Stable development is controlled by the growth of balanced excitatory and inhibitory connections within layer 2/3 and between layers 6 and 4. The model grows connections that simulate key properties of developmental anatomical data and adult neurophysiological data about this process, and the developed network quantitatively simulates key data about adult perceptual grouping, notably data that depend upon non-classical receptive field properties, thereby testing the linking hypothesis between brain mechanisms and perception. Many additional psychophysical data about both perceptual grouping and attentional modulation in the adult are simulated using the model in Grossberg and Raizada (2000), Grossberg and Williamson (2001), and Raizada and Grossberg (2000). With attentional connections also in place, the model may be summarized as in Figure 10.

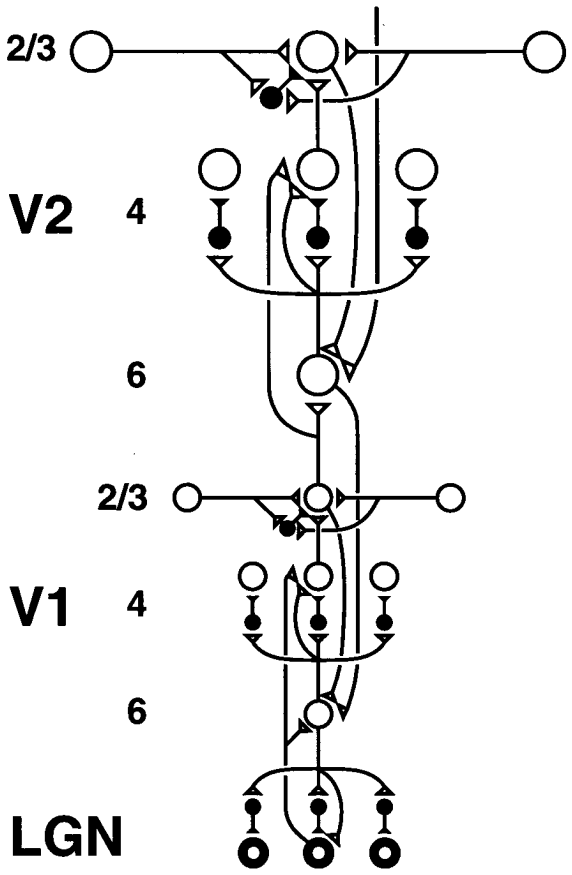

Figure 10. The LAMINART V1/V2 circuit: V2 repeats the laminar pattern of V1 circuitry, but at a larger spatial scale. In particular, the horizontal layer 2/3 connections have a longer range in V2, allowing above-threshold perceptual groupings between more widely spaced inducing stimuli to form (Amir *et al.*, 1993). V1 layer 2/3 projects up to V2 layers 6 and 4, just as LGN projects to layers 6 and 4 of V1. Higher cortical areas send feedback into V2 which ultimately reaches layer 6, just as V2 feedback acts on layer 6 of V1 (Sandell and Schiller, 1982). Feedback paths from higher cortical areas straight into V1 (not shown) can complement and enhance feedback from V2 into V1. [Reprinted with permission from Grossberg and Raizada (2000)].

As noted above, in both the brain and the model, layer 2/3 boundary signals feed back via connections to layer 6 via layer 5 (Gilbert and Wiesel, 1979; Ferster and Lindström 1985). Layer 6, in turn, activates the on-center off-surround network from layer 6-to-4 via folded feedback (Grossberg, 1999a). The feedback signals from layer 2/3 to layer 6 hereby gets transmitted in a feedforward fashion back to layer 4 and thereupon to layer 2/3. Folded feedback links cells in layers 2/3, 6, 5, and 4 into functional columns (Mountcastle, 1957; Hubel and Wiesel, 1962, 1977). In so doing, it enables the strongest grouping signals in layer 2/3 to use the on-center off-surround network from layer 6-to-4 to reinforce the strongest groupings and to inhibit weaker groupings, during both early development and adult grouping and learning. As noted above, this feedback circuit helps to stabilize model development by shutting off cells that should not become connected for purposes of grouping. The full LAMINART model also has the following remarkable properties which illustrate how mechanisms that ensure stable cortical development also lead to useful properties of adult grouping, attention, and recognition:

Fast Feedforward Grouping and Recognition: Although the competitive selection circuit from layer 6-to-4 is needed to choose correct groupings in response to complex scenes with many almost equally strong groupings, the system can automatically generate a fast grouping of an unambiguous scene using a one-pass *feedforward* wave of activation through layers 4-to-2/3 in one area, then from layer 2/3 to layer 4 in the next area, and so on; see Figure 10. Such fast feedforward recognition has been experimentally shown to be possible in humans

and monkeys (Thorpe *et al.*, 1996). If the scene is complex and ambiguous, however, then inhibitory interactions between the competing groupings in the various layers (e.g., from layer 6-to-4 and within layer 2/3) attenuate the cortical output from layer 2/3, and thereby prevent strong outputs from being generated to higher cortical areas until a clear choice can be made. As weaker groupings are suppressed, the strongest groupings win the competition and become more active. The winning groupings can then more quickly reach their output threshold. This self-regulating design enables fast feedforward processing when the data are unambiguous, and a functionally determined delay for selecting a correct grouping via feedback when they are not. Thus, *after* the selection circuit enables orientationally selective receptive fields and horizontal connections to develop, it still plays an important role whenever ambiguity exists in the visual scene, since it then helps to choose the strongest groupings while also suppressing spurious correlations in cell activation that could otherwise degrade previously learned connections.

Selective Object Attention: As noted above, one of the key selection circuits that helps to choose perceptual groupings is realized by an on-center off-surround network from layer 6-to-4. This selection circuit is activated by *intra*cortical feedback from the horizontal groupings that start to form in layer 2/3 (Figure 4b). The same selection circuit is activated by top-down attention via *inter*cortical feedback from layer 6 of higher cortical areas (Figure 10). Because of this property, development and learning between different cortical regions can also be stabilized, as predicted by Adaptive Resonance Theory (Grossberg, 1980b, 1999b). Simulations of the LAMINART model (Grossberg and Raizada, 2000) show how attention can selectively enhance an entire object, while suppressing nearby distractors. This remarkable property was demonstrated in neurophysiological recordings in the awake monkey by Roelfsema *et al.* (1998). Object-attention is achieved in the model through the sharing by both attention and grouping of the same selection circuit, whereby a correct grouping can be selected from among many possible groupings of a complex scene. Because attention and grouping both activate the same selection circuit, attention can propagate along an entire object's grouping after it reaches layer 2/3 via the 6-to-4-to-2/3 pathway.

Object Attention with Incomplete Boundaries: In humans, attention can selectively enhance an entire object when the object is defined by an image with lots of missing pixels, as can occur in high noise (Moore, Yantis, and Vaughan, 1998) or due to imperfections, such as a scotoma, in retinal processing. Model simulations in Raizada and Grossberg (2000) have shown how grouping can preattentively complete the object boundary over the missing pixels via illusory contours; then attention can selectively enhance the completed object grouping.

Attentional Selection of Important Data: Using the top-down layer 6-to-6 attentional route, attention can leap-frog between multiple cortical areas. In this way, figure-ground and cognitive constraints from higher cortical areas can help to select groupings and to thereby search for desired objects in a scene. Attention can also tune the bottom-up adaptive filters in layer 4 of each area to be particularly sensitive to important information by using a layer 6-to-6-to-4 folded feedback pathway (Figure 10).

Why does not the top-down 6-to-6 route turn on the entire cortex like a bottom-up input would? If this happened, it would blur the distinction between intention and reality. Hallucinations would be commonplace. This does not happen because, as predicted by ART, the on-center in layer 6-to-4 has *balanced* excitation and inhibition. It can modulate, sensitize, or prime layer 4 to respond more vigorously to desired inputs, but it cannot, by itself, turn layer 4 on. This hypothesis is consistent with neurophysiological data of Hupé *et al.* (1997) who have

shown that "feedback connections from area V2 modulate but do not create center-surround interactions in V1 neurons" and data from ferret visual cortex has shown that the layer 6-to-4 circuit is functionally weak (Wittmer, Dalva, and Katz, 1997). Such intercortical feedback connections from V2 to V1 can modulate the circuits of V1 with "higher-order" boundary completion and figure-ground perception properties of area V2 (Grossberg, 1994, 1997; Lamme, 1995; Zipser, Lamme, and Schiller, 1996), and/or other cortical areas (e.g., Hupé *et al.*, 1998; Watanabe *et al.*, 1998).

Why Two Bottom-Up Paths to Layer 4?: The predicted modulatory property of the layer 6-to-4 circuit helps to explain the otherwise mysterious existence of a direct input to layer 4 of V1 from the LGN (Figure 4b) and to layer 4 of V2 from layer 2/3 of V1 (Figure 10). Without the direct route to layer 4, cortex could never turn on at all, because the indirect 6-to-4 route, which can also be activated by bottom-up inputs, is merely modulatory. Why, in turn, is the indirect 6-to-4 route merely modulatory? Given that a similar arrangement seems to exist in *all* sensory and cognitive neocortex, why is this not a huge waste of wire? My proposed answer is: ART has proved mathematically that a modulatory feedback selection circuit is needed so that the cortex can stably develop its connections in the infant, and can stably learn in the adult (Carpenter and Grossberg, 1991; Grossberg, 1976b, 1980b, 1999a, 1999b). The rules of stable development are thus predicted to define what we *mean* by adult attention, as well as adult grouping and learning.

Development of Cortical Map

Triple-O Map Properties: Development of the primary visual cortex prior to visual experience produces orientationally tuned cortical neurons, classifiable according to the criteria of Hubel and Wiesel (1962) as either simple or complex. After several weeks of visual experience these cortical cells evolve adult responsivity (DeAngelis *et al.*, 1993; Ghose *et al.*, 1994; Hubel and Wiesel, 1974). The prenatal segregation of geniculocortical afferents into ocular dominance columns also occurs independently of visual experience (Horton and Hocking, 1996). Monocular, but not binocular, deprivation during the first few weeks of visual experience can lead to drastic changes in the arrangement of ocular dominance patches (Hubel *et al.*, 1977), but these changes may be blocked by the elimination of neural activity (Stryker and Harris, 1986), suggesting that an activity-dependent process is responsible for the development of ocular dominance.

Adult cortical cells are arranged into vertical columns with similar orientation tuning and ocular dominance, and these columns are arranged into smoothly changing two-dimensional maps of orientation and ocular dominance (Hubel and Wiesel, 1962, 1963, 1968). The properties of orientational tuning and of ocular dominance constitute two of the O's in the Triple-O map. The cortical map of orientation is arranged in swirling patterns around orientation centers in both cats (Bonhoeffer and Grinvald, 1991; Grinvald *et al.*, 1994) and monkeys (Blasdel, 1992b; Blasdel and Salama, 1986), but the patchy pattern of ocular dominance in cats (Anderson *et al.*, 1988; LeVay *et al.*, 1978; Löwel and Singer, 1987; Löwel *et al.*, 1988) differs somewhat from the stripe-like pattern in monkeys (Blasdel, 1992a, 1992b; Hubel *et al.*, 1977, 1978; LeVay *et al.*, 1975, 1985; Obermayer and Blasdel, 1993). In both species these patterns are evident at a spatial scale of about 1 mm.

The third O in the Triple-O concept concerns the existence of opponent simple cells at a much smaller spatial scale. That is, there exist nearby cortical simple cells which exhibit opposite spatial phase (Pollen and Ronner, 1981), and these cells may be connected by functionally

inhibitory connections (DeAngelis *et al.*, 1991; Liu *et al.*, 1992; Palmer and Davis, 1981). An arrangement of simple cells with complementary ON and OFF zones into mutually inhibitory pairs helps to explain the source of local intracortical inhibition, which provides functional antagonism between ON and OFF zones in simple cell receptive fields (Hubel and Wiesel, 1962). This complementary representation also helps to explain the robust expression of orientation tuning following blockade of ON retinal ganglion cells by the application of APB (Schiller, 1982). These facts are summarized well by models in which ON and OFF geniculate afferents synapse onto pairs of mutually inhibitory simple cells (e.g., Shulz *et al.*, 1993; Gove *et al.*, 1995).

Complex cells also respond to oriented stimuli, but do not have well segregated ON and OFF receptive field subregions. Complex cells are found in almost every layer of V1 (Gilbert, 1977), which is consistent with the intracortical feedback loops that exist, say, between layers 4, 2/3, and 6. Several models of how individual complex cells achieve their orientation tuning without segregated ON and OFF regions have been described which pool simple cell responses with differing spatial phases at a single complex cell (Emerson *et al.*, 1992; Gove *et al.*, 1995; Grossberg and Mingolla, 1985a , 1985b; Spitzer and Hochstein, 1985; Jacobson *et al.*, 1993).

Shared Properties of Cortical Map Development Models: A number of models have demonstrated how individual simple cell response characteristics and global maps can be simultaneously self-organized by local processes. One of the earliest models showed how a neural network with weights modified by an associative learning rule can produce orientation tuning when presented with oriented inputs (von der Malsburg, 1973; Grossberg, 1976b). Linsker (1986a, 1986b, 1986c) subsequently demonstrated the self-organization of orientationally tuned without oriented inputs. Other modeling work has shown how ocular dominance maps can arise from uncorrelated inputs (Swindale, 1980; Kohonen, 1982, 1989; Miller *et al.*, 1989; Rojer and Schwartz, 1989, 1990), how maps of orientation can form (Swindale, 1982), how maps of orientation and ocular dominance may develop simultaneously (Durbin and Mitchison, 1990; Obermayer *et al.*, 1990; Obermayer *et al.*, 1992; Sirosh and Miikkulainen, 1994; Swindale, 1992), and how the development of orientationally tuned simple cells and their arrangement into cortical maps may progress synchronously (Miller, 1992, 1994). Each of these models computes its maps with somewhat different equations. Some models, for example, focus on the learning that alters neural connections without modeling the dynamics of the cells themselves; e.g. Miller (1992, 1994). The fact that all of these models realize three computational principles (Grossberg and Olson, 1994) — a source of noise, a band pass filter, and normalization across all feature dimensions — clarifies what all these different models have in common from a computational viewpoint. Grossberg and Olson (1994) showed that these three factors are sufficient to generate cortical maps which exhibit the singularities, fractures, and linear zones that are found *in vivo* (Blasdel, 1992a, 1992b).

The neural model for cortical map development that is briefly reviewed here (Olson and Grossberg, 1998) builds upon these earlier developmental models. This model demonstrates the self-organization of cortical maps of ocular dominance and orientation, while simultaneously developing neighboring orientationally tuned simple cells that are sensitive to opposite contrast polarities, and that exhibit either even-symmetric or odd-symmetric receptive fields. These paired simple cells provide a natural explanation for such facts as how subcortical application of APB influences cortical orientation tuning and how cortical complex cells come to pool signals from oppositely polarized simple cells within a developing cortical map.

Opponent Simple Cells and Habituative Rebounds: In order to achieve these results,

the dynamics of both cortical cells and their intercellular interactions needed to be explicitly modeled. In particular, the model starts with arrays of spatially contiguous cortical cells that interact in pairs. (More loosely organized cell groupings would also work, but cell pairs are the simplest case.) These cells have no significant orientational preference before development occurs; they are activated by bottom-up inputs whose connection strengths are randomly chosen. The cell pairs do, however, have the property that offset of activity in one cell of a pair can lead to a transient antagonistic rebound of activity in the opponent cell of the pair. The cells in each pair are called ON cells and OFF cells because *offset* of an input to an ON cell can trigger transient activation in the corresponding OFF cell. This rebound is caused by an interaction of three factors: some of the cells interact via opponent competition; the chemical transmitters in some of the network pathways habituate in an activity-dependent way; and some of the cells receive an internal source of tonic activation. When these three factors are properly arranged in circuits (e.g., Figure 11) then, when an ON cell is activated by a bottom-up input, the transmitter that is released by the input activates the cell, but it also habituates, or depresses, in an activity-dependent way (Abbott *et al.*, 1997; Grossberg, 1972, 1976b). When the input to the ON cell turns off, the transmitter habituation lasts for a while afterwards. The OFF cell transmitter is not habituated to the same extent because the OFF cell was not active during this time.

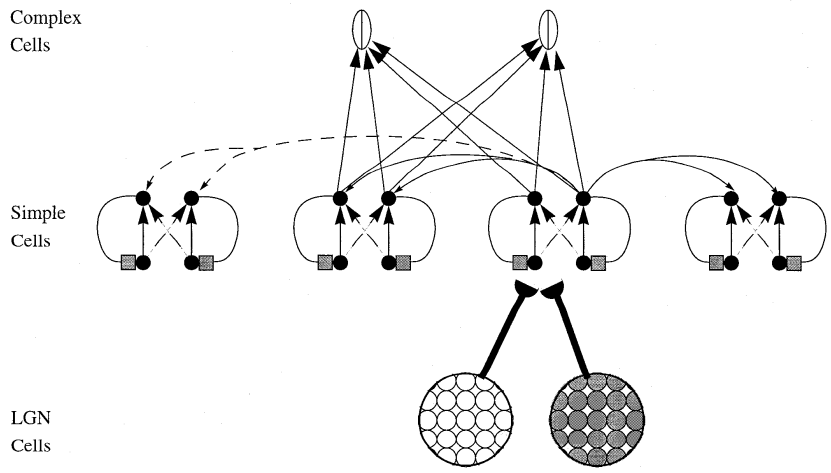

Figure 11. Pairs of unoriented cells (that will become simple cells) interact via opponent competition (dashed lines between simple cells) and habituative transmitters (square synapses). In addition, longer-range on-center recurrent excitation (horizontal solid lines between simple cells) and still longer-range off-surround recurrent inhibition (horizontal dashed lines between simple cells) exist. Inputs that are received from the lateral geniculate nucleus (LGN) ON cells (open circles) and OFF cells (gray disks). Adaptive weights under learning in the geniculocortical pathways (hemidisk synapses). Complex cells respond to a weighted sum of simple cell responses at the same cortical locations. All interactions are shown only with respect to one cell. [Reproduced with permission from Olson and Grossberg (1998)].

The bottom-up input is not the only input that can activate these transmitters. A tonically active input to both ON and OFF cell is also present and activates the ON and OFF cells equally; that is, it is a *nonspecific* input. When the input to the ON cell shuts off, the tonic input can activate the OFF cell more than the ON cell, because the ON cell transmitter is more habituated,

or depressed. After opponent competition between the cells occurs, there is a net OFF activation, leading to an antagonistic rebound of activity. The rebound is transient — that is, lasts only for a short amount of time — because the equal tonic inputs to both the ON and OFF cells gradually habituates the transmitters to both cells equally as well. Then the opponent competition between the cells shuts them both off.

Such antagonistic rebounds have elsewhere been used to explain psychophysical data about visual aftereffects (Francis and Grossberg, 1996; Grunewald and Lankheet, 1996), persistence (Francis *et al.*, 1994), and binocular rivalry (Grossberg, 1987), among others. Ringach *et al.* (1996) have reported direct neurophysiological evidence for rebound phenomena using reverse correlation techniques to analyze orientational tuning in neurons of cortical area V1.

Recurrent On-Center Off-Surround Network Dynamics: When embedded in a model whose mechanisms realize the three computational properties listed above — namely, a source of noise, a band pass filter, and normalization across all feature dimensions — these opponent cells develop into simple cells with similar orientation tuning but sensitivity to opposite contrast polarities. These additional mechanisms include medium-range recurrent excitation and long-range recurrent inhibition which interact with the short-range opponent mechanism (Figure 11). These longer-range interactions tend to normalize the total activity across the simple cells. They also contrast-enhance the inputs that are received from the lateral geniculate nucleus (LGN) ON cells (open circles) and OFF cells (black disks). When a random input first activates the LGN, it is filtered by the (then) random, and small, adaptive weights in the pathways between the LGN and the simple cells. The simple cells that receive the largest inputs win the contrast-enhancing competition that is realized by the recurrent on-center off-surround interaction. This interaction selects the winning cells and contrast-enhances their activity. Once the winning simple cells are selected, their activity helps to drive learning in the adaptive weights, or long-term memory traces, that exist within the synapses that abut them (see the hemidisks in Figure 11).

How do antagonist rebounds influence this learning process? When the input to a selected simple cell shuts off, an antagonistic rebound activates its corresponding OFF cell. At the same time, input offset also causes antagonistic rebounds of activity in the LGN. The adaptive weights in the pathways between the rebounded LGN cells and the rebounded simple cells learn the correlation between their activations. An antagonistic rebound in the LGN represents the same spatial pattern of activation as its ON response, but with an opposite contrast polarity. As a result, while the LGN-to-ON cell weights learn to code a prescribed orientation, the LGN-to-OFF cell weights learn to code the same orientation, but with an opposite contrast polarity. This property is schematized in Figure 12.

This opponent learning is more complicated than stated here, because OFF cell learning must also respond to direct LGN inputs to the OFF cells that are due to external inputs that have opposite contrast polarity from those that activate the ON cells. Computer simulations have demonstrated that the rebounded activity is sufficient to bias this learning to achieve the desired result; namely, the network can develop pairs of nearby simple cells that are sensitive to opposite contrast polarities but the same orientation (Figure 13). A second important result is that non-overlapping, oriented ON and OFF subregions develop in the model geniculocortical cell weights. This property helps to explain how simple cells in primary visual cortex receive direct excitatory connections from distinct regions of the LGN (Liu *et al.*, 1992; Reid and Alonso, 1995). These distinct ON and OFF subregions provide direct oriented input to cortical simple cells (Schiller, 1982; Hawken and Parker, 1984; Reid and Alonso, 1995; Ferster *et al.*, 1996).

The Triple-O model thus suggests how prenatal development leads to the segregation of initially intermingled ON and OFF inputs to cortical cells into oriented excitatory subregions.

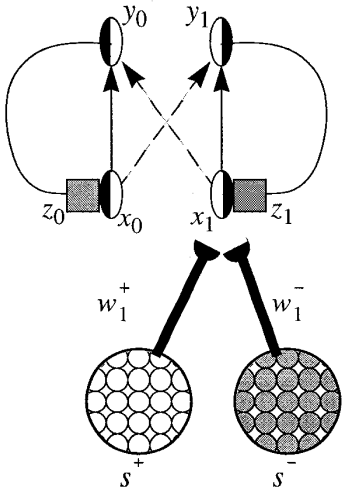

Figure 12. Local connectivity of opponent simple cells consists of a pair of input cells $x_i(j,k)$, feedback cells $y_i(j,k)$, and habituating transmitters $z_i(j,k)$ which make up two channels, corresponding to units with subscript 0 and to units with subscript 1, respectively. Each simple cell $x_i(j,k)$ receives LGN ON (+) and OFF (–) signals along weighted pathways. Vertical feedforward excitation (solid arrows) within and reciprocal feedforward inhibition (dashed arrows) between the channels produce an antagonistic relationship between simple cells. Positional indices (j,k) have been dropped. [Reproduced with permission from Olson and Grossberg (1998)].

Instar Synaptic Learning Law: Model adaptive weights are modified according to an associative learning rule that is called the *instar* learning rule or *gated steepest descent* learning rule (Grossberg, 1976a, 1976b; see also Kohonen, 1989; Obermayer *et al.*, 1992; Singer, 1983). According to this learning rule, changes in the weights are made only when the postsynaptic simple cell is active. Then the weights slowly change to track the input signals which they sense within their pathways. The instar learning rule normalizes the weights when activity in the presynaptic and postsynaptic neural fields is normalized by their on-center off-surround interactions. Normalization tends to render the sum of the weights that impinge on each cortical cell to be approximately constant.

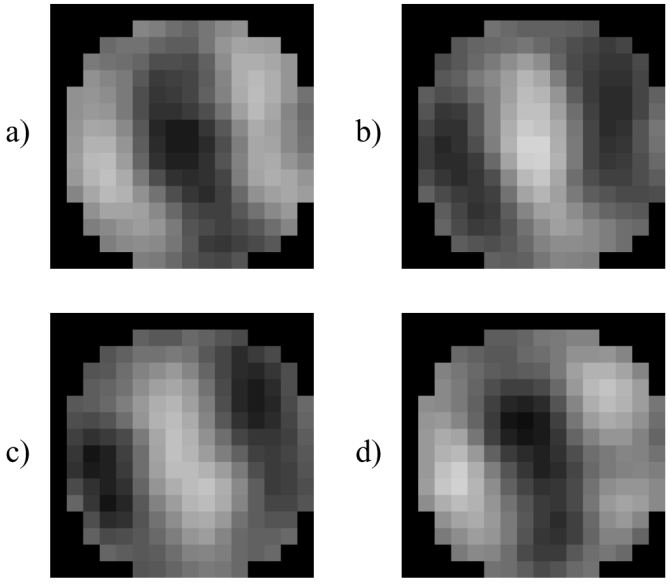

Figure 13. Opponency of learned adaptive weights: Model development leads to opposite polarity ON and OFF adaptive weights in opponent simple cells. (a) and (b) represent the ON and OFF weights corresponding to a given simple cell; (c) and (d) represent the ON and OFF weights corresponding to the opponent simple cell. Note that where ON cell weights are strong, OFF cell weights are weak, and conversely. [Reproduced with permission from Olson and Grossberg (1998)].

Map Properties: Singularities, Linear Zones, and Fractures: When all the mechanisms in the model interact together, map properties emerge that are similar to those reported experimentally. In particular, the developed map exhibits the swirling, gradually changing character of biological orientation maps as well as the key features of these maps: singularities (regions of low orientational selectivity around which all other orientations are grouped), linear zones (regions in which orientation changes relatively linearly with cortical distance), and fractures (regions in which orientation changes rapidly along one spatial direction and slowly or not at all in the orthogonal direction). Each of these key features is present in the simulated orientation map shown in Figure 14.

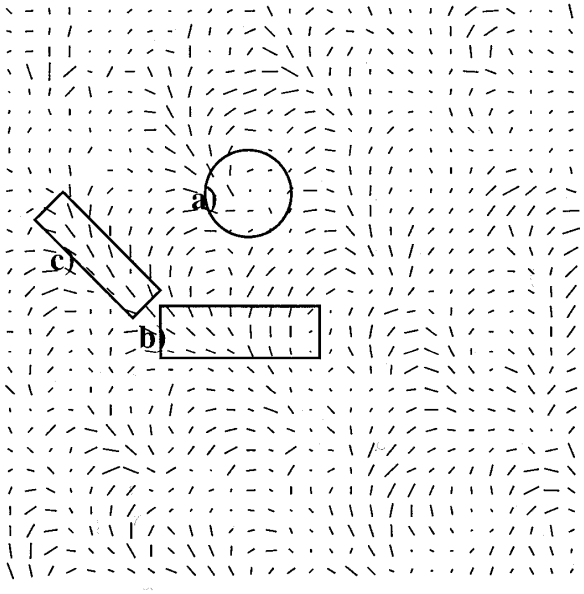

Figure 14. Orientation map: Subset of the simulated binocular orientation map. Key features of the biological orientation maps are present here: (a) singularities; (b) linear zones; (c) fractures. [Reproduced with permission from Olson and Grossberg (1998)].

In addition, ocular dominance maps, which reflect eye preference of cortical cells, also develop. An index of ocular dominance was computed by subtracting the total weight contributed by the ipsilateral eye from the total weight contributed by the contralateral eye at each cortical position. Figure 15 shows the orientation map superimposed on the map of ocular dominance. Regions dominated by the contralateral eye are colored white, and regions dominated by the ipsilateral eye are colored gray. As with cortical maps, this model map of ocular dominance is made up of interlaced dark and light patches corresponding to regions dominated by each eye. Ocular dominance and orientation preference are related in much the same way as they are in cortical maps: regions dominated by one eye or the other tend to line up with regions of low orientation selectivity, and regions of high orientational selectivity tend to be aligned with the borders of the ocular dominance bands (Blasdel, 1992b). Earlier modeling work has shown that using a spatial anisotropic filter can produce striped ocular dominance maps that even more closely resemble the patterns observed experimentally in monkeys (Swindale, 1980;

Rojer and Schwartz, 1989, 1990; Grossberg and Olson, 1994). This could be accomplished within the present modeling framework either through the use of an anisotropic pattern of lateral connections among simple cells or through an anisotropic pattern of geniculocortical connectivity. It remains to be seen if this enhancement naturally arises, for example, when the model in Figure 11 is embedded into the laminar model of Figure 4, including its anisotropic horizontal connections. Other properties of the Triple-O model include the development of both even-symmetric and odd-symmetric simple cells with well-segregated ON and OFF subregions. Because of the spatial arrangement of these simple cells, complex cells that are activated by a weighted average of simple cells across a small region do not have well-segregated ON and OFF subregions, and also pool responses from simple cells that are sensitive to opposite contrast polarities.

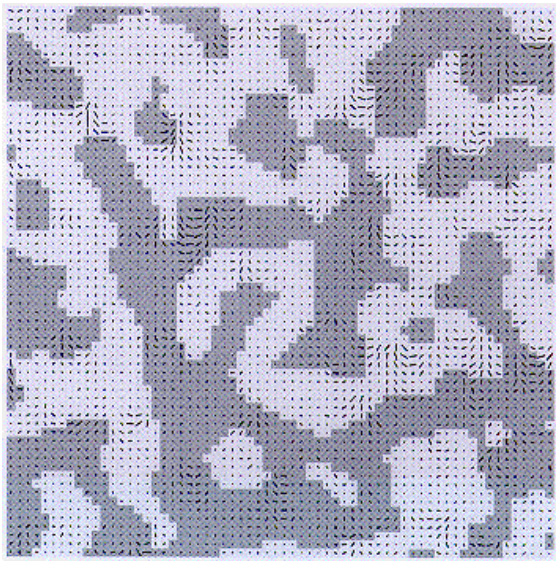

Figure 15. Orientation preference and ocular dominance maps: Orientation preference at each position is indicated by a line segment at the preferred orientation with length proportional to orientation selectivity. Regions dominated by the contralateral eye are colored white, regions dominated by the ipsilateral eye are grey. [Reproduced with permission from Olson and Grossberg (1998)].

This pooling process plays an important role in cortical models of visual perception. Because complex cells pool half-wave rectified output signals from pairs of oppositely polarized but similarly oriented simple cells, they compute an oriented, full-wave rectification of the image. Such an operation has become standard in models that explain data about human texture segregation (Grossberg and Mingolla, 1985b; Grossberg and Pessoa, 1998; Chubb and Sperling, 1989; Sutter *et al.*, 1989). Because complex cells pool signals from opposite contrast polarities, they can generate object boundaries around objects lying in front of textured backgrounds. In particular, a gray object lying in front of a black-and-white textured background will generate gray-to-white (light-to-dark) and gray-to-black (dark-to-light) contrasts along its perimeter. Because complex cells can respond to both contrast polarities, they can help to generate a boundary that encloses the entire gray object; see Figure 4a.

Rapid Development of Disparity-Sensitive Complex Cells

The Triple-O model does not show how connections from simple cells to complex cells develop. Nor does it indicate how complex cells can rapidly become sensitive to binocular disparity as a result of these connections. The study by Grunewald and Grossberg (1998) models how these events may occur. This model (Figure 16) suggests that both feedforward and feedback interactions among retinal, LGN, and cortical simple and complex cells contribute to the rapid development of highly tuned disparity-selective neural responses from the coarse level of stereopsis that is found in infants (Birch, Gwiazda, and Held, 1983; Blakemore, Hawken, and Mark, 1982; Blakemore and van Sluyters, 1974; Daw, 1994; Daw and Wyatt, 1976; Freeman and Ohzawa, 1992; Held, Birch, and Gwiazda, 1980; Leventhal and Hirsch, 1992; Movshon and Dusteler, 1977; Shimogo, Bauer, O'Connell, and Held, 1986). Some of the key modeling mechanisms that help to explain this developmental process will now be reviewed. Of particular interest are the top-down cortico-geniculate connections of the model, which help to assure that binocular disparity tuning can develop quickly without a loss of instability; that is, these top-down connections enable fast learning to occur without catastrophic forgetting, and thereby illustrate how Adaptive Resonance Theory principles can operate at even the earliest levels of visual processing (Grossberg, 1999b).

Competition and Habituative Rebound in Disparity Development: Monocularly activated simple cells from both eyes may initially activate a broad expanse of complex cells, as depicted in Figure 16. Contrast-enhancing competition across the complex cells determines a local winner, which tends to be the complex cell population that best matches the binocular disparity of the simple cells from both eyes which activate it. These winning complex cells can then learn, using instar learning, the pattern of activities that is active within the pathways from the active simple cells. As in the Triple-O model, the disparity-tuning model exploits dynamic rebounds between opponent ON and OFF simple cells that are due to imbalances in habituative transmitter gates. When these rebounds occur between oppositely polarized simple cells that are tuned to the same orientation, they help to explain how pairs of oppositely polarized simple cells, whose activity is *anti*-correlated through time, can become associated during development with a shared complex cell. The main idea is that, after a simple ON cell activates a particular complex cell, that complex cell's activity lingers for awhile after the simple cell shuts off. When the simple cell shuts off, an oppositely polarized simple cell is activated by an antagonistic rebound. This newly activated simple cell is then simultaneously active while the complex cell is still active. As a result, connections from this simple cell can be strengthened at the complex cell. This transient learning episode is enough to direct development of connections from both simple cells to the shared complex cell. In this way, complex cells develop that can pool opposite polarities of image contrast. At the same time, the simple-to-complex cell connections learn to binocularly fuse only stimuli for which both eyes process the same contrast polarity. Figure 17 illustrates a computer simulation of how disparity tuning evolves in time, including the fact that pairs of light-dark (LD) polarity and dark-light (DL) polarity simple cells develop the same disparity tuning.

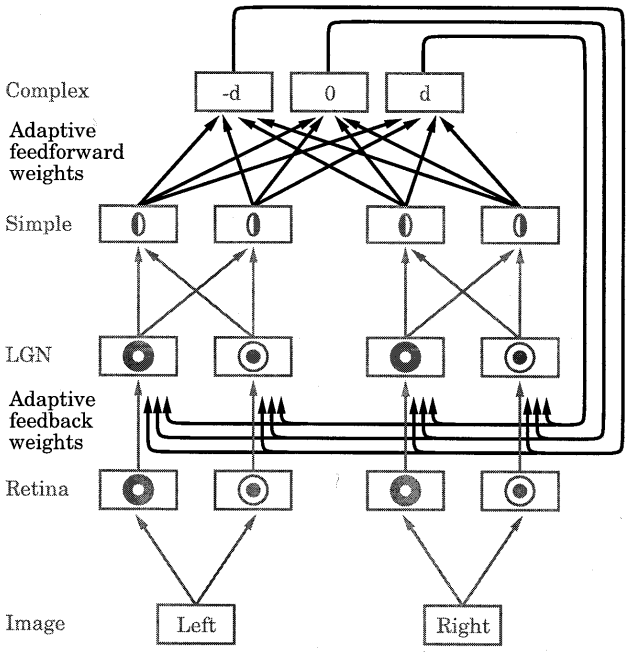

Figure 16. Model of how disparity-sensitive complex cells may develop. The dark lines indicate pathways that are adaptive and that contribute to this development. Symbols (-d, 0, d) stand for complex cells that will become sensitive to different disparities. [Reprinted with permission from Grunewald and Grossberg (1998)].

Cortico-Geniculate Matching in Stabilizing Disparity Tuning: The development of complex cell receptive fields and their disparity tuning in the model is impaired in the absence of either antagonistic rebounds or corticogeniculate feedback. The rebound response ensures that opposite-polarity simple cells develop the correct connections to the same complex cell. Corticogeniculate feedback prevents the learning of multiple disparity peaks and shifts in these peaks through time.

How is learning stabilized by corticogeniculate feedback in response to a changing visual environment? As in the LAMINART model, top-down ART matching properties seems to play an important role in this corticogeniculate pathway. In particular, whenever a complex cell emerges as a winner, a top-down matching, or confirmation, signal is sent to the LGN (Sillito, Jones, Gerstein, and West, 1994; Varela and Singer, 1987). When the confirmation signal matches the LGN activity pattern, then the matched LGN activities are amplified and synchronized. A mismatch between the confirmation signal pattern and the LGN input pattern leads to a reduction of LGN activity. This selective attenuation of mismatched LGN cells helps to stabilize the learning process and to trigger selection of a new complex cell winner if the match is bad enough. The data of Sillito *et al.* (1994) possess all of the predicted properties of the ART matching rule in this situation. In addition, Murphy, Duckett, and Sillito (1999) have shown that that the connections in the cortico-geniculate pathways share the orientation preference of the cortical cells that are involved.

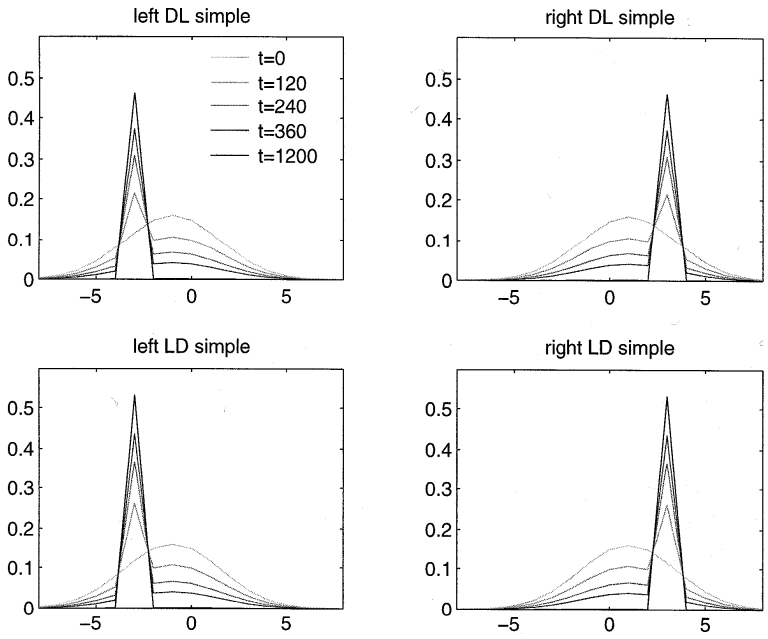

Figure 17. The development of individual bottom-up kernels to a complex cells of far, or uncrossed, disparity preference. The top panels show the kernels between DL simple cells and the complex cell. On the left is shown the kernel between the left DL simple cells and the complex cell, and on the right between the right DL simple cells and the complex cell. The bottom panel shows the kernels between LD simple cells and the same complex cell. Note that the DL and LD kernels are indistinguishable. Over time the kernels become narrower, and their preference shifts away from the central, zero-disparity location. [Reprinted with permission from Grunewald and Grossberg (1998)].

When the ART model was introduced in Grossberg (1976b), it was predicted that corticogeniculate feedback carries out a matching process in order to stabilize the development of cortical binocular tuning during the visual cortical period, and also that the top-down adaptive weights that control the matching process are also learned. The results from the Sillito lab are consistent with these predictions. It remains to be tested whether the bottom-up connections tend to get dynamically stabilized by the top-down corticogeniculate matching process. Gove, Grossberg, and Mingolla (1995) have predicted that elimination of the top-down matching process can cause the illusory brightness that is perceived in an Ehrenstein illusion to look dark, rather than bright. Grossberg and Grunewald (1998) have shown that the model can be used to explain psychophysical and neurobiological data concerning the dynamics of binocular disparity processing, including correct registration of disparity in response to dynamically changing stimuli, binocular summation of weak stimuli, and fusion of anti-correlated stimuli when they are delayed, but not when they are simultaneous (Cogan, Lomakin, and Rossi, 1993; Julesz, 1960). Simultaneous anti-correlated stereograms cannot be fused because only like-contrasts fuse. Delayed anti-correlated stereograms can be fused because the antagonistic rebound reverses contrast polarity, so the delayed response can be fused with the later response. More generally, the model's binocular circuit forms part of a larger theory of binocular vision that has been used to explain many data concerning 3-D vision and figure-ground separation (Grossberg, 1987,

1994, 1997; Grossberg and Kelly, 1999; Grossberg and McLoughlin, 1997; Grossberg and Pessoa, 1998; Kelly and Grossberg, 2000; McLoughlin and Grossberg, 1998).

Concluding Remarks

The present chapter reviews how key anatomical data about visual cortical development can be explained by recent neural models. The model cortical networks that emerge from these developmental processes can also explain a wide range of data about visual neuroscience and perception, notably data about binocular vision, perceptual grouping, and attention. This linkage of developmental, anatomical, neurophysiological, and perceptual data shows that the developmental hypotheses of the model are sufficient to generate cortical structures that can achieve key perceptual competences. In particular, the model's ability to simulate data about adult perceptual grouping after development is complete lends support to the hypothesis that the development of horizontal connections in cortical areas like V1 and V2 may be responsible for the rapid change in a human infant's ability, between the second and fourth month of life, to group image fragments into more complete object representations (Kellman and Spelke, 1983; Johnson and Aslin, 1996; Johnson, 2001).

Three types of models were reviewed here: the development of the cortical map of simple cells, which are the first stage of cortical processing; the development of complex cells, which receive inputs from simple cells; and the development of horizontal connections between complex cells, as well as the development of interlaminar connections that process inputs on their way to simple cells. The simulations of perceptual grouping and attention that derived from the third model assumed that the simple cells and complex cells had themselves already developed. Taken together, the three models clarify how the simple and complex cell properties themselves developed that were used to explain perceptual grouping and attention data. More generally, these component models provide a good foundation for the next steps of modeling cortical development in which all of these processes will be unified into a single comprehensive developmental model. Steps towards such a synthesis, along with an analysis of how the cortical subplate helps to coordinate development across the cortical layers, have already begun (Seitz and Grossberg, 2001).

None of the present developmental studies have considered how three-dimensional boundary and surface representations develop (although the development of disparity-sensitive complex cells is a step in that direction); how properties of figure-ground separation or motion perception develop; how processes or form and motion interact together; how boundary and surface representations are used for learned visual object recognition; or how these processes are realized within laminar circuits of visual cortex. Neural models of all these processes have, however, been described which are just a step away from these goals, and which utilize neural mechanisms similar to those described here; e.g., Baloch and Grossberg (1997), Bradski and Grossberg (1995), Grossberg (1994, 1997, 1999), Grossberg and McLoughlin (1997), Grossberg, Mingolla, and Viswanathan (2001), Grossberg and Pessoa (1998), Grossberg and Williamson (1999), Kelly and Grossberg (2000), and McLoughlin and Grossberg (1998). Thus the outlines of a general theory of visual perception are already discernable, along with some of the developmental mechanisms that lead to adult perception and recognition as we know it.

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