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Contrast-sensitive perceptual grouping and object-based attention in the laminar circuits of primary visual cortex

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Abstract

Recent neurophysiological studies have shown that primary visual cortex, or V1, does more than passively process image features using the feedforward filters suggested by Hubel and Wiesel. It also uses horizontal interactions to group features preattentively into object representations, and feedback interactions to selectively attend to these groupings. All neocortical areas, including V1, are organized into layered circuits. We present a neural model showing how the layered circuits in areas V1 and V2 enable feedforward, horizontal, and feedback interactions to complete perceptual groupings over positions that do not receive contrastive visual inputs, even while attention can only modulate or prime positions that do not receive such inputs. Recent neurophysiological data about how grouping and attention occur and interact in V1 are simulated and explained, and testable predictions are made. These simulations show how attention can selectively propagate along an object grouping and protect it from competitive masking, and how contextual stimuli can enhance or suppress groupings in a contrast-sensitive manner.

Keywords: Attention; Grouping; Layers; Striate cortex; Feedback

1. Introduction: attention and perceptual grouping in visual cortex

Perceptual grouping is the process whereby spatially distributed visual features become linked into object representations. There is evidence that it is a preattentive process that requires no top-down influences (e.g. Moore & Egeth, 1997). Attention enables observers to selectively process some object representations at the expense of others. It is clearly a top-down process. The past few years have seen an explosion of interest in the neurophysiological substrates of attention and perceptual grouping in visual cortex, at first in extrastriate areas but more recently also in striate cortex, or V1 (Grosof, Shapley & Hawken, 1993; Motter, 1993; Kapadia, Ito, Gilbert & Westheimer, 1995; Sheth, Sharma, Rao & Sur, 1996; Polat, Mizobe, Pettet, Kasamatsu & Norcia, 1998; Roelfsema, Lamme & Spekreijse, 1998; Watanabe, Sasaki, Nielsen, Takino & Miyakawa, 1998; Somers, Dale, Seiffert & Tootell, 1999). Anatomical studies have also revealed much of the intricate cortico-cortical and intracortical laminar circuitry of visual cortex which supports these processes (Callaway, 1998). However, explicit computational theories of how the cortical layers join these 'higher-order' perceptual functions to the better understood visual filtering processes have been lacking. In order to interpret the seemingly bewildering tangle of known laminar circuitry, an analysis is needed of the functional roles which these circuits subserve. Given the evidence cited above that those roles include attention and perceptual grouping, several sets of tight constraints emerge.

Three lines of neurophysiological and psychophysical evidence suggest that attention and grouping share some common mechanisms. Firstly, both processes act to enhance weak stimuli, but may have a neutral or even suppressive effect on stimuli that are already strong. In particular, the threshold level of contrast required to detect a target stimulus can be reduced, either by directing attention to the target location (Reynolds, Pasternak & Desimone, 1996) or by adding

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flanking stimuli with which the target can collinearly group (Kapadia et al., 1995; Polat et al., 1998). Such enhancement tends not to occur for higher-contrast stimuli (Reynolds et al., 1996; see also Hupé, James, Payne, Lomber, Girard & Bullier, 1998). This sort of contrast-dependent effect was found for perceptual grouping in the recent V1 study by Polat et al. (1998) (data shown in Fig. 1b), where neuronal responses to *low-contrast* Gabor patches were enhanced by the presence of collinear flankers outside the classical receptive field, but responses to Gabors which were well above contrast threshold were suppressed.

Secondly, attention and grouping both act to suppress rival stimuli. Distinct ways of perceptually grouping a scene compete against each other, for example the alternative horizontal and vertical groupings which may form when viewing a grid of dots (Kubovy, Holcombe & Wagemans, 1998). Spatially directed attention has an inhibitory off-surround which suppresses unattended stimuli (Somers et al., 1999), and which declines in strength with distance from the attended location (Caputo & Guerra, 1998). Both the suppressive and the enhancing effects of attention have been shown in work by Reynolds, Chelazzi and Desimone (1999) (data shown in Fig. 2d), in which attention directed to a target stimulus protects it from the competitive effects of a nearby distractor, and also suppresses neural responses to the distractor itself.

The third reason for suggesting partial sharing of their underlying mechanisms is that attention and perceptual grouping reciprocally interact. Although groupings may arise preattentively (Moore & Egeth, 1997), attentional task demands can influence which of various alternative groupings actually form, and these groupings in turn affect attentional phenomena such as the occurrence of illusory conjunctions (Prinzmetal & Keysar, 1989) or reaction times in a visual search task (Carrasco & Chang, 1995). Physiological recordings in striate cortex of macaques performing a visual curve-tracing task (Roelfsema et al., 1998, data illustrated in Fig. 3b) have shown that attention enhancement spreads along line segments which are grouped together to form a smooth curve, but not into contiguous segments which are grouped as a different object.

Despite these shared properties, attention and grouping must satisfy markedly different functional constraints if they are to avoid giving spurious output. Although top-down attention can slightly elevate the baseline firing rate of a neuron whose receptive field contains no visual stimulus (Luck, Chelazzi, Hillyard & Desimone, 1997), it does not produce suprathreshold neural activity of the sort that would be produced by bottom-up input. Violation of this constraint could cause continual hallucinations. However, illusory contours, prime examples of perceptual grouping, *do*, by definition, form over parts of visual space where there is no visual stimulus (von der Heydt, Peterhans & Baumgartner, 1984), clearly breaking the rule that attention must obey. Groupings must satisfy a quite different constraint referred to here as the 'bipole property' (Grossberg & Mingolla, 1985): an illusory contour may form between two or more inducers, such as the 'pacman' corners of a Kanizsa square, but cannot

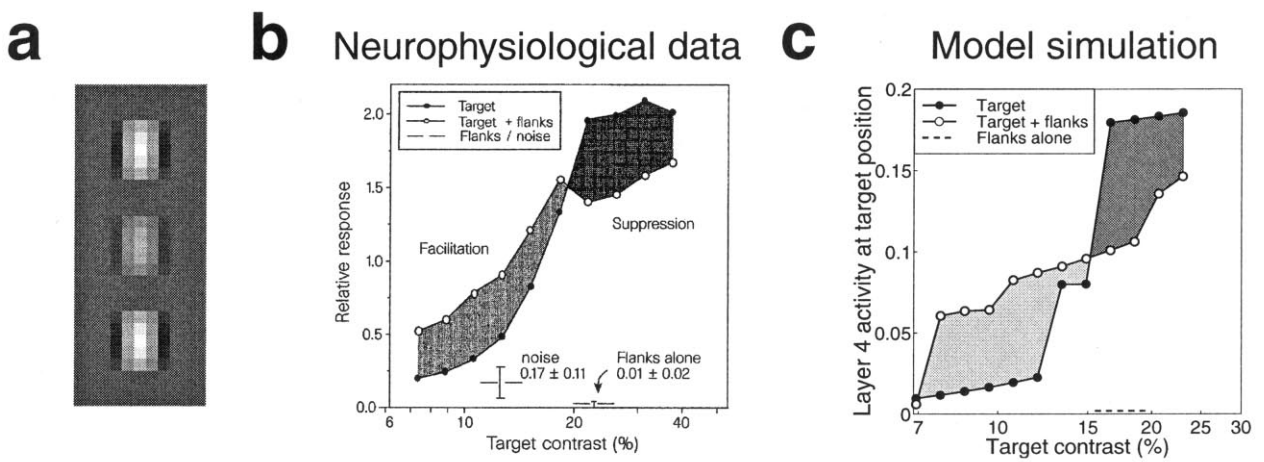

Fig. 1. Contrast-dependent perceptual grouping in primary visual cortex. (a) Illustrative visual stimuli. A variable-contrast oriented Gabor patch stimulates the classical receptive field (CRF), with collinear flanking Gabors of fixed high-contrast outside of the CRF. The stimulus shown here, based on those used Polat et al. (1998), was presented to the model neural network. (b) Neural responses recorded from cat V1. The collinear flankers have a net facilitatory effect on weak targets which are close to the cell's contrast-threshold, but they act to suppress responses to stronger, above-threshold targets. When the flankers are presented on their own, with no target present, the neural response stays at baseline levels. Reproduced with permission from Polat et al. (1998). (c) Model simulation of the Polat et al. data. See Section 3 for explanation of network behavior.

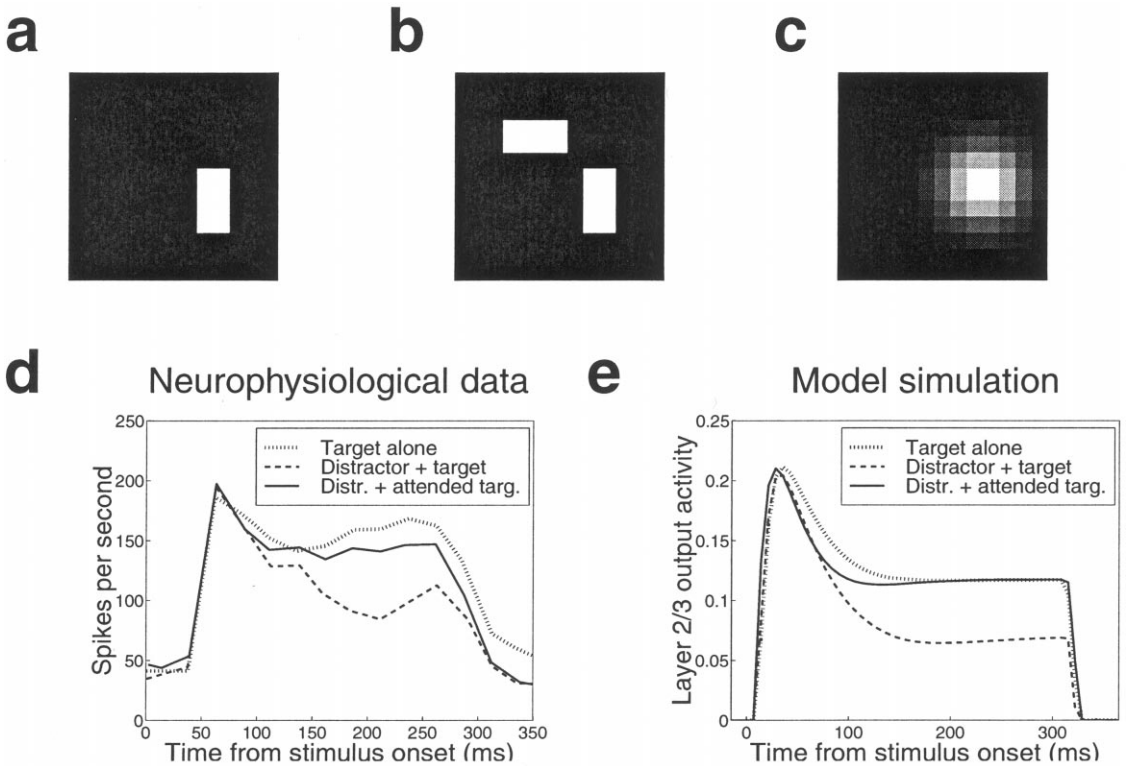

Fig. 2. The effect of attention on competition between visual stimuli. (a, b, c) Visual stimuli used in the experimental paradigm: a target stimulus, presented on its own (a), elicits strong neural activity. When a second, distractor stimulus is presented nearby (b), it competes against the target, and activity is reduced. Directing spatial attention to the location of the target stimulus (c), protects the target from this competition, and restores neural activity to the levels elicited by the target on its own. The stimuli shown here, based on those used by Reynolds et al. (1999), were presented to the model neural network. Spatial attention (c), was implemented as a Gaussian of activity fed back into layer 6. (d) Neurophysiological data from macaque V2, showing examples of the pattern described above: strong responses to an isolated target (dotted line), weaker responses when a competing distractor is placed nearby (dashed line) and restored levels of activity when the target is attended (solid line). Adapted with permission from Reynolds et al. (1999) (Fig. 5). (See also Reynolds, J., Nicholas, J., Chelazzi, L. & Desimone, R. (1995). Spatial attention protects macaque V2 and V4 cells from the influence of non-attended stimuli. *Society for Neuroscience Abstracts*, *21*, 693.1). (e) Model simulation of the Reynolds et al. data. The time-courses illustrated show the activity of a vertically oriented cell stimulated by the target bar. If only the horizontal, distractor bar were presented on its own, this cell would respond very weakly. If both target and distractor were presented, but with the horizontal distractor attended, the cell would respond, but more weakly than the illustrated case where the distractor and target are presented together, with neither attended. See Section 3 for explanation of network behavior.

sprout outwards from a single inducer, like a piece of stray hair (See Fig. 5). Violation of the bipole property would change perceptual grouping from an aid to scene segmentation into a source of distracting visual clutter.

How then, can attention and perceptual grouping play such similar roles, interact within primary visual cortex, and yet be subject to such different functional constraints, each of which would cause perceptual disaster if violated? We call this the *preattentive-attentive interface problem*. We suggest that the laminar structure of visual cortex implements a solution to this problem, and that a solution in the face such tight constraints provides the functional leverage needed to start interpreting what the cortical layers do. A discussion of the theoretical background to the new model can be found in Grossberg (1999a). Some of the present results have been previously presented in abstract form (Raizada & Grossberg, 1998, 1999b).

2. Model neural network

The laminar architecture of the present model is constructed out of two fundamental building blocks: an on-center off-surround circuit running from layer 6 to layer 4, and intrinsic horizontal connections in layer 2/3 which perform collinear integration and perceptual grouping. Each of these two subcircuits has assigned to it a well-defined functional role, and is constructed from model neurons with empirically determined connectivity and physiological properties, as summarised in Table 1. The anatomical structure and physiological behavior of these two functional subcircuits are illustrated in Figs. 4 and 5 respectively. When these building blocks are connected together according to the known anatomy of V1 and V2, as shown in Fig. 6, a cortical network is formed whose properties can be understood from the interactions of the functional subcircuits, but whose behavior is much richer than that of any subcircuit taken individually.

Attention in the model is mediated by a new mechanism that we call *folded feedback*, whereby signals from higher cortical areas, and also the V1 supragranular layers, pass down into V1 layer 6 and are then 'folded' back up into the feedforward stream by passing through the layer 6→4 on-center off-surround path (Figs. 4 and 6b), thus giving attention an on-center off-surround form, enhancing attended stimuli and suppressing those that are ignored.

Although facilitatory on-center attentional enhancement has been well-established, relatively few experiments have investigated possible suppressive off-surround effects, perhaps because the typical 'attended vs. unattended' paradigm cannot determine whether poorer 'unattended' responses are due to active inhibition or simply to the absence of facilitation. The necessary comparison is between a condition where a focus of attention is present but does not fall onto the probe stimulus, versus a baseline condition where the focus is absent or much further away from the probe. Posner, Snyder and Davidson (1980) demonstrated the attentional cost caused by an invalid cue with respect to

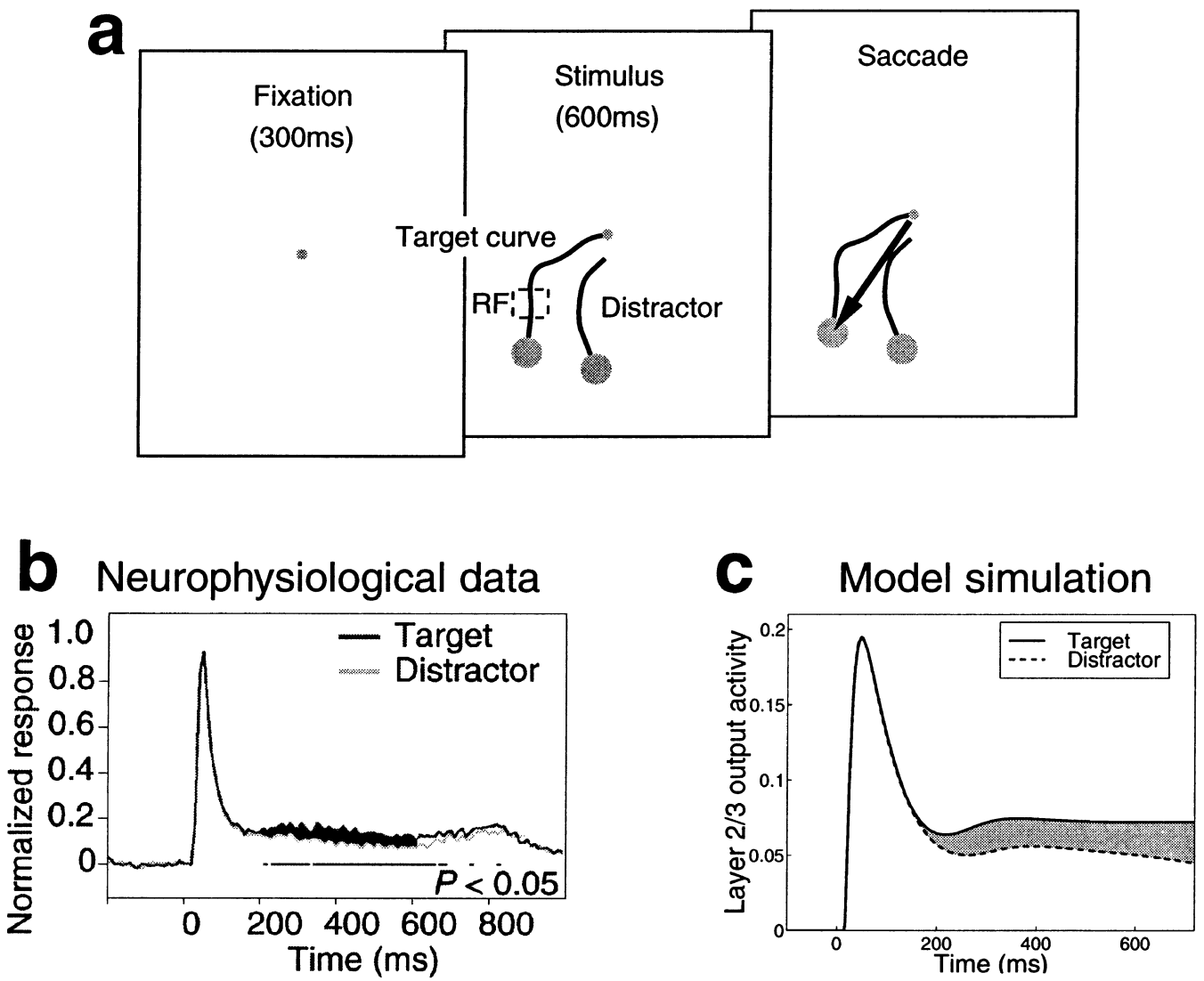

Fig. 3. Spread of visual attention along an object boundary grouping, from an experiment by Roelfsema et al. (1998). (a) The experimental paradigm. Macaque monkeys performed a curve-tracing task, during which physiological recordings were made in V1. A fixation spot was presented for 300 ms, followed by a target curve and a distractor curve presented simultaneously; the target was connected at one end to the fixation point. While maintaining fixation, the monkeys had to trace the target curve, then, after 600 ms, make a saccade to its endpoint. (b) Neurophysiological data showing attentional enhancement of the firing of a neuron when its receptive field (RF) lay on the target curve, as opposed to the distractor. Note that the enhancement occurs about 200 ms after the initial burst of activity. Further studies have indicated that the enhancement starts later in distal curve segments, far from the fixation point, than it does in proximal segments, closer to fixation (Pieter Roelfsema, personal communication). This suggests that attentional signals propagate along the length of the target curve. Figures (a) and (b) adapted with permission from Roelfsema et al. (1998). (c) Model simulation of the Roelfsema et al. data. See Section 3 for explanation of network behavior.

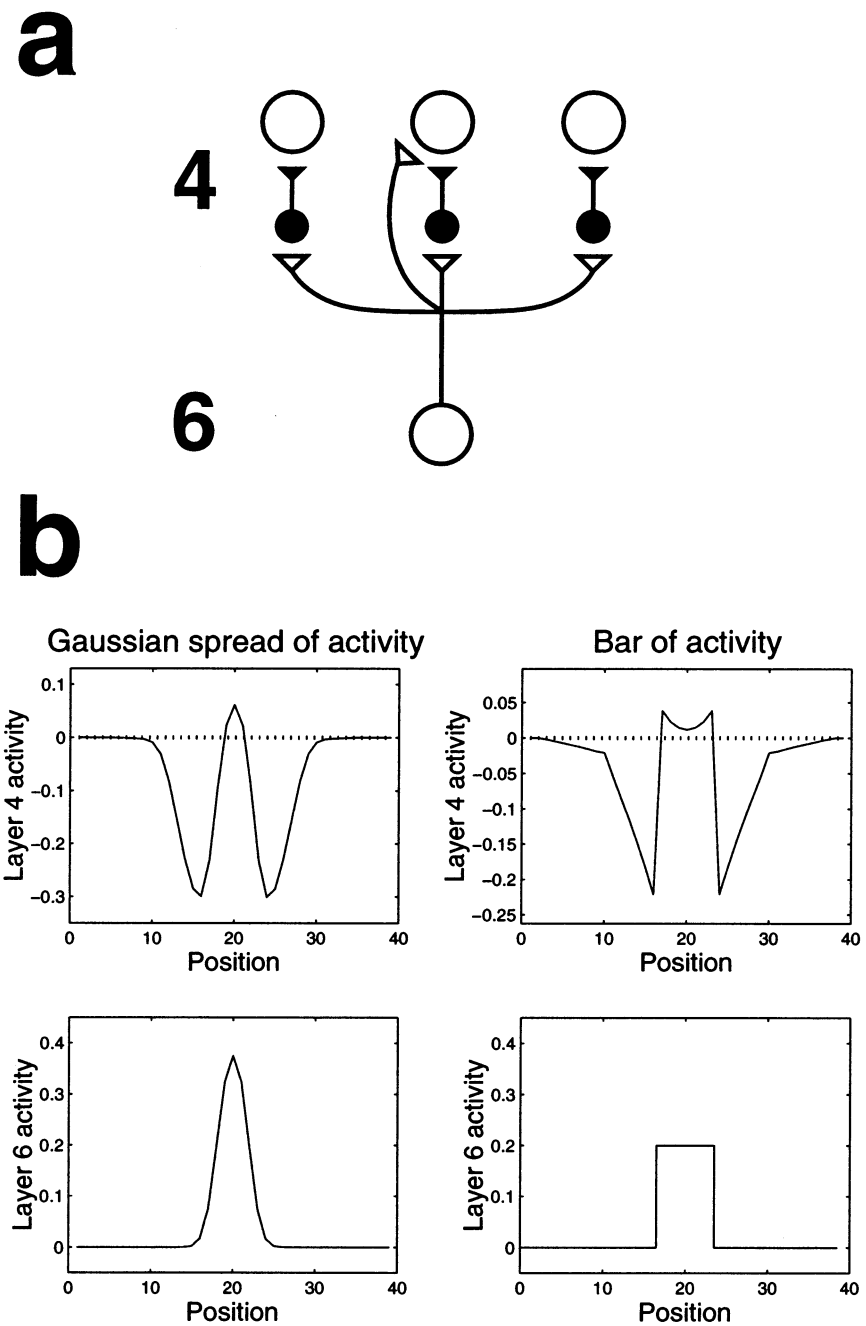

Fig. 4. (a) Schematic of the modulatory layer 6→layer 4 on-center off-surround path. Pyramidal cells in layer 6 give on-center excitation to layer 4 spiny stellates in the column above them, but also make medium-range connections onto layer 4 inhibitory interneurons, shown filled-in black (McGuire et 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, 1998). (b) Cross-sections of simulation output illustrating the modulatory on-center but strong off-surround of the 6→4 path. When a Gaussian spread of activation is created in just layer 6 of the network (bottom left), it gives rise to on-center excitation in layer 4. However, this excitation is only slightly above zero, having been partially balanced down by the overlapping off-surround (top left, dotted line marks zero activation, i.e. no net excitation.) A pool of strong inhibition surrounds the on-center. Attentional signals, which feedback from higher cortex into V1 layer 6, create a spread-out layer 6 activation profile of this sort. Thus, attention's characteristic modulatory on-center and strong off-surround (Somers et al., 1999) emerge as properties of the 6→4 path. (Bottom right) When a bar-shaped zone of activity is created in layer 6, it also gives rise to a weakly excitatory on-center and a strong inhibitory off-surround in layer 4 (top right). Feedback from above-threshold layer 2/3 boundary groupings via the 2/3→6 intracortical feedback path cause this sort of layer 6 activation profile Thus, these groupings subliminally prime their own layer 4 representations, and suppress layer 4 inputs to competing stimuli.

Table 1
Model neurons and their empirically determined connectivity and physiological properties[a]

Connection in model	Functional interpretation	Selected references
LGN → 4	Strong, oriented LGN input	Blasdel and Lund (1983); Ferster et al. (1996) (cat)
LGN → 6	LGN input sharpened by 6 → 4 on-center off-surround	Blasdel and Lund (1983)
6 → 4 Spiny stellates	Modulatory on-center of the 6 → 4 on-center off-surround	Stratford et al (1996) (cat), Callaway (1998) (p. 56)
6 → 4 Inhibitory Interneurons	Off-surround of the 6 → 4 on-center off-surround	McGuire et al. (1984) (cat), Ahmed et al. (1997) (cat)
4 Inhib. Int. → 4 Inhib. Int.	Context-dependent normalization of off-surround inhibition	Ahmed et al. (1997) (cat), Tamas et al. (1998) (cat)
4 → 2/3 Pyramidals	Feedforward of stimuli with bottom-up support	Fitzpatrick, Lund and Blasdel (1985), Callaway and Wiser (1996)
2/3 Pyr. → 2/3 pyr.	Long-range collinear integration along RF axes	Bosking et al. (1997) (shrew), Schmidt et al. (1997) (cat)
2/3 Pyr. → 2/3 Inhib. Int.	Keep outward grouping subthreshold (bipole property)	McGuire et al. (1984) (cat), Hirsch and Gilbert (1991)
2/3 Inhib. Int. → 2/3 Inhib. Int.	Normalize 2/3 inhibition (two-against-one part of bipole property)	Tamas et al. (1998) (cat)
V1 2/3 pyr. → V2 layer 4	Feedforward of V1 boundary groupings into V2	Van Essen et al. (1986), Rockland and Virga (1990)
V1 2/3 pyr. → V2 layer 6	Feedforward V1 groupings into V2 6 → 4 on-center off-surround	Van Essen, Newsome, Maunsell and Bixby (1986) (p. 470)
V1 layer 6 → LGN	Modulatory on-center off-surround feedback	Sillito et al. (1994) (cat), Montero (1991) (cat)
Feedback routes into V1 layer 6		
V2 layer 6 → V1 layer 1	Standard intercortical laminar feedback Patkin	Salin and Bullier (1995) (p. 110), Rockland and Virga (1989)
1 → 6 (within a layer 5 pyr.)	Corticocortical feedback into 6: Lay 5 pyr., apic.dend. in 1, axon in 6.	Lund and Boothe (1975) (Fig. 7), Gilbert and Wiesel (1979) (cat)
V2 (unknown layer) → V1 layer 6	Direct Corticocortical feedback into V1 layer 6	Gattass, Sousa, Mishkin and Ungerleider (1997) (Fig. 4)
2/3 → 6	Boundary groupings feedback into 6 → 4 on-center off-surround	Blasdel et al. (1989) (Fig. 13), Kisvarday et al. (1989) (Fig. 7)
1 → 5	Corticocortical fdbk into layer 5: pyr. with apic.dend. in 1	Valverde (1985); (Fig. 24o), Peters and Sethares (1991) (p. 7)
2/3 → 5	Part of indirect 2/3 → 6 path	Lund and Boothe (1975) (Fig. 8), Callaway and Wiser (1996)
5 → 6	Continuation of indirect routes into 6, via 5	Blasdel et al. (1985) (Fig. 17), Kisvarday et al. (1989) (Fig. 7)

[a] All references are to macaque monkey unless otherwise noted.

a neutral cue. This 'cost' however is not enough to entail the existence of an attentional off-surround with spatial structure. Using methods of signal detection theory, Downing (1988) showed that perceptual sensitivity, d', was above an uncued baseline level when the target was close to an attentional cue, and fell below baseline when the target was further away. Notably, she found that for brightness and orientation discrimination tasks, sensitivity rose back up to baseline levels at still greater distances from the cue, leading her to suggest that the attentional spatial sensitivity function might be best fit by an on-center off-surround difference-of-Gaussians (ibid., p. 196). Other psychophysical studies which have shown attentional off-surrounds include those by Steinman, Steinman and Lehmkuhle (1995) and Caputo and Guerra (1998). The study by LaBerge and Brown (1989) found an attentional gradient of decreasing performance with increasing distance from a cue, but did not speak to the issue of possible attentional off-surround suppression since it lacked a neutral baseline condition of the sort described above. Functional neuroimaging (Somers et al., 1999) and event-related potential (Luck, Hillyard, Mouloua, Woldorff, Clark & Hawkins, 1994) studies have provided physiological evidence for an attentional off-surround, but to our knowledge no single-unit neurophysiological study has yet addressed this question. Perhaps the closest to this are the studies by Connor, Gallant, Preddie & Van Essen (1996) and Connor, Preddie, Gallant & Van Essen (1997), who demonstrated a spatial gradient of attentional facilitation, but did not investigate possible inhibitory effects.

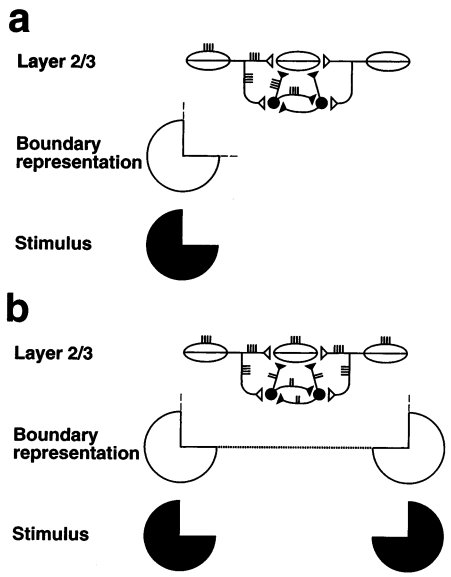

Fig. 5. Schematic of the boundary grouping circuit in layer 2/3. Pyramidal cells with collinear, coaxial receptive fields (shown as ovals) excite each other via long-range horizontal axons (Bosking et al., 1997; Schmidt et al., 1997), which also give rise to short-range, disynaptic inhibition via pools 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 to 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, subthreshold 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.

A key prediction of the model is that the on-center of the $6 \rightarrow 4$ path is modulatory (or priming, or subthreshold), consistent with the finding that layer 4 EPSPs elicited by layer 6 stimulation are much weaker than those caused by stimulation of LGN axons or of neighbouring layer 4 sites (Stratford, Tarczy-Hornoch, Martin, Bannister & Jack, 1996), and also with the fact that binocular layer 6 neurons synapse onto monocular layer 4 cells of both eye types without reducing these cells' monocularity (Callaway, 1998, p. 56). We suggest that the on-center excitation is inhibited down into being modulatory by the overlapping and broader off-surround (Fig. 4). Thus, although the center excitation is weak, the suppressive effect of the off-surround inhibition can be strong. Because attentional excitation must pass through the $6 \rightarrow 4$ path before it can effect visual processing, it inherits this path's properties: the attentional on-center is modulatory, able to enhance existing activity but only slightly to elevate neurons' baseline firing rates in the absence of visual input (Luck et al., 1997), but the off-surround can select strongly against unattended stimuli.

Several routes exist through which feedback from higher cortex can reach V1 layer 6, as shown in Table 1. Fig. 6(b) illustrates the route whereby feedback signals pass into layer 1, where the majority of V2 feedback axons terminate (Rockland & Virga, 1989), and then stimulate the apical dendrites of layer 5 pyramidal cells whose axons send collaterals into layer 6 (Lund & Boothe, 1975; Gilbert & Wiesel, 1979), where the attentional signals are 'folded' back up into the $6 \rightarrow 4$ on-center off-surround. Reversible deactivation studies of monkey V2 have shown that feedback from V2 to V1 does indeed have an on-center off-surround form (Bullier, Hupé, James & Girard, 1996), and moreover that the V1 layer whose activation is most reduced by cutting off V2 feedback is layer 6 (Sandell & Schiller, 1982).

We suggest that the mechanism of folded feedback is also used to help select the final layer 2/3 grouping. Like attentional signals from higher cortex, the groupings which start to form in layer 2/3 also feedback into the $6 \rightarrow 4$ path (Fig. 6c), to enhance their own positions in layer 4 via the $6 \rightarrow 4$ on-center, and to suppress input to other groupings via the $6 \rightarrow 4$ off-surround. There exist direct layer $2/3 \rightarrow 6$ connections in macaque V1, as well as indirect routes via layer 5 (Table 1). This competition between layer 2/3 groupings, via layer $2/3 \rightarrow 6 \rightarrow 4 \rightarrow 2/3$ feedback, causes the strongest grouping to be selected, while suppresses weaker groupings, ungrouped distractors, and noise. The interlaminar feedback also binds the cortical layers together into functional columns.

The fact that both attention and perceptual grouping share the properties of enhancing weak stimuli, and of suppressing signals from nearby rival inputs, can thus be parsimoniously explained by the hypothesis that both processes share the $6 \rightarrow 4$ folded feedback path. This laminar architecture also resolves the preattentive–attentive interface problem described above, since despite their shared properties and coexistence side-by-side within V1 and V2, attention and grouping behave quite differently in parts of visual space where there is no bottom-up visual stimulus. Above-threshold boundary groupings *can* form over regions with no

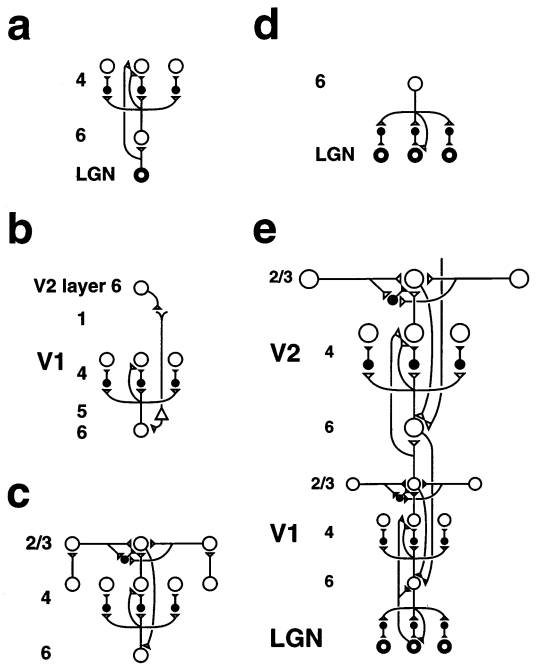

Fig. 6. How known cortical connections join the layer 6→4 (Fig. 4) and layer 2/3 (Fig. 5) building blocks to form the entire V1/V2 laminar model. Inhibitory interneurons are shown filled-in black. (a) The LGN provides bottom-up activation to layer 4 via two routes. Firstly, it makes a strong connection directly into layer 4. Secondly, LGN axons send collaterals into layer 6, and thereby also activate layer 4 via the 6→4 on-center off-surround path. Thus, the combined effect of the bottom-up LGN pathways is to stimulate layer 4 via an on-center off-surround, which provides divisive contrast normalization (Grossberg, 1973, 1980; Heeger, 1992) of layer 4 cell responses (see Appendix). (b) *Folded feedback* carries attentional signals from higher cortex into layer 4 of V1, via the modulatory 6→4 path. Corticocortical feedback axons tend preferentially to originate in layer 6 of the higher area and to terminate in the lower cortex's layer 1 (Salin & 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 6 exist (see Table 1 for references). 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). (c) 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 (Table 1). (d) Top-down corticogeniculate feedback from V1 layer 6 to LGN also has an on-center off-surround anatomy, similar to the 6→4 path. The on-center feedback selectively enhances LGN cells that are consistent with the activation that they cause (Sillito et al., 1994), and the off-surround contributes to length-sensitive (end-stopped) responses that facilitate grouping perpendicular to line ends. (e) The entire 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 an 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 & Schiller, 1982). Feedback paths from higher cortical areas straight into V1 (not shown) can complement and enhance feedback from V2 into V1.

bottom-up support, e.g. illusory contours (Fig. 5). These groupings form in layer 2/3. However, the only way top-down attentional signals can enter layer 2/3 is through the modulatory 6→4 path. Thus, attention can only modulate layer 2/3, but cannot on its own cause above-threshold activation, and the interface problem is resolved.

Some data suggesting that V2 feedback can actively *drive* the very V1 layer 2/3 neurons which themselves project forward to V2, rather than merely modulating them, as proposed by the present model, comes from a study of the cat by Mignard and Malpeli (1991). This result, we suggest, is unlikely to hold during normal primate visual processing, for three reasons: firstly, the Mignard and Malpeli study found evidence for V2 driving of supragranular V1 layers only during LGN inactivation. LGN cells are known to have high levels of spontaneous activity, even in the dark (Levick & Williams, 1964), and to project both to excitatory and inhibitory cortical neurons (Ahmed, Anderson, Martin & Nelson, 1997). Thus, removal of this tonic LGN input is likely to disrupt the balance of excitation and inhibition in V1, and with it the balance of feedforward and feedback influences. Secondly, the functional distinction between V1 and V2 is blurred in the cat, since both areas receive direct thalamic input (LeVay & Gilbert, 1976). Hence, feline V1/V2 connectivity may possibly bear more similarity to the intracortical circuits proposed in the current model than to the intercortical ones. Thirdly, there are strong functional reasons why top-down connections should be unable to drive feedforward paths in the absence of bottom-up input (c.f. the 'two-thirds rule' of Carpenter & Grossberg, 1987). In particular, a positive feedback loop of this kind could lead to over-sustained or even epileptiform activity. Feedback signals from V2 will tend to excite the layer 1 apical dendrites not just of pyramidals whose somata are in V1 layer 5, but also the dendrites of pyramidals in layer 2/3; here feedback from V2 seems to have a much weaker effect (Sandell & Schiller, 1982) a primate study which did not inactivate LGN), possibly because inhibitory interneurons with dendrites in layer 1 are present in the supragranular, but not the infragranular layers (Lund, 1987; Lund, Hawken & Parker, 1988; Lund & Wu, 1997). One possibility is that a balance between excitation and inhibition of the sort that keeps the layer 6→4 on-center subthreshold could

also mediate attentional feedback from layer 1 into layer 2/3, with the inhibitory interneurons ensuring that any direct attentional enhancement of layer 2/3 is kept modulatory rather than driving. This arrangement is not implemented in the present model, but its possible functional role has been analyzed, along with other V1 attentional and contextual effects, in a forthcoming paper (Raizada & Grossberg, 1999a).

3. Results

Computer simulations of the neural network described above demonstrate that its model neurons exhibit several types of behavior which have been observed experimentally in visual cortex (Figs. 1–3). The model's simulation of how attention affects competition between visual stimuli, as demonstrated by Reynolds et al. (1999), is shown in Figs. 2e and 8. This behavior follows from the properties of the layer $6 \rightarrow 4$ on-center off-surround path: when a distractor stimulus is presented nearby to the target, it inhibits the target through the $6 \rightarrow 4$ off-surround. Top-down attention, which in the simulation took the form of a two-dimensional Gaussian of activity directed at the location of the target, feeds back into layer 6, thereby activating its own on-center off-surround, with the on-center covering the target and the distractor falling into the attentional off-surround. Thus, the neural response to the target is increased, and the representation of the distractor is inhibited. Changes in layer 4 are passed on into layer 2/3 (plotted in Fig. 2e) which outputs to higher cortical areas. Attention thereby 'protects' the target from the distractor's competitive effects, as is observed experimentally. Note that for the simulation to work, the attentional Gaussian needs to be be able to cover the target without being so large that it also covers the distractor. The neurophysiological data of Reynolds et al. and related studies demonstrate that attentional foci can indeed be small enough to pick out one of two stimuli inside a single V2 receptive field, consistent with the mechanism proposed in the present model.

The key design issue underlying the model's architecture is the question of how attention and perceptual grouping interact (the preattentive-attentive interface problem, described above). Thus, a crucial test of the model is presented by the study by Roelfsema et al. (1998) of activity in macaque V1 during performance of a curve-tracing task, which provided evidence that attentional enhancement propagates between neurons which represent different segments of a smooth curve. Their data and the model network's simulation of it are shown in Figs. 3 and 9. Note that responses to the target curve are enhanced with respect to the distractor, but not until after a time delay of around 200 ms after stimulus onset. In the simulation, attentional signals were spatially directed only to that end of the target curve which corresponds to the fixation point. Attention took the form of a two-dimensional Gaussian of activity fed back into V1 layer 6, starting simultaneously with the onset of the visual stimulus itself. This attentional activity passed into the modulatory layer $6 \rightarrow 4$ path (Fig. 4), thereby strengthening the representation of the the end of the traced curve in layer 4, which in turn strengthened layer 2/3, where the extra activity propagated through intrinsic horizontal connections (Fig. 5) along the boundary representation of the curve. The delayed onset of the enhancement in the model, as observed in the experimental data, is because of the time taken for attentional signals to propagate laterally from their starting point at the end of the curve to the distal point on the curve, well outside the attentional on-center, from where the recorded activity was measured. Note that attentional feedback of the same strength as used here produced only subthreshold layer 2/3 excitation in a crucial control condition with attention presented in the absence of a bottom-up stimulus. This control also held for all the other simulations performed.

The third simulation is of the finding by Polat et al. (1998) of contrast-sensitive perceptual grouping in cat primary visual cortex (Figs. 1 and 7). The authors found that neural responses to a low-contrast target Gabor patch were facilitated when collinear flanking Gabor stimuli were added outside the receptive field, but that the flankers tended to suppress responses to Gabors that were of high enough contrast to cause above-threshold responses on their own (similar results were obtained in studies by Toth, Rao, Kim, Somers & Sur, 1996 and Sengpiel, Sen & Blakemore, 1997). As shown in Fig. 1(c), the model neurons also exhibit this behavior. The flankers exert both excitatory and inhibitory effects on the neurons whose receptive fields contain the target. Long-range horizontal axons in V1 layer 2/3, which link neurons with collinear receptive fields (see Fig. 5), carry excitation laterally from the flankers to the target. In V2 layer 2/3, this collinear facilitation has a longer range than it does in V1 (Fig. 6e), and a suprathreshold grouping forms between the two flankers, even when the target is absent or weak. The V2 grouping sends feedback via V2 layer 6 into V1, thus priming the V1 representation of the strip of space between the flankers, in particular the position of the target (Fig. 7a, b). This prime passes through the modulatory V1 layer $6 \rightarrow 4$ folded feedback path, therefore producing only subthreshold excitation in V1 layers 4 and 2/3 (the 'Flankers alone' condition in Fig. 1). Because of this top-down and lateral excitation, not as much bottom-up activity need come from the target itself for it to excite cells supraliminally. Thus, the flankers act to reduce the cells' target-contrast

threshold, raising the low-contrast section of the curve plotting neural response vs. target-contrast when the flankers are present.

However, the target also receives layer 6→4 off-surround inhibition from the flankers, which acts as a less specific 'lateral masking', as opposed to the collinear

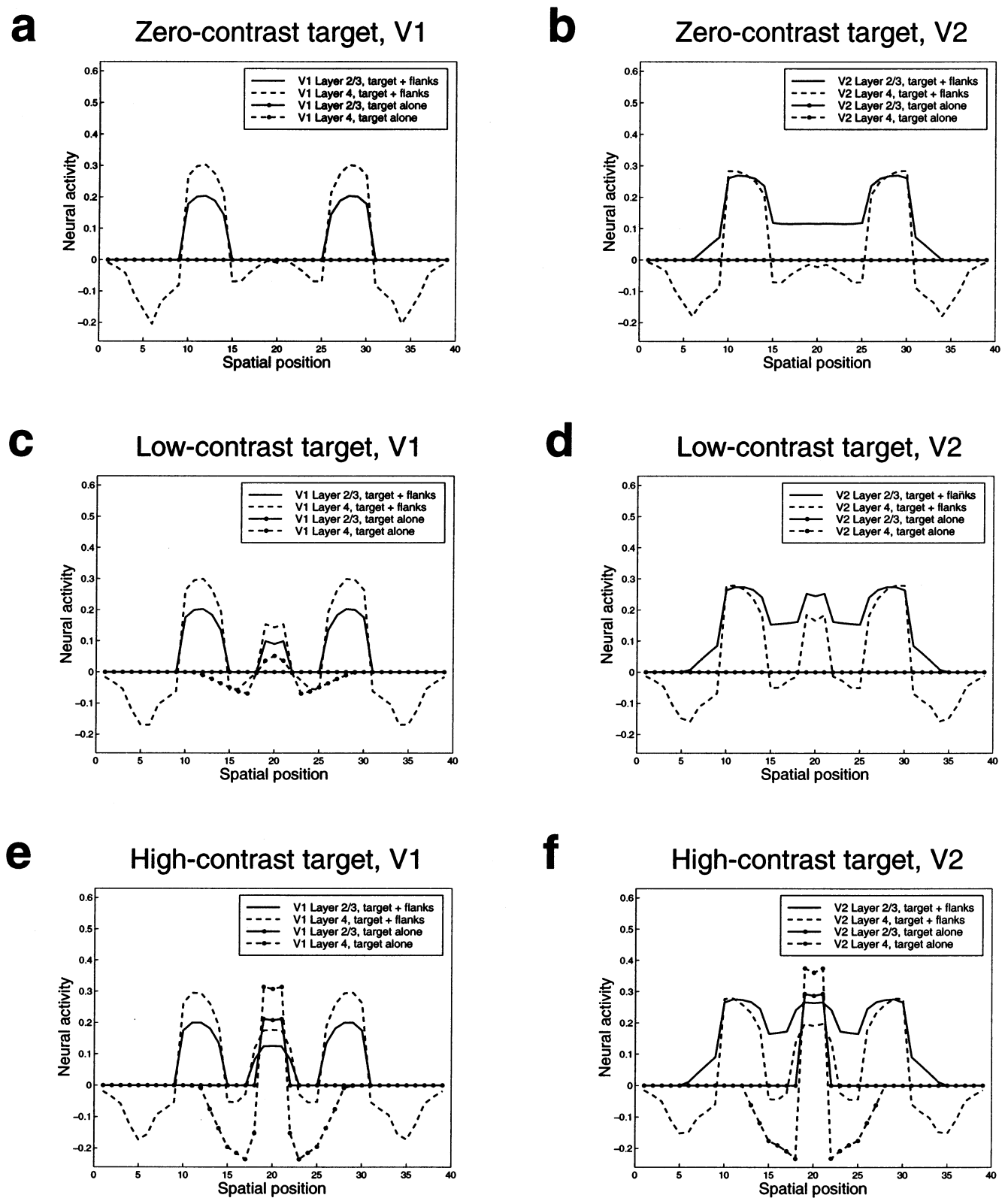

Fig. 7. (*Continued*)

facilitatory grouping carried by layer 2/3. This inhibition has a divisive, shunting effect (Grossberg, 1973, 1980; Heeger, 1992) on the target neurons (see Appendix A), with the consequence that equal increases in target contrast cause smaller rises in activity when the flankers are present than when the target is presented on its own. Thus, when the flankers are present, the slope of the neural response vs. target-contrast curve is reduced; the flankers-present response curve starts off higher (the flankers are net facilitatory), but then it rises more slowly and is overtaken by the flankers-absent curve when the isolated target exceeds threshold (the flankers become net suppressive), as found experimentally by Polat et al. (Fig. 1a). This 'cross-over' behavior occurs in layers 4 and 2/3 of the model V1. Note that in the model, as in the physiological data, the point at which the curves cross is determined by the threshold of the recorded simple or complex cell itself, not by the threshold of inhibitory interneurons which synapse onto it, as is postulated by other models (Stemmler, Usher & Niebur, 1995; Li, 1998; Somers, Todorov, Siapas, Toth, Kim & Sur, 1998).

4. Discussion

The model proposes laminar neural substrates for attention and the representation of visual groupings, or boundaries, and extends a general theory of how boundary and surface representations interact in the visual system (Grossberg & Mingolla, 1985; Grossberg, 1994; Grossberg, Mingolla & Ross, 1997): raw edge signals are pooled, sharpened and completed into closed boundaries, which are 'filled-in' by neural activity representing surface brightness and color. A full review (Grossberg, 1994; Pessoa, Thompson & Noë, 1998) of experimental evidence for this theory is beyond the scope of the current article, although particularly noteworthy are some recent neurophysiological (Lamme, Rodriguez-Rodriguez & Spekreijse, 1999) and psychophysical studies (Dresp & Grossberg, 1997; Elder & Zucker, 1998; Rogers-Ramachandran & Ramachandran, 1998), as is the recent demonstration by two independent laboratories (Lee, Mumford, Romero & Lamme, 1998; Rossi, Desimone, & Ungerleider, 1998) of fast filling-in of brightness signals in macaque V1, in the 50–100 ms timescale which has been observed psychophysically (Paradiso & Nakayama, 1991).

The model suggests that perceptual groupings are explicitly represented by the responses of oriented complex cells in layer 2/3 of the cortical network: visual objects are grouped, or bound together, by being *connected* by regions of above-threshold layer 2/3 firing. Thus, the discrete groupings within a visual scene will be the regions of layer 2/3 activity which are connected within themselves, but which are not connected to each other. Depending on whether the inducing stimuli also cause brightness differences across these boundary groupings, they may either be visible, like the illusory contours of a Kanizsa square, or recognized during form-processing but not actually seen as lines, as in a Glass pattern (see Fig. 8 of Grossberg (1994)). Examples of perceptually invisible groupings from the present simulations are those formed between the Gabor stimuli of Polat et al. (1998): the V2 groupings that connect the Gabors are not seen as illusory contours, because they do not separate regions of differing surface brightness (see Figs. 1 and 7). It has elsewhere been shown that bipole grouping by selective horizontal interactions can also rapidly synchronize the firing of the cells which are grouped together (Grossberg & Somers, 1991; Grossberg & Grunewald, 1997).

This type of representation of grouping has several advantages over those proposed by other computational models of visual cortex. Models which do not address the formation of illusory contours (Stemmler et al., 1995; Li, 1998; Somers et al., 1998; Yen & Finkel, 1998) not only fail to account for neurophysiological data (von der Heydt et al., 1984; Sheth et al., 1996) but also are unable to exploit the computational advantages that follow from closing incomplete boundaries: use of closure to guide surface reconstruction, boundary completion over the blind-spot and retinal veins, and more complete information for the recognition of partially occluded objects (Grossberg, 1994). Layer 2/3 bipole cells in the present model (Fig. 5) respond to both real

Fig. 7. Cross-sections of the model V1 and V2 neural activity for the simulation of the experiment by Polat et al. (1998). The sections are taken through the middle of the Gabor stimuli, and show the responses of vertically oriented cells. The panels in the left hand column; (a), (c) and (e), show V1 responses, and those in the right hand column; (b); (d) and (f), show V2. Each row illustrates the activity caused by a central target of zero, low or high-contrast respectively. Note that when no target is present (zero-contrast), a long-range grouping is formed between the two flanking Gabors in V2, shown in (b). This V2 grouping feeds back into V1 and primes the region of V1 layer 4 between the flankers (compare the almost zero layer 4 activity between the flankers with the strongly negative layer 4 inhibition to the flankers' sides). The operation of the bipole property can be observed by noting that a strong region of layer 2/3 activity forms between the two flankers, but only weak subthreshold fringes emerge at the sides, where grouping is insufficiently supported. (c) Without any flankers present, a low-contrast target causes some activity in V1 layer 4 (dotted-dash line) but is below threshold in V1 layer 2/3 (dotted-solid line) which stays at zero. Hence, no target activity passes up into V2. However, when flankers are present, feedback from V2 raises the weak target above-threshold, to a higher activity value (compare the undotted flankers-present lines to the dotted flankers-absent lines). This stronger target representation is passed up into V2, shown in (d). (e) and (f) When the target is high-contrast, it reaches threshold even without the flankers, whose net effect is now suppressive. See Section 3 for a full explanation of the network's behavior.

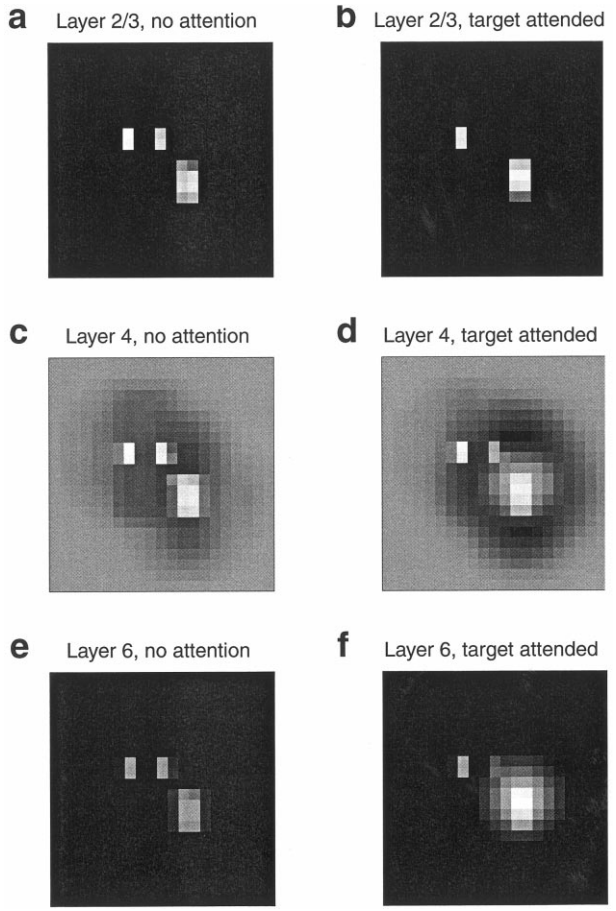

Fig. 8. Layer-by-layer two-dimensional plots of the network activity for the simulation of the experiment by Reynolds et al. (1999). The responses of vertically oriented cells are shown. Thus, the two short vertical islands of activity in the upper left correspond to the vertical end-cut responses to the ends of the horizontal distractor bar (see Grossberg and Mingolla, 1985). The panels in the left hand column, (a), (c) and (e), show activity when both the target and distractor stimuli are presented but with neither attended. Those in the right hand column; (b); (d) and (f), show activity when the target is attended. In layers 2/3 and 6, activity is half-wave rectified and hence always positive. Thus, black signifies zero activation. In layer 4, activity can be either positive or negative, and zero activation is represented by mid-level gray. Note especially the strong inhibition in layer 4 surrounding both stimuli, caused by the layer $6 \rightarrow 4$ on-center off-surround path. The inhibition surrounding the target becomes much stronger when the target is attended, due to the attentional off-surround, shown in (d). Thus, the attention acts to strengthen the target and weaken the distractor, thereby 'protecting' the target from the distractor's competitive effect.

and illusory contour stimuli of similar orientations, consistent with neurophysiological data (Sheth et al., 1996), and are connected by horizontal axons which are coaxial with the receptive fields' preferred orientation (Bosking, Zhang, Schofield & Fitzpatrick, 1997; Schmidt, Goebel, Löwel & Singer, 1997), not orthogonal, as has also been proposed (Peterhans & von der Heydt, 1991). Because groupings are explicitly represented by connected regions of above-threshold layer 2/3 firing, the model shows how a high-contrast item can group with its neighbors while still having its net neural response suppressed by their presence, as found by Polat et al. (1998). Models in which grouping is represented only by lateral facilitation (Stemmler et al., 1995; Somers et al., 1998) cannot account for this, and force the paradoxical conclusion that high-contrast items would never group with each other, which is demonstrably not the case (e.g. Elder & Zucker, 1998). The present model's representation of grouping as distinct from visible stimulus contrast also receives support from recent psychophysical work (Hess, Dakin & Field, 1998).

As shown in the simulation of the experiment by Polat et al. (1998), grouping in the model has a more facilitatory effect on low-contrast than on high-contrast visual stimuli (Figs. 1 and 7). This is also true of attention in the model; although high-contrast stimuli are indeed facilitated by attention, as in the simulations of the Reynolds et al. (1999) and Roelfsema et al. (1998) data (Figs. 2, 3, 8 and 9), lower-contrast stimuli tend to be facilitated even more, consistent with the findings of Reynolds et al. (1996) and Hupé et al. (1998). Two network properties cause grouping and attention to share this behavior: firstly, both processes feed into the layer $6 \rightarrow 4$ on-center off-surround pathway, where divisive shunting inhibition acts to reduce the gain on strong stimuli. Secondly, and relatedly, high-contrast stimuli excite cells to closer to their saturation points, leaving less room for any facilitatory boost.

By proposing detailed laminar circuits for how top-down attention operates within V1 and V2, the present model builds a connection between a theory of grouping in visual cortex (Grossberg & Mingolla, 1985; Grossberg, Mingolla & Ross, 1997) and a theory of how top-down feedback connections can stabilize learning and development, called Adaptive Resonance Theory, or ART (Grossberg, 1980, 1999a,b; Pollen, 1999). In ART, just as in the present model, modulatory on-center off-surround top-down attentional signals select and enhance behaviorally relevant bottom-up sensory inputs, and suppress those which are irrelevant. Mutual excitation between the top-down feedback and the bottom-up signals which they match strengthens and maintains existing neural activity long enough for synaptic changes to occur. Thus, attentionally relevant stimuli are learned, while irrelevant stimuli are suppressed and hence prevented from destabilizing existing memories. Recent experiments support both the predicted on-center off-surround top-down matching (Sillito, Jones, Gerstein & West, 1994; Bullier et al., 1996) and the role of attention in controlling adult plasticity and perceptual learning (Ahissar & Hochstein, 1993). These top-down feedback mechanisms also provide a means for higher-order perceptual operations, such as

sensitivity to figure vs. background (Grossberg, 1994), to modulate the activity of V1 cells (Lamme, Supèr & Spekreijse, 1998). The model's proposal that bottom-up sensory activity is *enhanced* when matched by top-down signals is in accord with a huge neurophysiological literature showing the facilitatory effect of attentional feedback (e.g. Luck et al., 1997; Roelfsema et al., 1998), but not with a recent model of visual cortex (Rao & Ballard, 1999) in which matches with top-down feedback cause suppression.

Finally, many testable neurophysiological predictions follow from the model's laminar functional architecture. A core prediction is that the layer $6 \rightarrow 4$ on-center should be subthreshold: in particular, intracellularly evoked layer 6 activity should modulate, but not drive, layer 4 spiny stellates and layer 2/3 pyramidals. The model proposes that attentional feedback into layer 6 passes into this modulatory $6 \rightarrow 4$ on-center to remain subthreshold in the absence of bottom-up visual input. Thus, it predicts that attentional elevation of a neuron's baseline firing rate when there is no stimulus in its receptive field, as observed by Luck et al. (1997), should cause above-threshold activation in layer 6, but below-threshold activation of layer 4 spiny stellates. Note that Luck et al. found this baseline elevation in V2 but not in V1. Since we suggest that the laminar mechanisms of attention are similar in both V1 and V2, differing only in spatial scale, we predict that this pattern of above-threshold attentional activation of layer 6 but not 4 should hold in both areas. It is possible that only very attentionally demanding tasks, requiring discriminations at fine spatial resolution, will reveal such activity in V1. A similarity between attention and grouping which the model proposes is that V2 groupings should feed back into V1 through the same pathway as attentional signals. For example, widely spaced collinear inducers (like the flankers in the study by Polat et al., 1998), should cause illusory contour activation in V2 layer 2/3, but not V1 layer 2/3, with feedback from this V2 grouping supraliminally activating V1 layer 6 but not 4, just like attention to empty space. Such detailed structural and functional predictions by the model will, we hope, help to stimulate further study of the laminar organization of visual cortex, as well as of other neocortical areas, whose horizontal interactions may be used to group different types of information, both as source and target of attentional modulation.

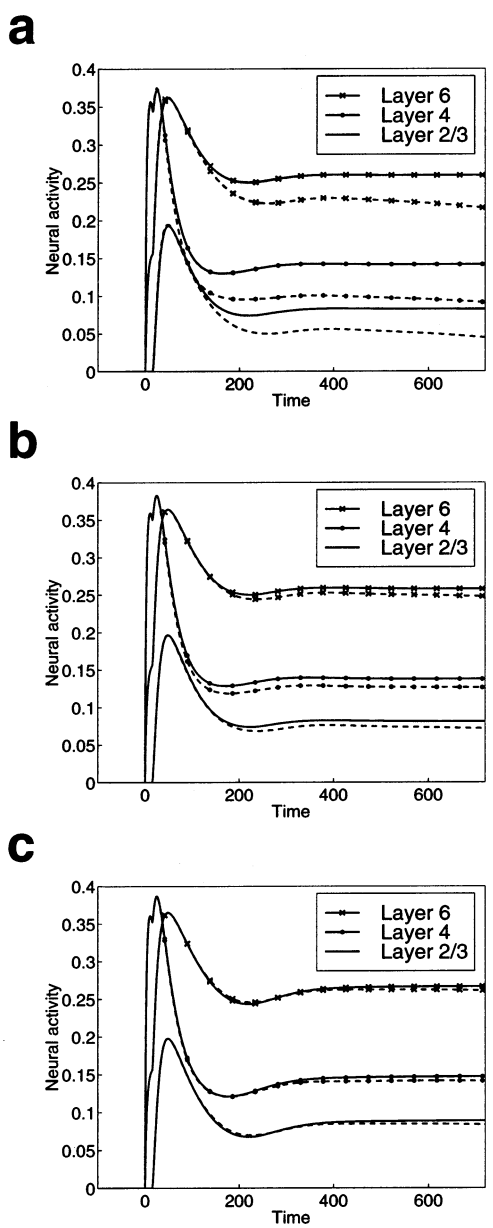

Fig. 9.

Fig. 9. Time-courses of activity across the layers of V1 at different positions along the traced curve in the simulation of the experiment by Roelfsema et al. (1998). Panels (a), (b) and (c) respectively show activity at increasing distances away from the attended end of the curve. For all panels, the solid lines represent activity when the receptive field of the recorded neuron falls on the target curve, and hence is attended, and dashed lines represent activity when the curve has become a distractor and is unattended. It can be seen that after an initial onset transient, attention produces a sustained enhancement of neural activity across cortical layers 2/3, 4 and 6. The inter-laminar layer $2/3 \rightarrow 6 \rightarrow 4$ feedback is responsible for yoking the layers' behavior together in this way. Note that at increasing distances from the attended zone, the strength of the attentional enhancement declines, and its latency increases. This can be seen most clearly by comparing the time at which the target and distractor activities start to diverge in panels (a) and (c). The weaker enhancement and longer latencies are caused by the gradual attenuation of attentional signals as they flow over time along layer 2/3. Note that although in these simulations layer 6 has stronger activity than layers 2/3 and 4, this is not a strong prediction of the model, since it is parameter-dependent. See the Discussion section for several testable predictions which derive from the model's functional architecture, rather than its parameter settings.

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Appendix A. Model equations

The model LGN, V1 and V2 were simulated as a network of interacting neurons, each with a single voltage compartment whose membrane potential, $V(t)$, was given by an equation of the form:

$$C_m \frac{\mathrm{d}V(t)}{\mathrm{d}t} = -[V(t) - E_{\mathrm{excit}}]\gamma_{\mathrm{excit}}(t) - [V(t) - E_{\mathrm{inhib}}]\gamma_{\mathrm{inhib}}(t) - [V(t) - E_{\mathrm{leak}}]\gamma_{\mathrm{leak}}. \quad (1)$$

In this equation, the time-varying conductances $\gamma_{\mathrm{excit}}(t)$ and $\gamma_{\mathrm{inhib}}(t)$ represent, respectively, the total inputs from the excitatory and inhibitory neurons synapsing onto the cell, as determined by the model architecture shown in Fig. 6. The γ_{leak} term is a constant leakage conductance, and the E terms represent reversal potentials. At equilibrium, the above equation can be written as $V = (E_{\mathrm{excit}}\,\gamma_{\mathrm{excit}} + E_{\mathrm{inhib}}\,\gamma_{\mathrm{inhib}} + E_{\mathrm{leak}}\,\gamma_{\mathrm{leak}})/(\gamma_{\mathrm{excit}} + \gamma_{\mathrm{inhib}} + \gamma_{\mathrm{leak}})$. Thus, increases in the excitatory and inhibitory conductances depolarise and hyperpolarise the membrane potential, respectively, as shown by the numerator of this term, and all the conductances contribute to divisive normalization of the membrane potential, as shown by the denominator. This divisive effect includes the special case of pure 'shunting' inhibition when the reversal potential of the inhibitory channel is close to the cell's resting potential (Borg-Graham, Monier & Fregnac, 1998). The following network equations are instances of this general membrane equation, where, for simplicity, the reversal potentials are set to: $E_{\mathrm{excit}} = 1$, $E_{\mathrm{inhib}} = -1$, $E_{\mathrm{leak}} = 0$, except where indicated. These continuous-time differential equations were implemented in Matlab and numerically integrated using a fourth-order Runge-Kutta algorithm until equilibrium was reached. Computed integrations were independently verified using Matlab's built-in adaptive-step-size second and third-order differential equation solvers.

A.1. Retina

The model retina has at each position (i, j) both an ON cell, u_{ij}^{+}, whose receptive field has the form of a narrow on-center and a Gaussian off-surround, and an OFF cell, u_{ij}^{-}, with a narrow off-center and a Gaussian on-surround (Schiller, 1992). As is observed in vivo, these ON and OFF cells feedforward into ON and OFF channels of the LGN, and enable the network to respond both to light increments and to light decrements. The retinal cell activities caused by constant visual inputs I have the equilibrium values:

$$u_{ij}^{+} = I_{ij} - \sum_{pq} G_{pq}(i, j, \sigma_1) I_{pq} \quad (2)$$

$$u_{ij}^{-} = -I_{ij} + \sum_{pq} G_{pq}(i, j, \sigma_1) I_{pq} \quad (3)$$

where $G_{pq}(i, j, \sigma)$ is a two-dimensional Gaussian kernel, given by:

$$G_{pq}(i, j, \sigma) = \frac{1}{2\,\pi\,\sigma^2} \exp\left(-\frac{1}{2\,\sigma^2}((p-i)^2 + (q-1)^2) \right). \quad (4)$$

The Gaussian width parameter was set to: $\sigma_1 = 1$.

A.2. Lateral geniculate nucleus

The ON and OFF cells of the LGN, v_{ij}^{+} and v_{ij}^{-}, are excited by the half-wave rectified ON and OFF cells of the retina, respectively. These retinal inputs are also multiplicatively gain-controlled by on-center off-surround feedback from V1 layer 6 (Sillito et al., 1994; Gove, Grossberg & Mingolla, 1995; Przybyszewski, Foote & Pollen, 1998). Layer 6 cells, x_{ijk}, at position (i, j) and of all orientations, k, send on-center excitation, A_{ij}, to LGN neurons at the same position, and send a two-dimensional Gaussian spread of off-surround inhibition, B_{ij}, to LGN neurons at the same and nearby positions:

$$\frac{1}{\delta_v}\frac{d}{dt} v_{ij}^{+} = -v_{ij}^{+} + (1 - v_{ij}^{+})[u_{ij}^{+}]^{+}(1 + A_{ij}) - (1 + v_{ij}^{+})B_{ij} \quad (5)$$

$$\frac{1}{\delta_v}\frac{d}{dt} v_{ij}^{-} = -v_{ij}^{-} + (1 - v_{il}^{-})[u_{ij}^{-}]^{+}(1 + A_{ij}) - (1 + v_{ij}^{-})B_{ij} \quad (6)$$

In Eqs. (5) and (6), the layer 6 on-center off-surround feedback terms, A_{ij} and B_{ij}, are given by:

$$A_{ij} = C_1 \sum_{k} x_{ijk} \quad (7)$$

$$B_{ij} = C_2 \sum_{pqk} G_{pq}(i,j,\sigma_1) x_{ijk}, \quad (8)$$

where the off-surround Gaussian, $G_{pq}(i, j, \sigma_1)$ is defined by Eq. (4), and the notation $[u_{ij}^{+}]^{+}$ signifies half-wave rectification, $[u_{ij}^{+}]^{+} = \max(u_{ij}^{+}, 0)$. The parameters for the LGN were: $\delta_v = 1.25$, $C_1 = 1.5$, $C_2 = 0.075$.

A.3. LGN inputs to cortical simple cells

Although the exact mechanisms through which simple cells gain their orientation tuning are still controversial (Reid & Alonso, 1995; Ferster, Chung & Wheat, 1996; Sompolinsky & Shapley, 1997; Adorjan, Levitt, Lund & Obermayer, 1999), recent intracellular studies of simple cells in layers 4 and 6 have revealed much about the structure and interaction of their ON and OFF subfields (Hirsch, Alonso, Reid & Martinez, 1998), supplementing and confirming earlier studies (Ferster, 1988). These results are incorporated, with some simplifications, into the current model.

At each position (i, j), and for each orientation, k, the model has a even-symmetric simple cell with two parallel elongated parts: an ON subregion, R_{ijk}, which receives excitation from LGN ON cells beneath it and is inhibited by LGN OFF cells at the same position; and an OFF subregion, L_{ijk}, which has the converse relation to the LGN channels (Reid & Alonso, 1995; Hirsch et al., 1998). This physiology is embodied in the equation for the ON subregion by subtracting the half-wave rectified LGN OFF channel, $[v^-_{pq}]^+$, from the rectified ON channel, $[v^+_{pq}]^+$, and convolving the result with the positive lobe of a Difference-of-Offset-Gaussians (DOOG) kernel, $[D^{(k)}_{ijk}]^+$, which has the simple cell subfield's characteristic oriented elongated shape. The OFF subregion, L_{ijk}, is similarly constructed:

$$R_{ijk} = \sum_{pq} ([v^+_{pq}]^+ - [v^-_{pq}]^+)[D^{(k)}_{pqij}]^+ \tag{9}$$

$$L_{ijk} = \sum_{pq} ([v^-_{pq}]^+ - [v^+_{pq}]^+)[-D^{(k)}_{pqij}]^+ \tag{10}$$

where the oriented DOOG filter $D^{(k)}_{pqij}$ is given by:

$$D^{(k)}_{pqij} = G_{pq}(i - \delta\cos\theta, j - \delta\sin\theta, \sigma_2) - G_{pq}(i + \delta\cos\theta, j + \delta\sin\theta, \sigma_2) \tag{11}$$

with $\delta = \sigma_2/2$ and $\theta = \pi(k-1)/K$, where k ranges from 1 to 2 K, K being the total number of orientations. For simplicity, the number of orientations was set to $K = 2$ (vertical and horizontal) in the present simulations. The width parameter for the DOOG filter was $\sigma_2 = 0.5$.

At an oriented contrast edge, a suitably oriented simple cell of the correct polarity will have its ON subfield stimulated by a luminance increment and its OFF subfield stimulated by an equal but opposite decrement. The optimal nature of this stimulus is embodied in the following equation, in which simple cell activity is the rectified sum of the activities of each subfield, minus their difference:

$$S_{ijk} = \gamma[R_{ijk} + L_{ijk} - |R_{ijk} - L_{ijk}|]^+ \tag{12}$$

Recent physiological studies have confirmed that layer 4 simple cells that are sensitive to opposite contrast polarities pool their outputs at layer 2/3 complex cells (Alonso & Martinez, 1998). In order to make the simulations manageable, cells in layers 6 and 4 were implemented with their simple cell inputs already pooled, thus halving the number of cells. Since the present model is not used to simulate any polarity-specific interactions in these layers, this simplification leaves the output unaffected. Thus, the polarity-pooled input from LGN to cortical layers 6 and 4 was calculated as the term C_{ijk}, which pools over opposite-polarity simple cells:

$$C_{ijk} = S_{ijk} + S_{ij(k+K)} \tag{13}$$

where k ranges from 1 to K. The parameter for the simple cell responses, was set to $\gamma = 10$.

A.4. Layer 6 cells

V1 layer 6 cells, x_{ijk}, receive input from the LGN (Blasdel & Lund, 1983), which, as described above, is represented by the contrast-polarity pooled oriented input, C_{ijk} They also receive two types of folded feedback excitation. The first type is intracortical feedback from above-threshold pyramidal cells in V1 layer 2/3, z_{ijk} (Blasdel, Lund & Fitzpatrick, 1985; Kisvarday, Cowey, Smith & Somogyi, 1989). These are passed through a thresholding signal function, F, given by:

$$F(z_{ijk}, \Gamma) = \max(z_{ijk} - \Gamma, 0), \tag{14}$$

where Γ is the threshold value. The second type of folded feedback is intercortical attentional feedback from V2, x^{V2}_{ijk} (Sandell & Schiller, 1982), originating in V2 layer 6 (Rockland & Virga, 1989). The feedback axons from V2 terminate predominantly in V1 layer 1 (Rockland, 1994). There exist several routes through which these layer 1 signals can pass down into layer 6, notably via the layer 1 apical dendritic tufts of layer 5 pyramidals with axon collaterals in 6 (Lund & Boothe, 1975; Gilbert & Wiesel, 1979, see also Table 1). These paths are not explicitly implemented in the present model. In attentional simulations where only the V1 part of the complete V1/V2 network is simulated, the corticocortical feedback term, x^{V2}_{ijk}, is replaced by a two-dimensional Gaussian spread of attentional signals, centered on the attended location and exciting all orientations equally. Thus:

$$\frac{1}{\delta_C}\frac{\mathrm{d}}{\mathrm{d}t}x_{ijk} = -x_{ijk} + (1 - x_{ijk}) \times (\alpha\, C_{ijk} + \phi\, F(z_{ijk}, \Gamma) + V_{21}x^{V2}_{ijk}), \tag{15}$$

This equation was solved at equilibrium, giving:

$$x_{ijk} = \frac{\alpha\, C_{ijk} + \phi\, F(z_{ijk}, \Gamma) + V_{21}\, x^{V2}_{ijk}}{1 + \alpha\, C_{ijk} + \phi\, F(z_{ijk}, \Gamma) + V_{21}\, x^{V2}_{ijk}}. \tag{16}$$

Parameters for the terms in the layer 6 equation were: $\delta_C = 0.25$, $\alpha = 0.5$, $\phi = 2.0$, $\Gamma = 0.2$, $V_{21} = 1.5$.

A.5. Layer 4 activity

Model spiny stellate cells in layer 4, y_{ijk}, as well as receiving the contrast-polarity pooled oriented input, C_{ijk}, described above, also receive on-center off-surround input from layer 6. The on-center consists of excitatory connections from layer 6, x_{ijk}, to layer 4 spiny stellates at the same position and of the same orientation (Stratford et al., 1996; Wiser & Callaway, 1997). The off-surround input is caused by medium-range projections from layer 6 onto layer 4 inhibitory interneurons (McGuire, Hornung, Gilbert & Wiesel, 1984; Ahmed et al., 1997). The spatial distribution and strength of these connections are determined by a two-dimensional kernel, W^{+}_{pqrijk}, which is in the present model a linearly scaled version of a self-organized 6–4 inhibitory kernel grown in the developmental study by Grossberg and Williamson (1998) using the same network architecture, but without the corticocortical feedback connections. The spatial distribution of this kernel, which is approximately Gaussian, is shown in Fig. 10(a). Therefore, the distribution of the off-surround inhibition in the present model is not hand-crafted by an algebraic equation, but is instead the product of a self-organized equilibrium reached by the same network architecture in response to naturally structured visual inputs. Thus, the equation for layer 4 spiny stellates is:

$$\frac{1}{\delta_C}\frac{d}{dt}y_{ijk} = -y_{ijk} + (1-y_{ijk})(C_{ijk} + \eta^{+}x_{ijk}) - (y_{ijk}+1)\sum_{pqr} W^{+}_{pqrijk}\, m_{pqr}. \tag{17}$$

This was solved at equilibrium, giving:

$$y_{ijk} = \frac{C_{ijk} + \eta^{+}x_{ijk} - \sum_{pqr} W^{+}_{pqrijk}\, m_{pqr}}{1 + C_{ijk} + \eta^{+}x_{ijk} + \sum_{pqr} W^{+}_{pqrijk}\, m_{pqr}} \tag{18}$$

Note that the 6–4 off-surround spatially overlaps with the on-center, with the consequence that the center excitation is inhibited down into being subthreshold, or modulatory (Callaway, 1998, p. 56). This property is the key to the attentional simulations performed, as discussed above (See Fig. 4).

Layer 4 inhibitory interneurons, m_{ijk}, also receive on-center off-surround input, the on-center again coming from layer 6 cells with the same position and orientation, x_{ijk}, and the off-surround inhibition coming via the spatial kernels, W^{-}, of the other inhibitory interneurons in layer 4 (Ahmed et al., 1997). These inhibitory-to-inhibitory synapses help to normalize the total amount of inhibition present at a given position in layer 4. Thus:

$$\frac{1}{\delta_m}\frac{d}{dt}m_{ijk} = -m_{ijk} + \eta^{-}x_{ijk} - m_{ijk}\sum_{pqr} W^{-}_{pqrijk}\, m_{pqr}. \tag{19}$$

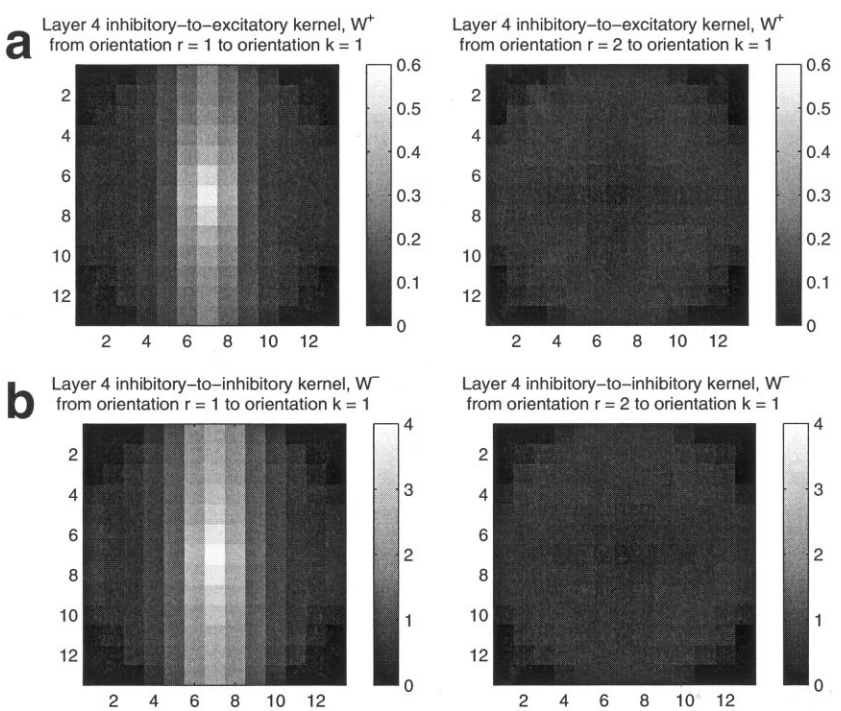

Fig. 10. (a) The inhibitory-to-excitatory off-surround kernels in Layer 4, W^{+}. Only the kernels operating on vertically oriented cells are shown, since those operating on horizontally oriented cells are the same, but rotated by ninety degrees. (b) The inhibitory-to-inhibitory off-surround kernels in layer 4, W^{-}. Again, only the vertical kernels are shown.

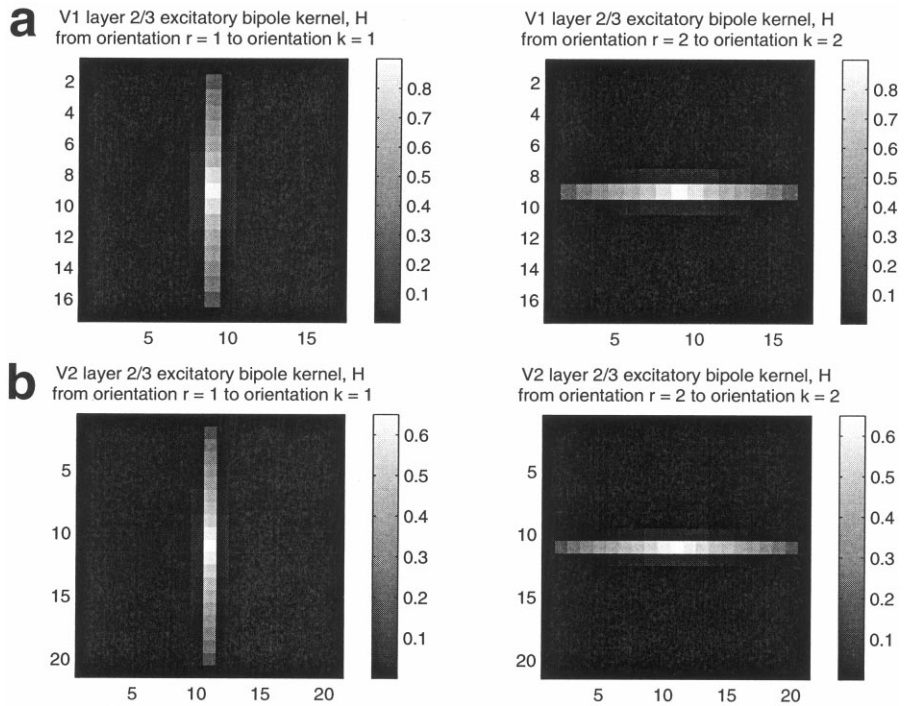

Fig. 11. (a) The bipole-grouping kernels in V1 layer 2/3, H. Since bipole facilitation is collinear, the cross-orientation bipole kernels have approximately zero strength. They are not shown. (b) The bipole-grouping kernels in V2 layer 2/3, H^{V2}. Note that they are longer-range than the corresponding V1 kernels.

As with the inhibitory-to-excitatory kernels, W^+, the inhibitory-to-inhibitory kernels, W^-, are also linearly scaled versions of the kernels which were self-organized in the model of Grossberg and Williamson (1998). They have a very similar spatial structure to the W^+ kernels, but are a little stronger, as shown in Fig. 10(b). Parameters for layer 4 were: $\delta_m = 0.01875$, $\eta^+ = 2.1$, $\eta^- = 1.5$.

A.6. Layer 2/3

Layer 2/3 performs collinear grouping, by implementing the bipole property, as illustrated in Fig. 5. The pyramidal cells in layer 2/3, z_{ijk}, receive excitatory input from Layer 4 cells, y_{ijk}, at the same position and orientation (Callaway & Wiser, 1996), and also long-range bipole excitation from the thresholded outputs of other layer 2/3 pyramidals with collinear, coaxial receptive fields, $F(z_{ijk})$ (Bosking et al., 1997; Schmidt et al., 1997). Inhibitory interneurons in layer 2/3, s_{ijk}, also synapse onto these pyramidals. As with the inhibitory kernels in layer 4, W^+ and W^-, the layer 2/3 cells synapse onto each other through linearly scaled versions of the self-organized kernels grown in the model of Grossberg and Williamson (1998). The excitatory-to-excitatory, long-range bipole kernels, H, are shown in Fig. 11. As well the long-range excitation, layer 2/3 pyramidals also receive short-range inhibition from inhibitory interneurons at the same position and of the same orientation, s_{ijk} (McGuire, Gilbert, Rivlin & Wiesel, 1991). This inhibition operates through a self-organized short-range kernel, T^+. Thus, the full equation for layer 2/3 pyramidals is as follows:

$$\frac{1}{\delta_z}\frac{\mathrm{d}}{\mathrm{d}t}z_{ijk} = -z_{ijk} + (1 - z_{ijk}) \times \left(\lambda[y_{ijk}]^+ + \sum_{pqr} H_{pqrijk}\, F(z_{pqr}, \Gamma)\right) - (z_{ijk} + \psi)\sum_r T^+_{rk}\, s_{ijr}. \quad (20)$$

The layer 2/3 inhibitory interneurons, s_{ijk} receive excitation only from layer 2/3 pyramidals, through the kernels H, and are inhibited by other layer 2/3 interneurons at the same position but of all orientations, via the self-organized short-range kernel, T^- (Tamas, Somogyi & Buhl, 1998):

$$\frac{1}{\delta_s}\frac{\mathrm{d}}{\mathrm{d}t}s_{ijk} = -s_{ijk} + \sum_{pqr} H_{pqrijk}\, F(z_{pqr}, \Gamma) - s_{ijk}\, T^-_{rk}\, s_{ijr}. \quad (21)$$

Parameters for layer 2/3 were: $\delta_z = 0.125$, $\delta_s = 2.5$, $\lambda = 2.5$, $\psi = 0.5$.

A.7. Feedforward projections from V1 to V2

The thresholded output of V1 layer 2/3 projects forward to layers 6 and 4 of V2, x^{V2}_{ijk} and y^{V2}_{ijk} respectively, following the same pattern as the LGN forward projections to layers 6 and 4 of V1. Hence:

$$\frac{1}{\delta_C}\frac{\mathrm{d}}{\mathrm{d}t}x_{ijk}^{V2} = -x_{ijk}^{V2} + (1 - x_{ijk}^{V2}) \times (V_{12}^{6} F(z_{ijk}, \Gamma) + \phi\, F(z_{ijk}^{V2}, \Gamma)) \quad (22)$$

$$\frac{1}{\delta_C}\frac{\mathrm{d}}{\mathrm{d}t}y_{ijk}^{V2} = -y_{ijk}^{V2} + (1 - y_{ijk}^{V2})(V_{12}^{4} F(z_{ijk}, \Gamma) + \eta^{+} x_{ijk}^{V2}) - (y_{ijk}^{V2} + 1)\sum_{pqr} W_{pqrijk}^{+}\, m_{pqr}^{V2}. \quad (23)$$

All other equations and parameters for V2 are exactly the same as for the corresponding layers of V1, except that the length of the V2 bipole kernel, H^{V2}, is greater than that of V1, reflecting the fact that intrinsic horizontal connections have a longer range in V2 than in V1 (Amir, Harel & Malach, 1993), and also that illusory contours can form between more widely spaced inducers in V2 than in V1 (Sheth et al., 1996). The V2 bipole kernels are shown in Fig. 11(b). Parameters for the forward projection from V1 to V2 were: $V_{12}^{6} = 1$, $V_{12}^{4} = 2$.

A.8. Network inputs for the simulations

The simulations of the physiological data from the experiments of Polat et al. (1998), Roelfsema et al. (1998) and Reynolds et al. (1999) all used the same set of network parameters. The full network with V2 as well as V1 was used only for the Polat simulations. The strengths of the raw inputs and attentional Gaussians used for the Roelfsema and Reynolds simulations were as follows: Roelfsema: raw input strength, $I = 0.31$, peak value of attentional Gaussian $= 0.4$. Reynolds: raw input strength, $I = 0.36$, peak value of attentional Gaussian $= 0.17$, Both of these attentional simulations used an attentional Gaussian with standard deviation of 1.5.

A.9. Self-organized kernels

The kernels, which were self-organized in the study by Grossberg and Williamson (1998), are represented here graphically (Figs. 7–10), except for the single-pixel layer 2/3 inhibitory kernels, T^{+} and T^{-}, which had the following self-organized equilibrium values. $T_{11}^{+} = 0.9032$, $T_{21}^{+} = 0.1384$, $T_{12}^{+} = 0.1282$, $T_{22}^{+} = 0.8443$. $T_{11}^{-} = 0.2719$, $T_{21}^{-} = 0.0428$, $T_{11}^{-} = 0.0388$, $T_{22}^{-} = 0.2506$. T^{+} in V2 was 0.625 times the value of T^{+} in V1.

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