A model of non-linear interactions between cortical top-down and horizontal connections explains the attentional gating of collinear facilitation

Kris De Meyer *, Michael W. Spratling

Division of Engineering, King's College London, WC2R 2LS, United Kingdom

Abstract

Past physiological and psychophysical experiments have shown that attention can modulate the effects of contextual information appearing outside the classical receptive field of a cortical neuron. Specifically, it has been suggested that attention, operating via cortical feedback connections, gates the effects of long-range horizontal connections underlying collinear facilitation in cortical area V1. This article proposes a novel mechanism, based on the computations performed within the dendrites of cortical pyramidal cells, that can account for these observations. Furthermore, it is shown that the top-down gating signal into V1 can result from a process of biased competition occurring in extrastriate cortex. A model based on these two assumptions is used to replicate the results of physiological and psychophysical experiments on collinear facilitation and attentional modulation.

Key words: Contrast detection, Contour integration, Lateral interactions, Attention, Perceptual grouping, Cortex, Neural network, Dendrites

* Corresponding author.
Email address: kris@corinet.org (Kris De Meyer).

1 Introduction

Recent years have seen an important shift in the understanding of the early stages of cortical vision. The traditional view held that information, relayed from the retina, is processed by simple local feature detectors in primary visual cortex (V1), followed by increasingly complex information processing in the later stages of a hierarchy of cortical areas (Marr, 1982). However, not only does it appear that cells in early visual cortex respond to more complex stimuli than previously thought (Hegde and Van Essen, 2007), it is also becoming apparent that their response properties are not static, but can be flexibly and dynamically altered by the surrounding context of the stimulus, as well as by task context and attentional state. For instance, in V1 the response of a neuron to a stimulus placed in its "classical" receptive field (RF) can be enhanced or suppressed by stimuli falling outside the RF (Gilbert, 1998; Series, Lorenceau, and Fregnac, 2003; Angelucci and Bressloff, 2006). These contextual effects are commonly referred to as *centre-surround interactions*. Recent studies have shown that these interactions come in many forms: differences in spatial and temporal characteristics of various inhibitory and excitatory effects indicate that they are caused by different neural circuits or mechanisms (Series et al., 2003; Angelucci and Bressloff, 2006).

One particularly well-studied contextual effect is *collinear facilitation*. It refers to the fact that the response of V1 cells to a low-contrast oriented stimulus (such as a bar or Gabor patch) can be enhanced by the presence of high-contrast collinear, coaxial flanking stimuli (Kapadia, Ito, Gilbert, and Westheimer, 1995; Polat, Mizobe, Pettet, Kasamatsu, and Norcia, 1998; Chen, Kasamatsu, Polat, and Norcia, 2001; Mizobe, Polat, Pettet, and Kasamatsu, 2001). The effect is likely to be mediated by long-range horizontal connections in the superficial layers (layers 2 and 3 or L2/3) of V1 (Gilbert, 1998; Series et al., 2003; Angelucci and Bressloff, 2006). Moreover, it is thought to give rise to the psychophysical phenomenon of the same name, i.e., the increase in contrast sensitivity for a low-contrast central target when presented in conjunction with high-contrast collinear flankers (Polat and Sagi, 1993, 1994).

Physiological and psychophysical experiments have shown that collinear facilitation is modulated by task context or attentional state (Ito and Gilbert, 1999; Gilbert, Ito, Kapadia, and Westheimer, 2000). In particular, Gilbert et al. (2000) suggested that attention – through top-down connections from extrastriate cortical areas – *gates* the facilitatory effect of collinear flanking stimuli, i.e., attention effectively switches lateral interactions on and off. In a series of subsequent psychophysical experiments Freeman et al. (Freeman, Sagi, and Driver, 2001; Freeman, Driver, Sagi, and Zhaoping, 2003; Freeman, Sagi, and Driver, 2004; Freeman and Driver, 2005) investigated a number of competing explanations for this effect and settled with some confidence on a two-part

hypothesis: firstly, attention gates the effects of collinear flankers by *modulating flanker-target integration* (Freeman et al., 2003); secondly, attention acts by resolving a *biased competition between different perceptual groupings* of the stimulus configuration (Freeman and Driver, 2005).

Freeman et al. did not speculate on the neural mechanisms giving rise to their psychophysical observations. Similarly, Gilbert and Sigman (2007) note that the precise neural mechanisms that cause the top-down gating of lateral interactions remain unknown. In this paper we present a biologically plausible model that can explain both physiological and psychophysical results. Our model is based on the following critical assumptions: firstly, gating is caused by non-linear dendritic interactions between inputs arriving on different parts of the dendritic tree of cortical pyramidal cells; secondly, the top-down gating signal into V1 originates from a competition between nodes in extrastriate areas V2 and V4. This competition, in turn, may be biased by an attentional feedback signal originating in frontal cortex (Moore and Armstrong, 2003; Armstrong, Fitzgerald, and Moore, 2006).

We construct a model of cortical areas V1, V2 and V4 by extending a model, previously used to simulate a range of attentional effects in cortical areas V2 and V4 (Spratling and Johnson, 2004; Spratling, 2008), to incorporate long-range horizontal connections in area V1. We show that the model succeeds in generating the attentional gating of collinear facilitation reported in (Freeman et al., 2001, 2003, 2004), and we demonstrate how biased competition between nodes in extrastriate areas V2 and V4 may lead to the observed modulation of contextual interactions in V1 (Freeman and Driver, 2005). The model thus provides a unified account of a range of disparate but related visual phenomena, namely, collinear facilitation, perceptual grouping, and the biased-competition theory of attention (Desimone and Duncan, 1995).

The paper is organised as follows: in Sect. 2 we introduce the model and explain how it is grounded in anatomical and physiological constraints. In Sect. 3, we discuss in more detail the neural correlate of collinear facilitation in V1 and attentional effects in cortical areas V1, V2 and V4. We add simulation results to show that the model can successfully replicate empirical data on the level of single-cell and population responses. Sect. 4 contains simulation results replicating the psychophysical data of (Freeman et al., 2001, 2003, 2004; Freeman and Driver, 2005). Finally, in Sect. 5, we discuss testable predictions, potential future experiments, and how the model aids theory formation.

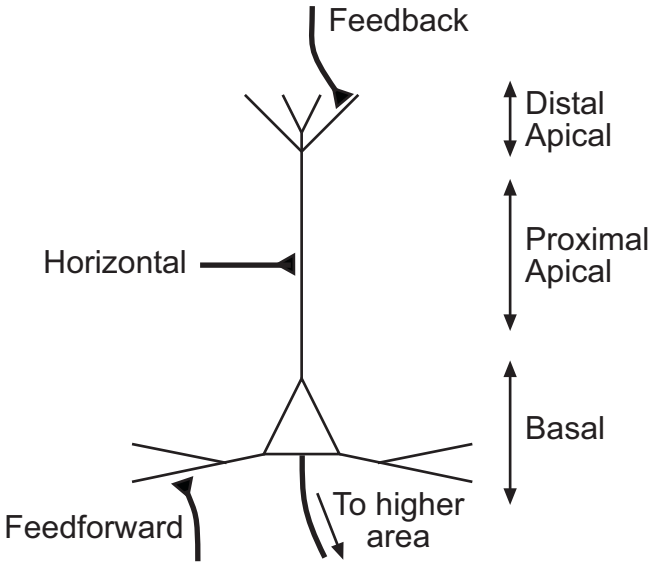

Fig. 1. Schematic of a pyramidal cell in the superficial layers (L2/3) of neocortex. Morphologically L2/3 pyramidal cells are characterised by basal dendrites that extend laterally from the soma, and by an apical dendrite that extends vertically into L1 and ends in a tuft of fine branches. Feedforward stimulation, relayed by spiny stellate cells in L4, targets the basal dendrites, while feedback or top-down connections from areas higher up in the cortical hierarchy target the apical tuft. L2/3 cells predominantly send axonal projections to L4 spiny stellate cells in higher cortical areas and are the main "output" neurons of each area. In V1, collateral branches from these axonal feedforward projections form intrinsic horizontal connections, targeting parts of the apical dendrite more proximal to the soma.

2 Model

2.1 Neuron

Neocortical pyramidal cells generally receive feedforward and feedback connections on different parts of the dendritic tree: they receive feedforward stimulation at the basal dendrites and feedback stimulation at the apical tuft (Fig. 1). Physiological evidence suggests that this anatomical segregation of input sources may have functional significance (Spratling, 2002; Hausser and Mel, 2003; Spruston, 2008). Feedback stimulation arriving at the apical tuft is integrated relatively independently from the feedforward stimulation integrated at the soma. These two integration results are associated through mechanisms involving dendritic action potentials (Yuste, Gutnick, Saar, Delaney, and Tank, 1994; Larkum, Zhu, and Sakmann, 1999). Pyramidal cells contain at least two spike initiation zones: an axosomatic zone giving rise to "conventional" axonal spikes and, simultaneously, to *back-propagating action potentials* (bAP) travelling from the soma into the apical dendrite (Stuart, Sprus-

ton, Sakmann, and Hausser, 1997; Waters, Larkum, Sakmann, and Helmchen, 2003); and a dendritic zone just below the apical tuft giving rise to dendritic spikes propagating forwards to the soma (Larkum et al., 1999; Larkum, Zhu, and Sakmann, 2001; Larkum, Waters, Sakmann, and Helmchen, 2007). Both *in vitro* and *in vivo* experiments have shown that the threshold for dendritic spike initiation is generally quite high, but is lowered significantly by the arrival of a bAP at the apical tuft (Larkum et al., 1999; Waters et al., 2003). Furthermore, Larkum et al. (1999, 2007) observed that when a dendritic spike reaches the soma it can trigger one or several axonal spikes. The combination of these dendritic properties thus suggests how feedback arriving at the apical tuft can modulate a neuron's response to feedforward stimulation arriving at the basal dendrites: supra-threshold stimulation of the axosomatic initiation zone triggers an axonal spike and a bAP travelling into the apical dendrite; if arrival of the bAP at the apical tuft coincides with sufficient local synaptic stimulation from feedback sources it generates a dendritic spike; arrival of this dendritic spike at the soma triggers additional axonal spikes, effectively multiplying the number of spikes generated by the feedforward stimulation (Larkum et al., 1999; Hausser and Mel, 2003; Spruston, 2008).

One of the authors has previously used a model with separate basal and apical compartments to simulate attentional modulation in extrastriate areas V2 and V4 (Spratling and Johnson, 2004; Spratling, 2008). In this model the response of a cell is driven by the feedforward activity generated at the basal compartment, and modulated multiplicatively by attentional top-down input arriving at the apical compartment. In the current paper we extend the previous model by incorporating long-range excitatory horizontal connections in area V1. These connections arise from collateral branches of the main axons of superficial layer (L2/3) pyramidal cells. Axons of V1 L2/3 pyramidal cells form the dominant feedforward projection to extrastriate cortical areas, and these cells are therefore regarded as the "output" neurons of the visual pathway (Felleman and Van Essen, 1991; Kapadia, Westheimer, and Gilbert, 2000). The collateral branches are intrinsic to V1; they connect regions several millimetres apart and reciprocally link cells with similar orientation preferences (Series et al., 2003). Anatomical evidence suggests that these lateral connections target the apical dendrite more proximal to the soma (McGuire, Gilbert, Rivlin, and Wiesel, 1991; Yoshimura, Sato, Imamura, and Watanabe, 2000). The functional role of synaptic contacts on this part of the apical dendrite may be to regulate the *coupling* between the apical tuft and the soma (Larkum et al., 2001). Two mechanisms may be involved: firstly, bAP amplitude decreases with distance from the soma, meaning that bAPs often fail to propagate to distal parts of the apical dendrite (Stuart and Hausser, 2001; Waters et al., 2003). However, even modest synaptic depolarisation along the apical dendrite can strongly *boost* bAPs and significantly increase the proportion of bAPs reaching the apical tuft (Stuart and Hausser, 2001; Waters and Helmchen, 2004). Secondly, the proportion of forward-propagating dendritic

spikes reaching the soma is variable and may, likewise, be a function of the depolarisation of the proximal apical dendrite – as has been demonstrated for L5 (Larkum et al., 2001) and hippocampal pyramidal cells (Jarsky, Roxin, Kath, and Spruston, 2005).

Based on these anatomical and physiological observations, we derive a *rate-based* model of L2/3 pyramidal cells. We focus on L2/3 cells for several reasons: firstly, they are the most prominent cell type in neocortex (Zilles, 1990); secondly, in each of the early visual areas they are the "output" neurons of the dominant feedforward pathway (Felleman and Van Essen, 1991; Kapadia et al., 2000); thirdly, they are the main source of horizontal connections in V1 (Series et al., 2003; Angelucci and Bressloff, 2006). For V1, we propose a model pyramidal cell with 3 functional compartments: the basal compartment receives feedforward input, the distal apical compartment receives feedback input, and the proximal apical compartment receives long-range horizontal input (Fig. 1). Feedforward connections drive the neuron, while feedback and horizontal connections can only modulate the cell response. The relationship between the activation of the dendritic compartments and a neuron's output can be modelled as:

$$O = F \times (1 + \sigma_d(B) \times \sigma_p(H)) \tag{1}$$

Where O stands for the output, F for the feedforward activation, B for the feedback activation, and H for the horizonal activation. $\sigma_d(.)$ and $\sigma_p(.)$ are sigmoid functions modelling a non-linear saturation of distal and proximal apical compartments. They can be understood as follows: horizontal stimulation affects the proportion of bAPs reaching the apical tuft; its influence therefore saturates when *all* bAPs reach their destination. Likewise, feedback depolarisation of the apical tuft is necessary to turn a bAP into a forward-propagating dendritic spike; its influence saturates when *all* bAPs result in a forward-propagating dendritic spike. The mathematical relationship between $\sigma_d(B)$ and $\sigma_p(H)$ is multiplicative because both sets of connections need to stimulate the post-synaptic cell *together*; if either of them is absent or too weak the chain of dendritic events described above is interrupted and modulation of the cell's output does not occur. The product of F and the bracketed term represents the multiplicative modulation of the feedforward, basal stimulation by the combined result of the distal and proximal apical stimulation.

The activation of individual compartments is calculated as a weighted sum of the input strength. However, integration of the feedforward input is affected by local lateral inhibition, modelled as a divisive normalisation operating on the feedforward input. It causes cells with overlapping receptive fields to compete for the right to represent stimuli rather than for the right to generate output, as is common in other models of lateral inhibition. For a discussion of the remit of this form of inhibition we refer to earlier papers (Spratling and Johnson, 2001,

2002, 2004, 2006; Spratling, 2008; Spratling, De Meyer, and Kompass, Sub.), and for further mathematical details of the model we refer to the Appendix.

Mathematically, cells in model areas V2 and V4 differ from V1 cells only in the saturation $\sigma_p(.)$ of Eq. 1: in V2 and V4 the proximal apical compartment *always* saturates for any input strength. V2 and V4 cells in the current model are therefore functionally equivalent to the two-compartment cells used in (Spratling and Johnson, 2004) and (Spratling, 2008). In physiological terms the saturation of the proximal apical compartment signifies that bAPs and dendritic spikes – once initiated – always reach their target. This is in accordance with empirical results from single-cell recordings which show that cell output in areas V2 and V4 is *directly* modulated by top-down input, as opposed to the indirect modulation of centre-surround interactions in V1 (Lamme, Super, and Spekreijse, 1998; Reynolds and Chelazzi, 2004). Such systematic differences between cortical areas could be caused by differences in cell morphology (Larkum et al., 2001), dendritic membrane excitability (Jarsky et al., 2005), or temporal properties of the input (Larkum et al., 2001).

2.2 Network

The physiological and psychophysical experiments modelled in this paper use small, static, oriented patches arranged into larger stimulus configurations. In primates such perceptual stimuli are thought to be processed by the ventral pathway, the visual subsystem that is primarily involved in shape representation (Milner and Goodale, 2006). The ventral pathway forms a hierarchy of distributed cortical areas linked by feedforward and feedback connections (Felleman and Van Essen, 1991). Here we simulate its relevant early stages: in Sects. 4.1, 4.2 and 4.3 we simulate orientation-selective cells in V1 only; in Sect. 3 and 4.4 we also include populations for cortical areas V2 and V4. The former set of experiments allows full control over how feedback targets cells in V1; the latter experiments demonstrate how the top-down signal into V1 can be generated in a biologically plausible way. Overall connectivity between model areas is consistent with cortical anatomy: the feedforward pathway runs from V1 over V2 to V4; feedback projections run from V4 to V2, from V2 to V1, but also directly from V4 to V1 (Felleman and Van Essen, 1991; Salin and Bullier, 1995). Cortical area V4 receives feedback connections from several areas that are presumably involved in the allocation of attention (Felleman and Van Essen, 1991). Model area V4 is therefore a plausible target for *external* attentional feedback. In the experiments where V2 and V4 are not modelled, external feedback arriving from these areas is modelled as direct top-down input to V1. Neurons in all model areas are retinotopically organised, receiving direct or indirect feedforward projections from well-defined parts of the input image. The overall layout of the network can be seen in Fig. 2. All experiments

Table 1
Population sizes per cortical area.

Cortical Area	Neurons per RF	Total RFs	Total Neurons	RF Size
V1	4	25	100	5x5
V2	10	4	40	11x11
V4	6	1	6	17x17

are performed with moderate population sizes: 100 nodes in V1, 40 nodes in V2, and 6 nodes in V4 (Table 1). Input to the network consists of 17x17 pixel images containing combinations of short bar segments. Single bars fall entirely inside one 5x5 pixel V1 RF, and combinations of two adjacent bars falls entirely inside one 11x11 V2 RF. The RF size of neurons in V4 covers the entire input image.

2.3 Representation

In order to avoid setting synaptic weights by hand, network training procedures developed in earlier work (Spratling and Johnson, 2002, 2006; Spratling et al., Sub.) are used to obtain all synaptic connection strengths (with the exception of *external* feedback connections – see below). The training procedure consists of repeatedly presenting input images to the network and updating synaptic weight values using unsupervised learning rules based on pre- and postsynaptic cell activity (please refer to the Appendix for the mathematical formulation). Learning occurs in three distinct stages: first all feedforward weights for area V1 are learned using images consisting of single bars at different orientations (Fig. 3(a)). Subsequently, feedforward weights from area V1 to area V2, horizontal weights in area V1, and feedback weights from V2 to V1 are learned using images consisting of conjunctions of two bars (Fig. 3(b)). Finally, feedforward weights from area V2 to V4, and feedback weights from area V4 to V1 and V2 are learned using contours consisting of 3 bars (Fig. 3(c)). The representation or "preferred stimulus" for each cell is determined by its feedforward weights, which, in turn, is determined by the choice of the training set. Nodes in V1 thus learn to represent single bars, nodes in V2 represent conjunctions of two bars, and nodes in V4 represent conjunctions of three bars. These representations are consistent with known response selectivities of cells in primate visual cortex: short oriented stimuli in V1 (Hubel and Wiesel, 1968), angles between line segments in V2 (Ito and Komatsu, 2004), and longer contour segments in V4 (Hegde and Van Essen, 2007). Feedback connections are directly or indirectly reciprocal to feedforward connections: e.g., a neuron in model V4 sends feedback to all nodes in V2 from which it receives a direct feedforward connection; it also directly targets all neurons in

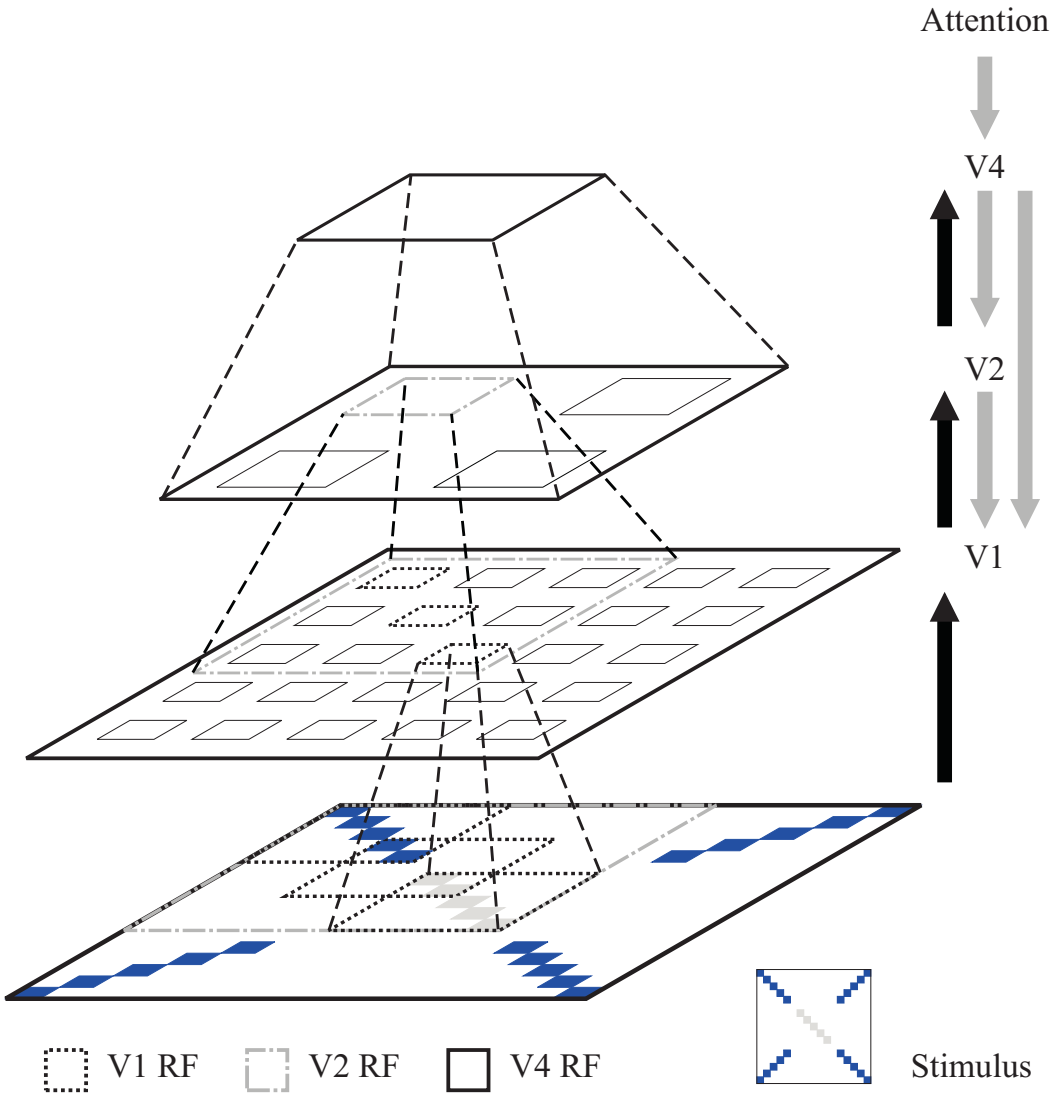

Fig. 2. Network structure and receptive fields (RFs). In all experiments input to the network consists of 17x17 pixel images. Squares in V1, V2 and V4 denote cell populations; individual neurons are not shown. For population sizes in all areas see Table 1. All neurons within a square have fully overlapping RFs; neurons in neighbouring squares have partially overlapping RFs (as shown for three V1 RFs). In V1, neurons receive input directly from 5x5 image patches. In V2, neurons receive projections from a subset of all cells in V1, giving rise to RFs of 11x11 pixels. Neurons in V4 receive projections from the whole of V2, which means that their RFs cover the entire input image. Feedback projections are reciprocal to feedforward projections, i.e., areas in V2 and V4 project to all areas in V1 and V2 from which they receive feedforward input. Attention, arriving from cortical regions not modelled here, targets neurons in V4 via feedback connections. In experiments where V2 and V4 are not modelled explicitly, external feedback targets V1 neurons directly.

V1 from which it receives indirect feedforward stimulation through neurons in V2. *External* feedback is one-to-one, i.e., each node from the uppermost area in the simulated hierarchy (either V1 or V4, dependent on the experiment) receives top-down input from a specific source. Horizontal connections in model area V1 reciprocally link pairs of neurons if their representations co-occur in longer contours in the training set (in this case the training set with two-bar stimuli used during the second training stage – see Fig. 3(b)). However, they can only link neurons at most 2 V1 RFs away. Figure 4 shows a representative sample of horizontal connections. The constraints on horizontal connections in the model are consistent with general principles of horizontal connectivity in V1: they tend to link cells with similar response properties and typically avoid connecting cells with orthogonal response properties. Moreover, horizontal fibres predominantly run anisotropically along the axis of preferred orientation of the pre-synaptic neuron, and are limited to distances of a few cortical hypercolumns (Series et al., 2003; Angelucci and Bressloff, 2006).

3 Neural correlates of collinear facilitation and attention

Several physiological studies have investigated the effects of collinear flankers on the response properties of pyramidal cells in primary visual cortex (Kapadia et al., 1995; Ito and Gilbert, 1999; Polat et al., 1998; Chen et al., 2001; Mizobe et al., 2001). In this section we review these results, as well as physiological results on the attentional modulation of collinear facilitation in V1 (Ito and Gilbert, 1999) and notable differences with attentional effects in extrastriate areas V2 and V4 (Reynolds, Chelazzi, and Desimone, 1999; Reynolds and Chelazzi, 2004). These empirical data, obtained from pyramidal cells in response to perceptual stimuli in awake or anesthetised animals, link the anatomical and physiological observations from the previous section to the human psychophysical data in the next section. In order to show that these physiological data themselves can be explained by the critical assumptions that form the basis of our model, we include simulation results replicating various single-cell and population responses. All simulation experiments in this section have been performed with the full network consisting of areas V1, V2 and V4 (see Fig. 2). The perceptual stimuli consist of 17x17 pixel images as described in Sect. 2.3. The exact stimulus configuration and whether or not external attentional feedback has been applied to cells in V4 is explained below for each experiment. The same parameter values have been used throughout all simulations in this and the next section; the choice of these values is discussed in the Appendix.

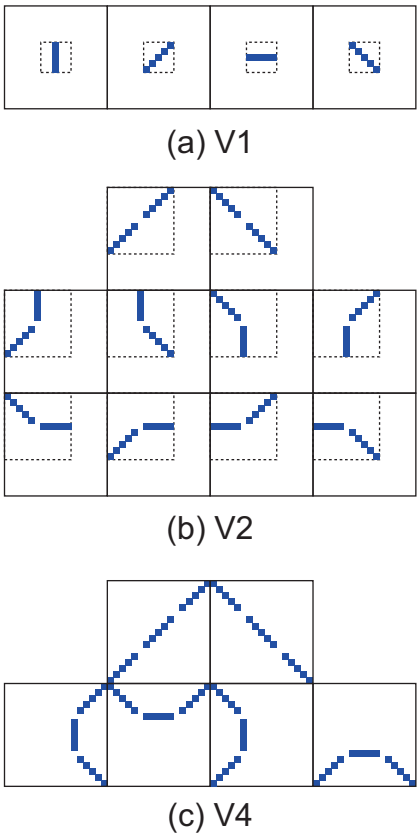

Fig. 3. Neural representations. A neuron's representation is determined by its feedforward weights, but the images shown here are not of the weights, but of the "preferred stimulus" of each cell. (a) Neurons in V1 represent short oriented bars located at 1 of the 25 V1 RF centres in the 17x17 input field. Shown here are the representations of the four neurons with an RF in the centre of the input image (indicated by the dotted square). Feedforward weights for a single node are always normalised such that their total sums to 1. For V1, the value of the weights is thus a fraction of the pixel strengths of the images they represent. (b) Each V2 neuron is linked by feedforward weights to a pair of V1 neurons, thereby representing a conjunction of two bars. Depicted here are the representations of the ten neurons within the V2 RF shown in Fig. 2. (c) Each of the six V4 neurons is connected to a pair of V2 neurons, resulting in longer contour representations of three bars.

3.1 Excitation dominates flanker effects at low target contrast

The response of an orientation-selective pyramidal cell to its preferred stimulus can be enhanced, inhibited or remain unaffected by the addition of high-contrast flankers placed outside the RF, along the main axis of the preferred stimulus orientation (i.e., *coaxial* with the central stimulus). Such modulatory effects are strongest when the flankers are *collinear* with the target, and virtually absent for flankers *orthogonal* to the target (Kapadia et al., 1995; Mizobe et al., 2001). The effect also appears to be strongly dependent on target con-

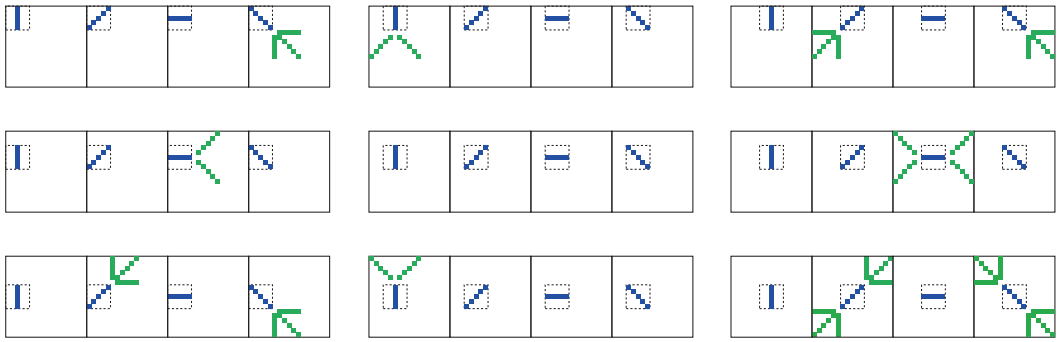

Fig. 4. Horizontal connections in V1. Each block shows the representations of 4 V1 neurons with the same RF centre (indicated by the dotted square) in dark/blue, and representations of the cells they are laterally connected to in light/green. Only the cells whose preferred stimuli have co-occurred in the training set are linked through horizontal connections. Depicted here are all V1 RFs falling inside the V2 RF of Fig. 2. The block in the bottom right corner of the figure depicts the central V1 RF of Fig. 2. Horizontal connections in these 9 RFs are representative of the ones not shown.

Table 2
Summary of physiological data on the contrast-dependence of modulatory effects of collinear flankers, showing the percentages of orientation-selective cells that experience excitation (E), inhibition (I) or are unaffected (U) by flanking stimuli.

Source	Target contrast level	% E	% I	% U
(Kapadia et al., 1995)	supra-threshold	42%	26%	32%
(Ito and Gilbert, 1999)	supra-threshold	37%	30%	33%
(Chen et al., 2001)	near-threshold	67%	18%	15%
(Chen et al., 2001)	high	38%	47%	15%

trast. Table 2 summarises the available data on the contrast-dependence of collinear facilitation for bar stimuli (Kapadia et al., 1995; Ito and Gilbert, 1999) and Gabor stimuli (Chen et al., 2001). At low target contrast levels (i.e., contrast levels near the threshold needed to evoke a response in the cell), facilitation dominates, with a shift towards more inhibition as target contrast increases. These results are independent of pyramidal cell type (simple vs. complex) or cortical layer (L2/3 vs. L5) (Chen et al., 2001; Mizobe et al., 2001). The latter may be explained by the fact that, although L2/3 pyramidal cells are the main source of horizontal connections, their postsynaptic targets may be L2/3 as well as L5 pyramidal cells (McGuire et al., 1991).

The relevance of these contrast-dependence physiological effects for the psychophysical contrast-detection experiments considered in Sect. 4 lies in the fact that high-contrast flankers have a predominantly facilitatory effect on neural response properties for the stimuli (Gabor patches) and target contrast levels

used in those experiments. Because in this paper we are primarily interested in explaining these psychophysical results, we have included only long-range horizontal excitation into our model (i.e., the excitatory dendritic interactions described in Sect. 2.1). Consequentially, we will focus on the excitation caused by collinear flankers in the physiological experiments we seek to replicate in this section. We will briefly discuss in Sect. 5 how the inhibitory component of long-range lateral interactions can be included in future work.

Figure 5(a)-(b) shows two examples of how the addition of collinear flankers enhances the time-averaged response of pyramidal cells in L2/3 of V1 (Kapadia et al., 1995). For the cell in Fig. 5(a), addition of the first flanker has a large facilitatory effect, while a second flanker has a much smaller additional effect. In contrast, in Fig. 5(b) it is the second flanker that has the strongest facilitatory effect. Simulation results in Fig. 5(c) and (d) show a similar dependence on the addition of one or two flankers as for Fig. 5(a) and (b) respectively. Note that, whereas (a) and (b) were recorded from two different cortical cells with different contrast sensitivity profiles, (c) and (d) result from the same model cell at different points along its contrast sensitivity function. Flankers presented without a central target have no influence on spiking response, indicating the *modulatory* rather than *driving* effect of these stimuli. The physiological experiments in (Kapadia et al., 1995) did not manipulate attentional state, and, likewise, in the simulation experiments there is no external attention targeting cells in model area V4. The top-down signal into V1 that enables the collinear facilitation is generated by bottom-up activation of nodes in V2 and V4 feeding back into V1.

Figure 6(a) depicts how flankers modulate neural spike rate over time (Ito and Gilbert, 1999). The figure shows the population response averaged over all cells that experienced facilitation when a flanker was added to the target stimulus (37% of all cells recorded). Important to note is that the facilitation is present from the onset of the neural response and occurs for the entire duration of the response. Figure 6(b) demonstrates how the model cell representing the central target simulates this population effect. The dynamical response shown in this figure is representative for the response of model cells in further experiments. Moreover, the collinear facilitation follows a time-course similar to the physiological experiments. The experiments in (Ito and Gilbert, 1999) manipulate attentional state, but Fig. 6(a) shows facilitation regardless of attentional condition. Accordingly, the simulation experiments are not generated using external attention into area V4; again, the top-down signal into V1 is entirely generated by bottom-up activation of nodes in V2 and V4.

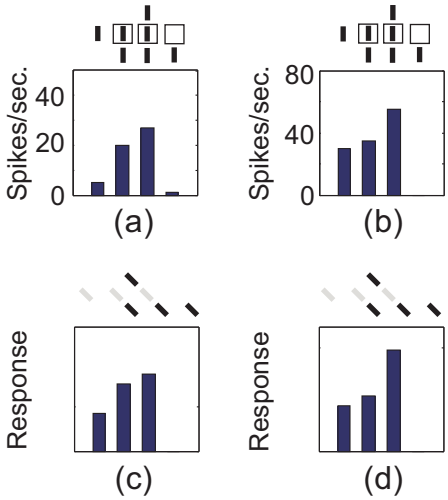

Fig. 5. Facilitatory effects of collinear flankers on single cell response in V1. The stimulus conditions are shown at the top of each figure. (a)-(b) Physiological data adapted from (Kapadia et al., 1995, Fig. 12(B)-(C)). Response recorded from two pyramidal cells in superficial layers of V1. Target contrast levels in (Kapadia et al., 1995) were between 10% and 22%, and the flanker contrast was always 62%. The exact target contrast levels for these two recordings, however, were not reported. (c)-(d) Average simulated response recorded from the model cell in V1 that represents the central target shown in light grey in the stimulus conditions. In (c) the input strength of the central target was 0.15, and in (d) the input strength was 0.10. The input strength of the flankers was 0.50 in both cases. The response values were obtained by averaging the cell's output over a simulation of 50 computational steps of the network's equations (a single "iteration" – see Appendix), and scaled to be comparable to the physiological results. No external feedback was applied to nodes in V4.

3.2 Attention modulates collinear facilitation

Ito and Gilbert (1999) demonstrated that attention modulates the collinear facilitation of neural responses in V1. In particular, they showed that a change in attentional state can effectively switch the collinear facilitation *on* or *off*. In their experiments they distinguished between three different attentional conditions: focal attention *on* the central RF of the recorded cell, attention *away* from the recorded cell (but focussed on another part of the stimulus configuration), and attention *distributed* over the recorded and 3 other locations in the stimulus configuration. Figures 7(a) and (c) shows neural response properties averaged over the recorded population for one of their primate subjects. [1]

[1] Results for a second primate subject were different from the ones shown here. These differences were attributed by Ito and Gilbert (1999) to differences in training procedure and problem solving strategies employed by the two subjects. However, they have recently been explained in terms of a difference in spatial integration mechanisms in foveal and peripheral vision (Roberts, Delicato, Herrero, Gieselmann,

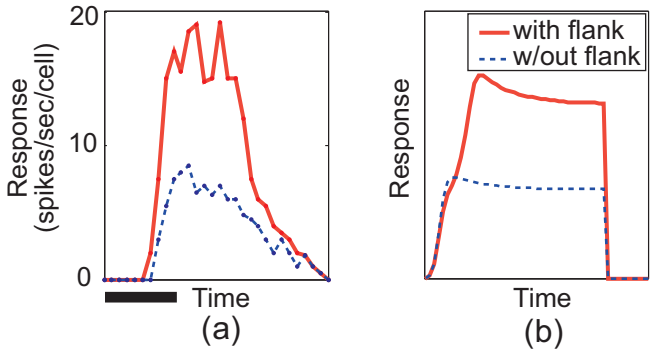

Fig. 6. Collinear facilitation of cell response over time. (a) Average population response over time for all cells in V1 that demonstrated significant flanker facilitation under any attentional condition, adapted from (Ito and Gilbert, 1999, Fig. 2(C)). The bar at the bottom of the graph indicates the duration of the stimulus presentation. The target in these experiments is presented at supra-threshold contrast levels, higher than for Fig. 5. (b) Simulated response over time of the V1 cell representing the central target (same as in Fig. 5 (c)-(d)) without flankers and with a single flanker. The network is simulated for 50 computational steps, with the stimulus presented to the network at time 0 and removed at time 40. Input strength of the central target is 0.25, and of the flanker 0.50. The response is in arbitrary units and has been scaled to resemble (a). No external feedback was applied to nodes in V4.

Figure 7(a) demonstrates that, in the *absence* of a flanking stimulus, attention has little or no effect on the response properties of V1 pyramidal cells. In the presence of a flanking stimulus, however, there is a marked difference in the amount of collinear facilitation for the different attentional conditions (Fig. 7(c)): flanker facilitation is much stronger for focal attention *on* the target in comparison with the other two attentional conditions. Figures 7 (b) and (d) show the simulation results for the V1 model cell responding to the same low-contrast central target as in previous figures without (b) and with (d) a flanker. In this experiment, external attention targeted the distal apical dendrite of the cell in V4 representing the long, straight contour overlaying the central target (the second node in Fig. 3(c)). The three attentional conditions in this simulation are modelled as *no* external attention, a *weak* (i.e., 0.1) external attentional signal, and *strong* (1.0) external attention. In the case of no attention or weak attention only a weak top-down signal is generated. A strong external attentional signal generates a stronger cell response in model area V4, and hence a strong top-down signal into model areas V1 and V2 which, in turn, leads to a much enhanced facilitatory effect of the collinear flankers.

and Thiele, 2007).

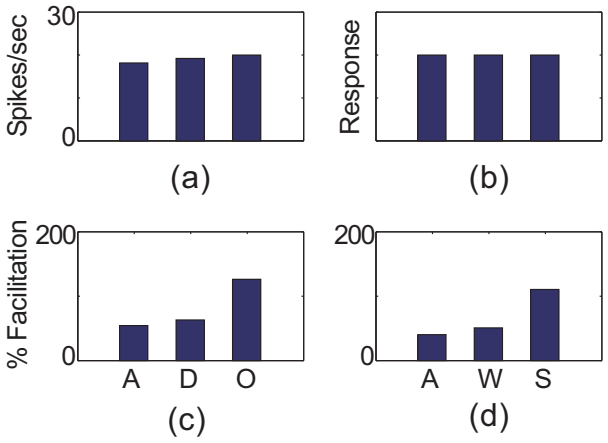

Fig. 7. Attention modulates collinear facilitation. (a) Physiological results; population response of cortical pyramidal cells in V1 to a central target stimulus without flankers, for 3 different attentional conditions: away (A), distributed (D) or focal on the target (O). (b) Simulation results for the model cell in V1 representing the central target, without flankers and for 3 different external attentional strengths targeting a single cell in V4: away (A; 0.0), weak (W; 0.1) and strong (S; 1.0). Target input strength was 0.125, and flanker input strength 0.50. (c) Physiological results; % facilitation of population response in the presence of one collinear flanker, for 3 different attentional conditions. (d) Simulation results; % facilitation of the cell response representing the central target, in the presence of a single flanking bar and for 3 different attentional conditions. (a) and (c) are adapted from (Ito and Gilbert, 1999, Fig. 7(B) and (D)).

3.3 Attention modulates cell response directly in extrastriate cortex

In contrast with the absence of attentional modulation of isolated stimuli in V1, as seen in Fig. 7(a), attention in extrastriate areas can directly modulate a cell's response to isolated stimuli or to combinations of stimuli falling entirely inside its RF (Desimone and Duncan, 1995; Desimone, 1998; Reynolds et al., 1999; Reynolds and Chelazzi, 2004). For example, Fig. 8(a) shows the effect of attention on a pyramidal cell in V2 (Reynolds et al., 1999): a poor stimulus, failing to elicit a significant response from the cell when presented in isolation, nevertheless has a suppressive effect when presented together with the cell's preferred stimulus. However, when attention is directed to the cell's preferred stimulus, the response to the pair of stimuli is restored to the response of the cell to its preferred stimulus alone. In other words, attention filters out the suppressive effect of the poor stimulus (Reynolds et al., 1999). In this particular example, attention is thought to bias an ongoing competition between different cells with overlapping RFs (Desimone and Duncan, 1995).

This example of the biased competition theory of attention has previously been replicated with neural models that are functionally equivalent to areas V2 and V4 of the current model (Spratling and Johnson, 2004; Spratling,

2008). To emphasise that the current model is a generalisation of previous work, we demonstrate that it also replicates the above attentional effect in area V2. The simulation results and the different stimulus conditions can be seen in Fig. 8(b). The response is recorded from the V2 node representing the conjunction of the two collinear diagonal bars in the upper left corner of the input image (the second node in Fig. 3(b)). The distracter is a short vertical bar. By itself, this distracter generates no response from the recorded cell. It exerts its suppressive influence by partly activating other nodes in V2 which compete with the recorded node in order to represent the stimulus. In the three 'attend away' conditions, no external attention is applied to the network. In the 'pair attend pref' condition, a strong (i.e., 1.0) external attentional signal targets the V4 node representing the diagonal overlaying the preferred stimulus of the V2 cell (i.e., the attentional condition is the same as the *strong* condition from Fig. 7(b) and (d)). The external attention enhances the response of the node in V4, which, as a result, sends a stronger top-down signal to the recorded node in area V2. This selective top-down signal biases the competition between the V2 nodes in favour of the preferred stimulus and filters out the suppressive effect of the distracter.

4 Attentional gating of collinear facilitation: psychophysics

In Sect. 3 we demonstrated that the model successfully replicates excitatory effects of collinear flankers and attentional modulation at the level of single cells and neural populations. In this section we go on to demonstrate that the same model can also replicate the results of a series of psychophysical experiments conducted by Freeman and coworkers to investigate the attentional gating of collinear facilitation and the nature of the attentional signal itself (Freeman et al., 2001, 2003, 2004; Freeman and Driver, 2005).

Simulations in the previous section were all performed with the full network model consisting of areas V1, V2 and V4. The top-down signal into V1 was generated inside areas V2 and V4 in response to bottom-up activation from V1. External attention was either absent, or targeted a single cell in area V4. In contrast, in Sects. 4.1, 4.2 and 4.3 we only simulate area V1 in order to have full control over how feedback targets cells in V1, and to establish necessary and sufficient characteristics for the top-down gating signal. In Sect. 4.4 we then again add model areas V2 and V4 to demonstrate that the appropriate feedback signal is, in fact, generated by the biased competition of attention occurring in those areas. The stimuli used in these experiments are adapted from the original psychophysical experiments into the 17x17 pixel images that serve as input to the network (see Fig. 9).To obtain contrast detection thresholds – as reported by Freeman et al. (2001, 2004) – from model contrast response functions we used the procedure explained in Fig. 10.

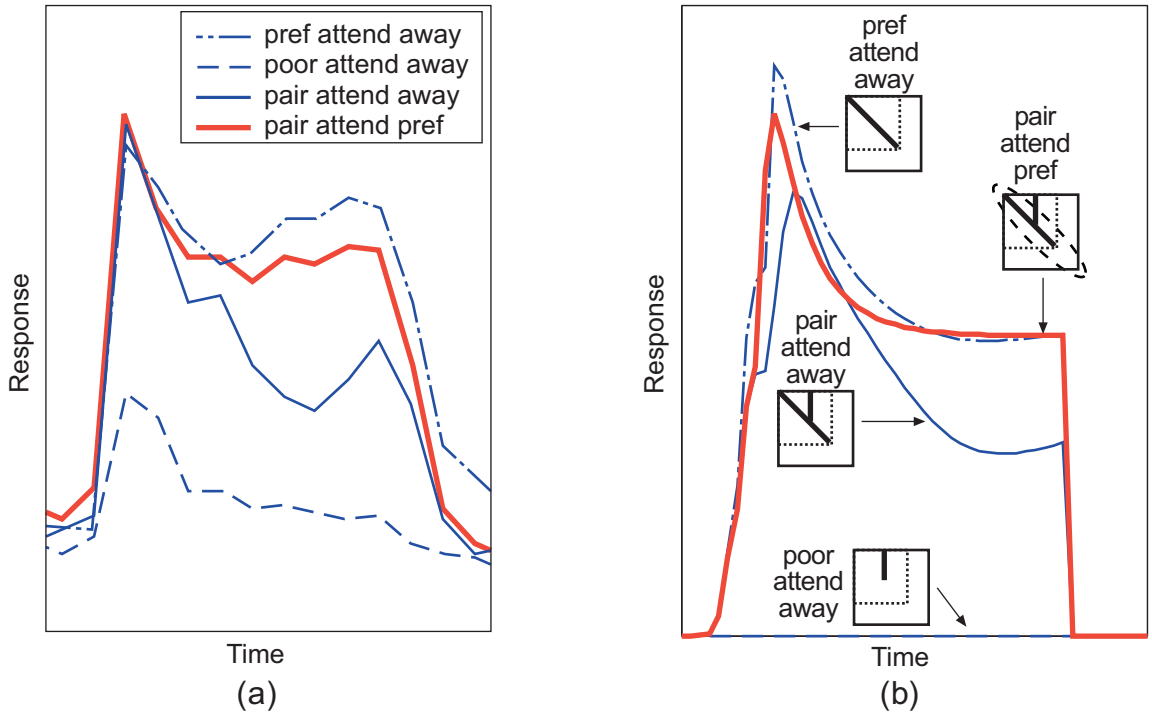

Fig. 8. The effect of attention on a node in V2. Responses are shown for 4 different combinations of stimuli falling inside the cell's RF. (a) Single cell response over time, recorded from cortical area V2. The stimuli were bars shown at high contrast (99%), and similar to the configurations shown in figure (b). Adapted from (Reynolds et al., 1999, Fig. 6(A) and (B)).(b) Simulation results; response over time of the node in model V2 with the RF indicated by the dotted square inside the input images, and with as preferred stimulus the diagonal bar. Input strength for all stimuli was 0.75. In the 'pair attend pref' condition, attention targets a single cell in V4, as explained in the main text. For each curve, the simulation runs for 50 computational steps; the input is presented at step 0 and removed at step 40. The response is in arbitrary units and has been scaled to resemble (a).

4.1 Stimulus and attentional effects

Studies investigating contextual influences on contrast sensitivity commonly use stimuli as depicted in Fig. 9(a): a central target is flanked by stimuli that are either orthogonal to or collinear with the target. Experiments are generally conducted using a two-alternative, forced-choice paradigm: a stimulus is briefly presented twice, once with and once without the central target; observers then have to indicate in which of the two stimulus presentations the target was present. For intermediate target-flanker separations, detection rates for a target at near-threshold contrast levels are generally higher when the target has collinear flankers than when it has orthogonal flankers or no flankers at all (Polat and Sagi, 1993, 1994; Polat, 1999). Attentional state is not manipulated and thought to remain constant for the central target, while the flankers are assumed to be unattended. However, it is likely that flankers

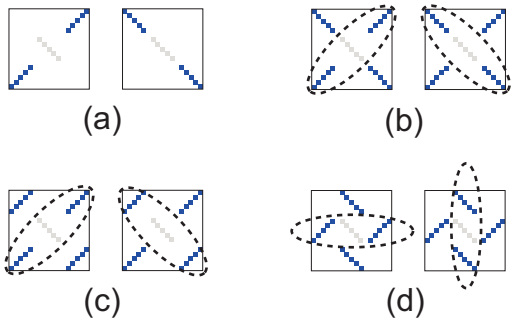

Fig. 9. Experimental stimuli – adapted from (Freeman et al., 2001, 2003, 2004) – consisted of a central target at low contrast and one or two sets of high-contrast flankers. Each individual bar falls entirely inside a single 5x5 V1 RF, and a combination of two adjacent bars falls entirely inside a single 11x11 RF in V2. The RF of V4 nodes covers the entire 17x17 input image. (a) Single-axis stimuli with flankers that are orthogonal to (left) or collinear with (right) the target. (b) Dual-axis stimuli with attention directed towards the orthogonal (left) and collinear (right) flankers. (c) Local-rotation conditions: flankers on one axis have been rotated 90°. Target orientation is orthogonal (left) or similar (right) to the global axis of attention. (d) Global-rotation conditions: both axes have been rotated by 45° but individual flanker orientation has not been changed. The orientation of attended flankers is orthogonal (left) or similar (right) to the target orientation.

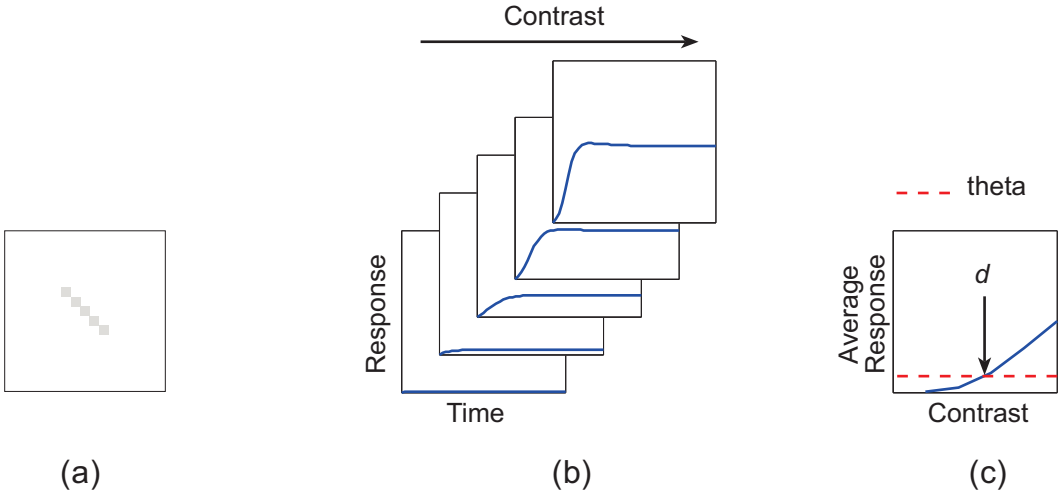

Fig. 10. Determining contrast detection thresholds. In each experiment, the network is simulated using multiple input strengths ("contrast" levels) for the central target. (a) In this example the input consists of the target alone at contrast levels between 0 and 0.1. (b) Response over time of the V1 neuron representing the target. Each sub-plot shows the response generated for a different contrast. (c) The contrast response function is constructed by averaging the neuron's temporal response for each of the simulated contrast levels. Values in between simulated contrast levels are obtained through linear interpolation. Contrast detection threshold d is calculated as the contrast level where the averaged response of the neuron exceeds response threshold θ. In all these experiments response threshold $\theta = 0.01$.

are, in fact, attended as they are typically the most salient items in a sparse display (Freeman et al., 2001).

To investigate the role of attention in this process Freeman et al. (2001) adapted the single-axis experiments into a stimulus configuration that permits the manipulation of attention to the flankers without diverting attention from the target location. In this important respect, the experiments differ from the attentional manipulation in (Ito and Gilbert, 1999), where attention was either directed to or away from the target stimulus, or distributed over multiple targets at different locations in the field of vision. Freeman et al. (2001) placed four flankers in an X-shaped formation around the central target; this results in a stimulus configuration where the target is always surrounded by one orthogonal and one collinear flanker pair (Fig. 9(b)). In addition to the primary target detection task, a secondary task was used to manipulate the allocation of attention. It consisted of a Vernier task – a judgement of misalignment – imposed on either the orthogonal or the collinear flanker pair. Freeman et al. (2001) found that, when attention was directed to the collinear flankers, contrast detection thresholds were lower than when attention was directed to the orthogonal flankers (Fig. 11(a)). Furthermore, they found that this *attentional effect* is almost identical in magnitude to the original *stimulus effect*, i.e., the reduction in contrast detection thresholds in the case of single-axis stimuli. In other words, withdrawing attention from the collinear flanker pair has the same effect on detection thresholds as removing the flanker pair altogether.

We simulated the network consisting of area V1 with the patterns of Figs. 9(a) and (b) to obtain simulation results for the stimulus and attentional effects. The flanker input strength was set to 0.5 and the target input strength was varied between 0 and 0.1 in steps of 0.01. Contrast detection thresholds were obtained from the target neuron's response as described in Fig. 10, and are shown in Fig. 11(b). The psychophysical experiments used a secondary task to manipulate the allocation of attention to different parts of the input image. We did not simulate the secondary task, but rather allocated attentional bias directly through external feedback connections that target the distal apical compartment of neurons in V1. External feedback strength was set to 1.0. The single-cell model, as explained in Sect. 2.1, stipulates that top-down and lateral input need to activate the cell *together* for response modulation to occur. In this experiment, where we are interested in replicating changes in the contrast detection threshold for the central target, we therefore need to look only at how top-down input is directed to cells in the central RF. At first sight this appears to contradict the conclusion of Freeman et al. (2001) that what counts is attention to the *flankers*. How these two seemingly conflicting ideas can be reconciled is explored in Sect. 4.4. There are several plausible combinations of how attention can affect cells in the central V1 RF, but most will produce simulation results that are incompatible with the psychophysical

observations of the dual-axis experiment. For instance, top-down attentional input could target *all* of the four nodes in the central RF indiscriminately (i.e., attention is *spatial*). However, given that the dual-axis stimulus always has one flanker pair that is collinear with the target, both lateral and top-down input would stimulate the cell representing the central target and there would be no attentional effect in the simulated data. This would make the model inconsistent with the psychophysical data, and attention can therefore not be spatial. Top-down attentional input could be directed to a single central node with a specific orientation (i.e., it could be *featural*). This cannot by default be the node that represents the target itself as this means again that facilitation would always occur as the target always has a collinear flanker pair. However, the model does reproduce the physiological attentional effect when attention is featural, and directed at the central node with the same orientation as the global axis of the flanker pair attended for the secondary task.

In the current simulation experiment the model does not allow to make a distinction between the strictly necessary condition of feature-based attention targeting the central RF only, and allocation of attention over a larger part of the visual field. For instance, the model would give the same results if attention were feature-specific over the entire field of vision (i.e., all nodes in V1 with the same orientation as the attended flanker pair receive feedback). This can be understood as follows: even if strong horizontal input, arriving from the cells representing the high-contrast flankers, were further enhanced by the combination of horizontal and top-down input, then this increase in signal strength would not have an extra facilitatory effect on the central target cell because of the non-linear saturation term $\sigma_p(.)$ in Eq. 1. Moreover, the reverse collinear influence from the target on the cells representing the flankers is weak because the target is presented at low contrast levels. This results in weak overall modulation of flanker response. We will return to this issue of localised vs. field-wide attention in Sects. 4.3 and 4.4.

4.2 Flanker contrast dependence

Prior psychophysical studies have indicated that contrast sensitivity in case of single-axis stimuli is not systematically affected by the contrast level of flankers once they exceed detection threshold levels (Zenger and Sagi, 1996; Polat, 1999; Solomon, Watson, and Morgan, 1999; Solomon and Morgan, 2000; Woods, Nugent, and Peli, 2002). In light of these results Freeman et al. (2003) proposed that manipulating flanker contrast in the dual-axis paradigm would allow to distinguish between two alternative explanations for the attentional effect. The first potential explanation states that attention modulates "effective" flanker contrast by enhancing the response to the attended flankers and/or suppressing the response to the ignored flankers (the *flanker-*

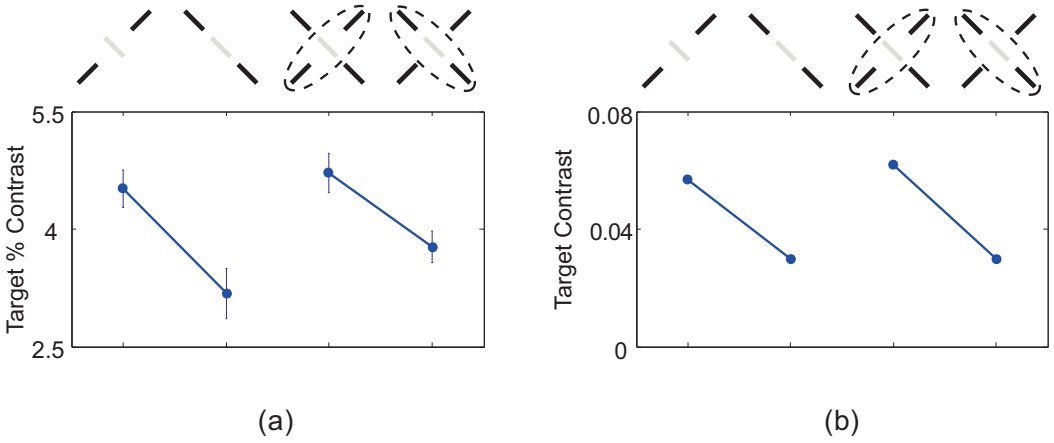

Fig. 11. Contrast detection thresholds for single and dual axis stimuli. (a) Mean thresholds for 7 human observers, adapted from (Freeman et al., 2001, Table 1). (b) Simulation results. The magnitude of the facilitatory effects depends on the choice of parameter values, but overall trends in the data are relatively insensitive to parameter choice (see Appendix).

modulation-only hypothesis). This hypothesis predicts that the attentional effect should reduce for increasing flanker contrast because (1) if the target is collinear with the *attended* flankers, the collinear facilitation saturates exactly like the saturation observed in the contrast sensitivity curves of single-axis stimuli; (2) if the target is collinear with the *ignored* flankers, no such saturation is expected because the attentional suppression much more *gradually* fails to offset the modulatory effects of increasing flanker contrast (see Fig. 12(a)). The second hypothesis states that attention influences target-flanker integration directly by modulating how strongly the flanker signal affects the cells representing the central target. This *connection-weighting* hypothesis predicts that the attentional effect should remain constant for increasing flanker contrast (see Fig. 12(b)). Results from psychophysical experiments reported in (Freeman et al., 2003) are shown in Fig. 12(c). They clearly follow the prediction of the connection-weighting hypothesis, making it unlikely that attention acts by directly modulating the response to the flankers.

Simulation results for the model consisting of area V1 are shown in Fig. 12(d). Input to the network consists of dual-axis stimuli with target contrast fixed at 0.05 and flanker contrast varied between 0.16 and 0.8 in steps of 0.16. Attention is feature-specific (as explained for the previous experiment) and has a strength of 1.0. Target sensitivity is taken as the average response over time of the neuron representing the target. The results are consistent with the predictions of the connection-weighting hypothesis. The saturation observed in the model data is caused by the saturation of the proximal apical compartment, as described at the end of previous section: for a flanker contrast level of 0.16 the horizontal input does not fully saturate the proximal compartment, while for a contrast level of 0.32 it does; further increase in flanker-contrast has little

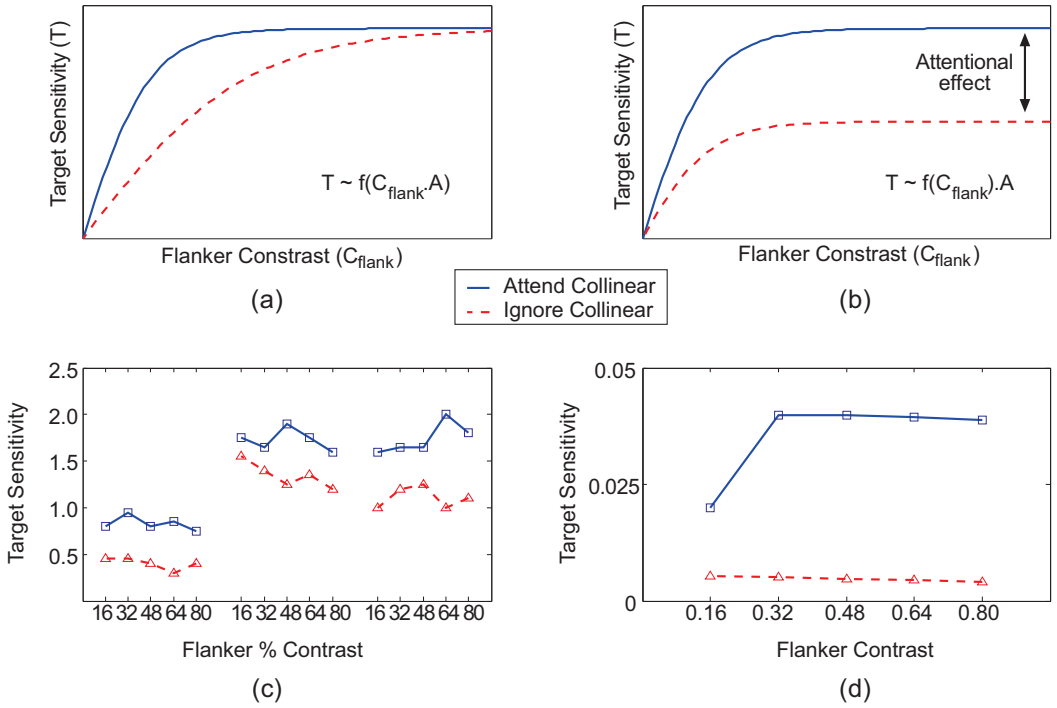

Fig. 12. Target contrast sensitivity in function of flanker contrast. (a)-(c) are adapted from (Freeman et al., 2003, Figs. 2 and 4). The saturating shape of $f()$ – the function relating flanker contrast to target contrast sensitivity – was motivated by experiments with single-axis stimuli (see start of Sect. 4.2). (a) Prediction of the flanker–modulation-only hypothesis. Attentional factor A modulates flanker contrast C_{flank} before the compressive operation of $f()$. (b) Prediction of the connection-weighting hypothesis. Attentional factor A modulates $f()$ directly. (c) Target contrast sensitivity for supra-threshold flanker contrast – for three human observers. Target sensitivity scale, d', is a measure of target sensitivity for two-alternative forced–choice experiments (Macmillan and Creelman, 2005). (d) Model simulation results. Target sensitivity is the time-averaged response of the target neuron.

influence on the target contrast sensitivity.

4.3 Local- and global-rotation conditions

The 'attend collinear' condition for the original dual-axis stimuli implies two types of orientation similarity: firstly, the target has the same orientation as the *global* virtual contour linking the flankers relevant to the secondary task; secondly, the target has the same *local* orientation as the attended flankers. Manipulating the attention from the 'attend collinear' to the 'attend orthogonal' condition disrupts both the global and local orientation similarity. To test if either global or local similarity by itself could be responsible for the attentional effect Freeman et al. (2004) repeated the contrast detection experiments with a novel set of stimuli. One set of stimuli (Fig. 9(c)) was obtained

by *local* 90° rotation of one pair of flankers, removing collinearity with the target but maintaining global orientation similarity between the target and the attended axis for one of the secondary-task conditions. Any account that favours the idea that attention directly modulates the response of the cells whose orientation preference coincides with the globally-attended orientation axis – rather than modulating the collinear target-flanker integration – would predict significant facilitation for the 'attend similar' vs. the 'attend orthogonal' condition (see Fig. 9(c) for clarification of these terms). The second set of stimuli was obtained by rotating the two global axes of the original dual-axis stimuli over 45° but by keeping the local orientation of the individual elements fixed (see Fig. 9(d)). This operation maintains local orientation similarity between target and attended flankers for one of the secondary-task conditions but disrupts the orientation similarity between the target and the globally-attended axis. For such a stimulus configuration facilitation would be expected if attention is featural over the field of vision, i.e., if attention to flankers with a specific orientation modulates the response of all neighbouring cells with similar orientation preferences (Saenz, Buracas, and Boynton, 2002; Treue and Martinez Trujillo, 1999).

The results shown Fig. 13(a) clearly demonstrate that either local or global orientation similarity alone are not sufficient to facilitate the central target detection task. Simulation results for the model consisting of area V1 are shown in Fig. 13(b). Flanker strength is set to 0.5, and the contrast detection thresholds are obtained as in Fig. 11. For the original dual-axis stimuli feedback was allocated in the same way as in Sect. 4.1. For all of the rotated stimuli, collinearity between target and flankers is entirely disrupted. Under these stimulus conditions, feedback by itself can have no effect on V1 response altogether. To illustrate this, for these stimuli *all* cells in V1 received top-down stimulation.

Given the results of the psychophysical data it is unlikely that the attentional effect in the case of the original dual-axis stimuli is caused by a top-down signal that directly modulates all cells with the same orientation preference across the field of vision. However, these results leave open the possibility that an attentional signal generated by local feature similarity modulates the target-flanker integration rather than modulate cell response directly. In terms of the model, it is still unclear whether feedback is feature-specific for the central V1 RF only, or affects a larger part of the field of vision. This issue will be resolved in the next section.

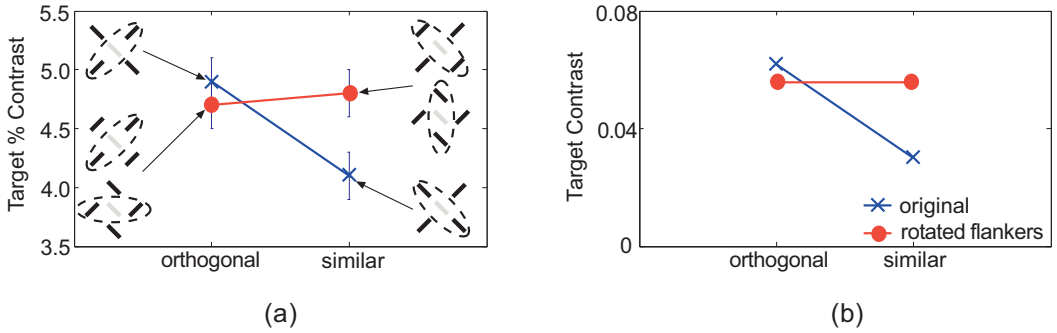

Fig. 13. Contrast detection thresholds for original dual axis and rotated-flankers stimuli. Results for rotated-flanker configurations are pooled into two groups: 'attend orthogonal' and 'attend similar' (see Fig. 9(c)-(d) for an explanation of these labels). (a) Contrast detection thresholds, averaged across experiments and subjects, adapted from (Freeman et al., 2004, Figure 5). (b) Simulation results.

4.4 Biased competition of perceptual groupings

In a final set of experiments Freeman and Driver (2005) varied the nature of the secondary task used to manipulate the allocation of attention to the flankers. Previously they had used the same secondary task (judging flanker misalignment or *Vernier* task) which imposed a global spatial relationship on the task-relevant flankers. The motivation for varying the secondary task was to investigate if such a global spatial relationship is a necessary condition, or if *any* sufficiently demanding task on the collinear flanker pair can produce facilitation. They therefore introduced secondary tasks that require a comparison between *local* flanker attributes (contrast, colour, and local orientation) and compared them with secondary tasks that require judgments on the global virtual contour connecting the relevant flanker pair (global orientation judgement and Vernier misalignment). Facilitation with both categories of secondary task would be expected if it were merely sufficient to direct spatial attention to the flankers, or if attention were "object-based", i.e., attending to one attribute of a stimulus element (e.g., colour) means that all its attributes are attended. Conversely, if the top-down signal into V1 depended critically on attention to the global relationship between the flankers, then only facilitation for the global tasks would be expected. For dual-axis stimuli the outcome of these experiments clearly established that tasks requiring discrimination of local flanker attributes do not produce facilitatory effects, while facilitation does occur for tasks that involve judgement of global stimulus characteristics. However, for single-axis stimuli facilitation did occur regardless of the secondary task. Freeman and Driver (2005) argued that this difference may follow from the ambiguous nature of the dual-axis stimulus. It contains two axes along which a grouping may occur, or it may even be perceived as an 'X' pattern. Top-down input directed to one of the two axes may act to resolve the ambiguity and result in a stable perceptual grouping along a single axis.

For secondary tasks judging local or non-spatial stimulus attributes the global structure of the stimulus is task-irrelevant and top-down intervention into the perceptual grouping process may therefore not occur. For single-axis stimuli there is only ever one unambiguous grouping; top-down input is therefore not required to resolve the perceptual grouping process, or may be generated automatically by bottom-up stimulation due to the perceptual saliency of the single global axis.

Freeman and Driver (2005) compared the involvement of top-down input in resolving the perceptual grouping to the biased competition theory of attention (Desimone and Duncan, 1995). Together with the results from Sect. 3.3, the simulation results we present in this section suggest that both the task-dependent collinear facilitation in V1 and the direct attentional modulation in areas V2 and V4 may be part of the same perceptual process. In particular, our results indicate that a biased competition between stimuli falling inside the RF of single cells in extrastriate areas V2 and V4 results in exactly the type of task-dependent feedback into V1 that would be expected from the psychophysical results of (Freeman and Driver, 2005). We simulated the network consisting of areas V1, V2 and V4 (see Fig. 2) with the dual- axis stimuli from Fig. 9(b). Flanker contrast is set to 0.5 and target contrast is varied between 0.0 and 0.1; contrast detection thresholds are obtained as before. External feedback targets the apical dendrites of neurons in V4 (with a strength of 1), and feedback into V1 is generated internally in areas V2 and V4. There are three attentional conditions: feedback targeting the V4 node that represents the global contour collinear with the target; feedback targeting the global contour orthogonal to the target; and no feedback into V4 at all. The first case corresponds to the psychophysical 'attend collinear' condition, the second to the 'attend orthogonal' condition, and the third case simulates the psychophysical tasks which require judging *local* stimulus attributes as described in previous paragraph. The results presented in Fig. 14(a) are consistent with the psychophysical results: facilitation only occurs for the 'attend collinear' condition, but not for the 'attend orthogonal' nor the 'no attention' condition. The cell activity in the different areas makes clear why this happens. Each graph in Fig. 14(d) shows the response of cells in area V4 for near-threshold contrast levels. When there is no top-down input into V4 the ambiguity of the dual-axis stimulus causes ongoing and unresolved competition between the nodes in V4. Figure 14(c) shows that the same happens in V2. As a result there is no significant feedback into area V1 and facilitation does not occur. When attention is directed towards the contour orthogonal to the target the corresponding V4 node does show weak response enhancement, but sends its feedback to the central RF node that is orthogonal to the target; this node receives little or no feedforward input, hence no modulation occurs in V1. Finally, when attention biases the representation of the contour collinear with the target it leads to a resolution of the competition, a strong response of the V2 and V4 nodes consistent with the resulting perceptual grouping, and

a corresponding facilitation of the target node in V1. Increasing the contrast levels of the central target has the effect of reducing the ambiguity of the dual-axis configuration, and leads to a faster response for nodes in all areas. In the 'no attention' and 'attend orthogonal' case, increasing the target contrast to supra-threshold levels (results not shown) gives rise to a feedforward-driven grouping of the collinear contour which becomes more salient with increasing central target contrast. Although this bottom-up perceptual grouping would in turn cause response enhancement in the V1 cell representing the target, which now receives feedback and horizontal stimulation at the same time, this does not affect the detection *threshold* as target contrast is already at supra-threshold levels before this perceptual pop-out can occur. This phenomenon, however, leads to an important model prediction discussed in Sect. 5.

The psychophysical and simulation results presented in this section provide answers to the open questions that remained after the previous sections. Firstly, they reconcile the original proposition of Freeman et al. (2001) – namely, that the flankers require attention – with the model-imposed requirement that the central RF be attended in a feature-specific manner. Secondly, the psychophysical results on task-dependency make it unlikely that feedback into V1 is feature-specific for the entire field of vision (i.e., all neighbouring nodes with the same orientation preference as the attended flankers receive feedback stimulation). If that were the case, the psychophysical task of judging *local* flanker orientation (Freeman and Driver, 2005) should have produced facilitatory effects for the 'attend collinear' condition. Instead, the model suggests how orientation-specific top-down input into V1 may result from a biased competition in extrastriate areas. The model furthermore suggests that the biased competition of perceptual groupings, as proposed in (Freeman and Driver, 2005), is part of the same perceptual process as the biased competition of attention previously demonstrated in extrastriate cortical areas (Desimone and Duncan, 1995; Desimone, 1998; Reynolds et al., 1999; Reynolds and Chelazzi, 2004).

5 Discussion

In this paper we proposed two mechanisms that together may explain the attentional modulation of collinear facilitation in primary visual cortex. Our hypothesis is novel, testable (see Sect. 5.1), and provides a unified account of disparate but related visual phenomena, namely, collinear facilitation, perceptual grouping, and the biased-competition theory of attention.

The psychophysical experiments discussed in Sect. 4.4 demonstrated that attention does not simply act as a switch, turning lateral interactions on and off, but operates by biasing a competition between different perceptual group-

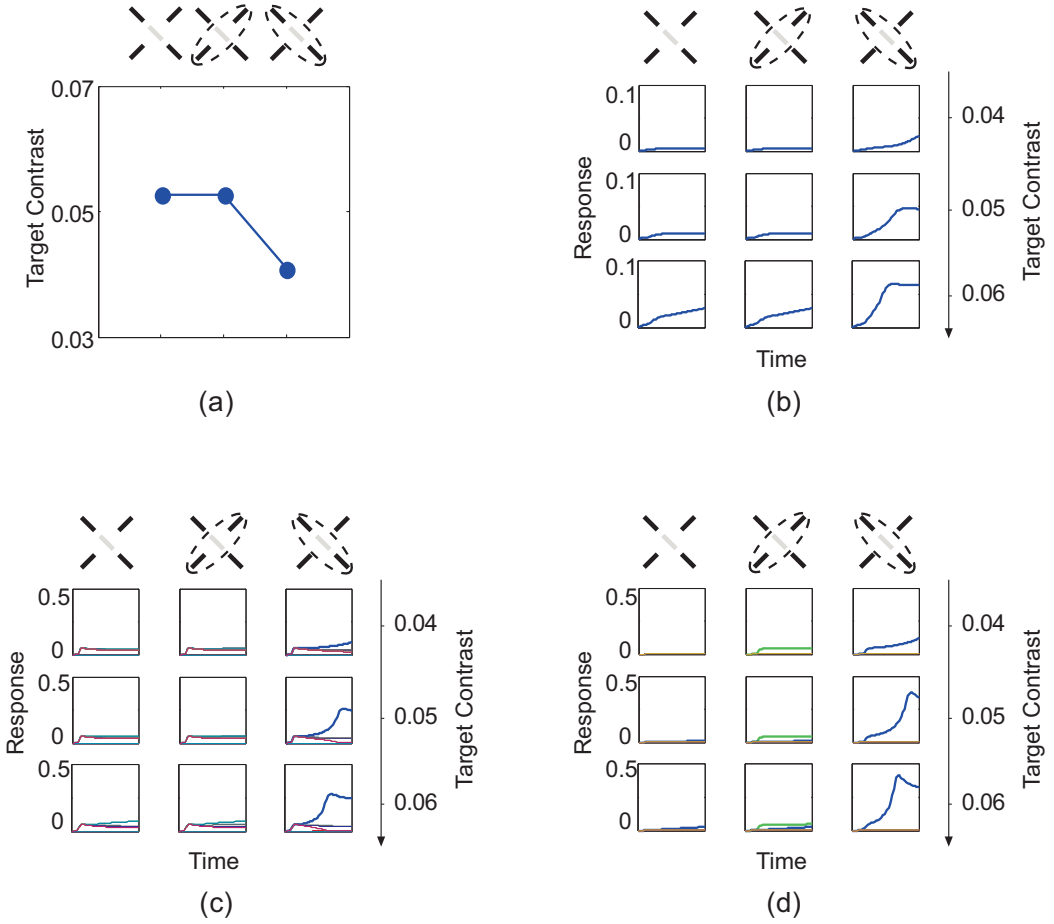

Fig. 14. Simulation results illustrating the biased competition in extrastriate areas and the resulting increase in contrast sensitivity in V1.(a) Contrast detection thresholds for the dual axis stimulus, for 3 different attentional conditions. (b) Temporal response of the central target node in V1, for different contrast levels and attentional conditions. (c) Temporal response of all neurons in V2 for the same conditions as in (b). (d) Temporal response of all neurons in V4 for the same conditions as in (b).

ings of the dual-axis stimuli. In our model the biased competition of perceptual groupings occurs in extrastriate cortical areas. This distinction between collinear facilitation proper (occurring within model area V1) and the perceptual grouping or contour integration process (distributed over all model areas) is supported by recent psychophysical evidence. Huang, Hess, and Dakin (2006) found that collinear facilitation and contour integration occur at different stages of the cortical pathway. The former occurs at the earliest stages of visual cortical processing, while the latter appears to involve extensively, but not exclusively, extrastriate cortical processing.

5.1 Testable predictions

In the previous sections we demonstrated that a biophysically plausible model of cortical areas V1, V2 and V4 can successfully replicate physiological and psychophysical experiments on collinear facilitation and attentional modulation. To the best of our knowledge this model is the first to provide an integrated account of these empirical data. The model is based on two critical assumptions: (1) attention modulates target-flanker integration in V1 through non-linear interactions between top-down and horizontal input targeting different parts of pyramidal cell dendrites; (2) the attentional top-down signal is generated by a biased competition in extrastriate areas V2 and V4. These critical assumptions can be tested on anatomical, physiological as well as psychophysical level. We present a number of specific and testable predictions in the following paragraphs.

5.1.1 Resolving the perceptual ambiguity of the dual-axis stimulus abolishes the attentional effect

At the end of Sect. 4.4 we noted that for increasing target contrast the model shows a bottom-up perceptual grouping of the combined target-flanker axis – and hence a facilitation of target cell response – regardless of how the external attention is allocated. This effect only comes into play at supra-threshold target contrast levels, and hence does not affect the target detection threshold itself. However, this phenomenon in the model gives rise to a prediction that can be tested psychophysically: if the top-down signal into V1 is caused by a perceptual grouping process in extrastriate cortex, then the attentional effect should disappear when the ambiguity in the dual-axis stimulus is resolved. On the other hand, if the top-down signal depends only on the secondary task itself – hence, attention targets nodes in V1 *directly* instead of operating through the perceptual grouping process in extrastriate cortex – then the attentional effect should remain even in the presence of a disambiguating stimulus. For example, adding a relatively low-contrast central *pedestal* to the dual-axis stimulus would favour perceptual grouping of one axis over the other. In this case, the primary psychophysical task would then become one of contrast *increment* detection rather than contrast detection *per se*. Such psychophysical experiments have been performed for single-axis stimuli (Solomon et al., 1999), but so far not for dual-axis configuration.

5.1.2 Critical timing of feedforward, horizontal and top-down stimulation

In Sect. 2.1 we proposed that horizontal and top-down stimulation of the apical dendrite need to co-occur for response modulation to take place. In fact,

the dendritic spiking mechanisms upon which our single-cell model is built predict a precise, critical time-window for the co-occurrence of dendritic events (Stuart and Hausser, 2001; Larkum et al., 2001). For instance, for boosting of bAPs to occur, depolarisation of the apical dendrite needs to occur in an interval 15ms to 0ms before the bAP (Stuart and Hausser, 2001). Such critical time dependencies for the co-occurrence of top-down and horizontal input may be investigated physiologically on the level of the single cell with e.g., advanced calcium-imaging techniques, but are also open to investigation on the behavioural level. For instance, (Cass and Alais, 2006) developed a technique used to study the critical time-dependence between collinearity onset and target stimulus presentation for single-axis stimuli. This technique could be combined with the dual-axis paradigm and a manipulation of the onset of attention to investigate if the three-way time dependence of target presentation, collinearity, and attention is consistent with the critical time-windows observed for the dendritic mechanisms.

5.1.3 The horizontal pathway gates the top-down pathway

Gilbert et al. (2000) proposed that attention, through top-down connections, gates the effect of collinear flankers thought to operate through horizontal connections. However, in our description of the single-cell model in Sect. 2.1 we observed that the gating of top-down and horizontal input is *mutual*. One could therefore swap the proposition around and state that the horizontal pathway gates the effects of top-down connections arriving at the distal apical dendrite. Such gating of distally arriving connections by more proximally arriving connections has recently been shown to exist for hippocampal cells (Jarsky et al., 2005). Moreover, it seems to depend on the same type of active dendritic membrane properties that exist in cortical pyramidal cells (see Sect. 2.1). What remains to be demonstrated is that a similar gating of the top-down pathway by the horizontal pathway is at work on the level of neural circuits in primary visual cortex. We consider this to be a critical test of our model and its underlying assumptions.

5.2 Related and future simulation studies

The model we propose focusses on excitatory dendritic interactions to explain the attentional modulation of collinear facilitation in V1 and attentional modulation in cortical areas V2 and V4. However, many centre-surround interactions in primary visual cortex are inhibitory (Series et al., 2003; Angelucci and Bressloff, 2006). Horizontal connections in primary visual cortex make around 20% of synapses with inhibitory interneurons (McGuire et al., 1991), and disynaptic inhibition mediated by horizontal connections has been demonstrated

(Hirsch and Gilbert, 1991; Yoshimura et al., 2000; Tucker and Katz, 2003a,b). This inhibitory component of the horizontal pathway may be responsible for some (but not all (Angelucci and Bressloff, 2006)) of the long-range suppressive centre-surround interactions. For instance, it appears to give rise to the contrast-dependence of the number of cells experiencing excitation or inhibition in the presence of collinear flankers (Chen et al., 2001) – see also Sect. 3.1, Table 2.

Several previous models (Li, 1999; Grossberg and Raizada, 2000; Schwabe, Obermayer, Angelucci, and Bressloff, 2006; Schafer, Vasilaki, and Senn, 2007; Setic and Domijan, 2007) have been used to successfully replicate facilitatory and suppressive centre-surround interactions, although none of them has been able so far to replicate the task-dependent modulation of collinear facilitation described in Sect. 4.4. These models generally employ an additional population of inhibitory interneurons to model a long-range inhibitory pathway. Because our model only contains short-range lateral inhibition, it is unlikely to reproduce suppressive centre-surround interactions caused by this long-range inhibitory pathway. Extending the current model with such an inhibitory pathway – by including additional inhibitory interneurons contacted by the horizontal connections – should enable it to simulate a wider range of centre-surround interactions. We do believe, however, that the current model is capable of explaining other facilitatory centre-surround interactions, such as the *attractive tilt illusion* (Kapadia et al., 2000). We also believe that in its present form it can already replicate suppressive surround interactions that are not the consequence of the long-range inhibitory pathway, but of a withdrawing of excitatory top-down stimulation (Sullivan and de Sa, 2006). We plan to address these issues in further modelling studies.

6 Conclusion

The simulation results we presented in this paper show that a neural network model incorporating mechanisms of intrinsic dendritic computations can account for physiological and psychophysical data on the attention gating of contextual interactions. In our model, attentional gating follows from a mutual gating of horizontal and top-down connections in primary visual cortex. The biological plausibility of this mechanism is supported by in vitro and in vivo studies on dendritic computation in cortical pyramidal cells. Our results also indicate that the biased competition of perceptual groupings, proposed to generate the signal for attentional gating in V1 (Freeman and Driver, 2005), may be a special instance of the more general biased competition theory of attention.

A Implementation Details

A.1 Activation

For each node in the network the activation of the distal (d) and proximal (p) apical compartments are calculated as a weighted sum of inputs:

$$y^t_{jk,d} = \sum_{i=1}^{m_d} u_{ijk} x^t_{ik,d} \tag{A.1}$$

$$y^t_{jk,p} = \sum_{i=1}^{m_p} v_{ijk} x^t_{ik,p} \tag{A.2}$$

Where $y^t_{jk,d}$ and $y^t_{jk,p}$ are the activations of the distal and proximal apical compartments of node j in area k at time t; $x^t_{ik,d}$ and $x^t_{ik,p}$ are the input activities received by the distal or proximal apical dendrite at time t; u_{ijk} and v_{ijk} are the synaptic weights from input i to node j in area k for distal and proximal apical dendrite respectively; m_d and m_p denote the total number of synapses on the distal and proximal apical dendrite.

For each node the activation of the basal (b) compartment is calculated as:

$$y^t_{jk,b} = (y^{t-1}_{jk} + \epsilon_1) \sum_{i=1}^{m_b} w_{ijk} \hat{x}^t_{ik,b} \tag{A.3}$$

$$\hat{x}^t_{ik,b} = \frac{x^t_{ik,b}}{\sum_{q=1}^{n} (\hat{w}_{iqk} y^{t-1}_{qk}) + \epsilon_2}, \quad \hat{w}_{iqk} = \frac{w_{iqk}}{\max_i w_{iqk}} \tag{A.4}$$

Where $y^t_{jk,b}$ is the activation of the basal dendrite of node j in area k at time t; y^{t-1}_{jk} is the response of node j in area k at time $t-1$ (defined below in Eq. A.5); w_{ijk} is the feedforward – basal – synaptic weight from input i to node j in area k; $\hat{x}^t_{ik,b}$ denotes input activation received at the basal dendrite after application of a form of divisive lateral inhibition; $x^t_{ik,b}$ is the uninhibited feedforward input; $\hat{w}_{iqk}$ are the feedforward weights normalised by the maximum incoming weight for that node; n is the total number of cells in area k. The inhibitory operation on feedforward inputs $x^t_{ik,b}$ can be interpreted as a divisive form of pre-integration lateral inhibition (Spratling and Johnson, 2001, 2002) or as a non-linear form of predictive coding (Spratling, 2008). ϵ_2 is a small constant introduced to prevent division-by-zero errors. The ratio $\frac{\epsilon_1}{\epsilon_2}$ determines the input/output gain of the cell when $y^{t-1}_{jk} \approx 0$ for all j in k. The latter condition applies when the uninhibited feedforward inputs $x^t_{ik,b}$ are very

weak or at the start of the iterative process when $y^0_{jk} = 0$ for all nodes j in area k. Parameter sensitivity is discussed below.

y^t_{jk}, the response of cell j in area k at time t, is calculated from the activation of the dendritic compartments: basal, distal apical and proximal apical.

$$y^t_{jk} = y^t_{jk,b}(1 + \sigma_d(y^t_{jk,d})\sigma_p(y^t_{jk,p})) \tag{A.5}$$

This formulation enables bottom-up, sensory-driven, stimulation to drive the response of the node even in the absence of top-down activity. In contrast, feedback and/or horizontal activation cannot drive the node's activity in the absence of feedforward activation. $\sigma(.)$ is a sigmoid function modelling the saturation of the two apical compartments, as discussed in Sect. 2.1:

$$\sigma_d(y^t_{jk,d}) = \frac{1}{1 + e^{-\alpha_d(y^t_{jk,d} - \beta_d)}} \tag{A.6}$$

$$\sigma_p(y^t_{jk,p}) = \frac{1}{1 + e^{-\alpha_p(y^t_{jk,p} - \beta_p)}} \tag{A.7}$$

Parameters α_d, α_p, β_d and β_d determine the shape of the saturation functions. For cells in V1 they are chosen such that horizontal and top-down input can only modulate the cell response when both sources of stimulation are active simultaneously. For cells in V2 and V4 they are chosen such that top-down input can modulate cell response directly.

The presence of reciprocal excitatory connections can lead to positive feedback effects resulting in run-away activation values. To prevent this the activity of each node is attenuated in proportion to the cumulative strength of its previous activity (C^{t-1}_{jk}):

$$y^t_{jk} = \frac{y^t_{jk}}{1 + C^{t-1}_{jk}} \tag{A.8}$$

With the cumulative activity, C^t_{jk}, of the node calculated as:

$$C^t_{jk} = \tau_c y^t_{jk} + (1 - \tau_c)C^{t-1}_{jk} \tag{A.9}$$

τ_c is a time constant influencing the temporal dynamics of the cell response. The response of the network to a particular input is obtained by iterating – for all cells – through the above equations for $t = 1 \rightarrow t_{max}$, with initial conditions $y^0_{jk} = 0$ and $C^0_{jk} = 0$.

Table A.1
Simulation parameters, as defined in Sect. A.1. The last parameter, θ, is the response threshold defined in Fig. 10.

Parameter	Area	Value	Range
α_d	V1,V2,V4	20	8–50
α_p	V1,V2,V4	20	≥ 1
β_d	V1,V2,V4	0.2	0.15–0.25
β_p	V1	0.2	0.1–0.6
	V2,V4	-0.5	≤ -0.1
$\epsilon 1$	V1,V2,V4	0.001	0.00001–0.01
$\epsilon 2$	V1,V2,V4	0.05	0.02–0.06
τ_c	V1,V2,V4	0.1	0.01–1.0
θ	(Fig. 10)	0.01	0.005–0.05

A.2 Parameter sensitivity

All experiments were performed using the parameter values given in Table A.1. These values were selected such that the magnitude of the model results are comparable to the results of the psychophysical experiments. Changing the values of these parameters can give rise to results that are qualitatively similar (i.e., show a clear attentional effect), but are quantitatively different. To obtain an estimate of the parameter sensitivity we repeated the experiment of Sect. 4.4 with different parameter values, changing one parameter at a time while keeping all other parameters fixed. The range of values for which qualitatively similar results are obtained is given in the last column of Table A.1. As can be seen, most parameters can be varied over quite a large range without affecting the qualitative results.

A.3 Synaptic weight values

All synaptic weights – except the weights of the external feedback connections – were obtained by training. The primary reason for this approach wss to avoid having to set synaptic weights by hand. We used tried and tested training procedures from previous work instead (Spratling and Johnson, 2006; Spratling, 2008). The different model areas were trained in different stages and with different sets of training images. The network first learned the feedforward (basal) weights of model area V1 using simple bar patterns in various orientations and locations, such as the ones depicted in Fig. 3(a). These weights

were fixed and the network then learned – simultaneously – the weights of the horizontal connections in V1, the feedforward weights from V1 to V2, and the feedback weights from V2 to V1, with two-bar training patterns as depicted in Fig. 3(b). Finally, the network learned simultaneously the feedforward weights from V2 to V4, the feedback weights from V4 to V2, and the feedback weights from V4 to V1 using the longer contours depicted in Fig. 3(c).

During an iteration of the training procedure, an input image was presented to the network and the equations for all network nodes, as described in Sect. A.1, were iterated t_{max} times. The final input and output activation values were then used to adjust the synaptic weights. The feedforward connections were adapted using the learning rule from (Spratling, 2008):

$$w_{ijk} \leftarrow w_{ijk}(1 + y_{jk}(\hat{x}_{ik,b} - 1) \tag{A.10}$$

Where $\hat{x}_{ik,b}$ is the inhibited input activation and y_{jk} is the cell response after t_{max} steps. The total sum of the synaptic weights received at each node's basal dendrite is kept equal to one ($\sum_{i=1}^{m_b} w_{ijk} = 1$). Before the start of the training procedure, feedforward weights were initialised to reflect the overall retinotopical structure of Fig. 2. Inputs falling outside a node's receptive field were initialised to zero and remained zero during the entire training procedure, as follows from Eq. A.10. For inputs falling within the receptive field of a node, the weights were initialised to 1, and a small amount of noise (drawn from a normal distribution with $mean = 0$ and $std = 0.01$) was added, after which weights were normalised as described above. For nodes in V1, the addition of noise is essential to ensure that they have slightly different preferences at the start of the training procedure, and subsequently develop unique representations during training. Furthermore, to ensure that model area V2 and V4 learn distinctive representations (feedforward weights) for each of the training patterns, it was found necessary to supply a top-down bias to the distal apical compartment of one distinct node for each of the different patterns. Experiments in (Spratling and Johnson, 2006) have demonstrated that such bias leads to *exemplar* learning, as opposed to *prototype* learning that may occur in absence of the bias.

The horizontal and feedback connections were modified using the learning rule employed in (Spratling and Johnson, 2006):

$$u_{ijk} \leftarrow u_{ijk} + \frac{\gamma(x_{ik,d} - \bar{x}_{k,d})}{\sum_{q=1}^{m_d}}(y_{jk} - \bar{y}_k)^+ \tag{A.11}$$

$$v_{ijk} \leftarrow v_{ijk} + \frac{\gamma(x_{ik,p} - \bar{x}_{k,p})}{\sum_{q=1}^{m_p}}(y_{jk} - \bar{y}_k)^+ \tag{A.12}$$

Where $\bar{x}_{k,d}$ and $\bar{x}_{k,p}$ are the means of input activations of distal and proximal

dendrites in area k; γ is a parameter controlling the learning rate ($\gamma = 0.1$ was used here); $\bar{y}_k$ is the mean cell response in area k; operation $()^+$ denotes positive rectification, i.e., its result is 0 when its operand is negative, and the unaltered value of the operand otherwise. Synaptic weights that reached a value of zero were clamped to zero. Furthermore, weights were clipped at a maximum value of 1. The net effect of this learning procedure is that weights tend to grow towards 1 for horizontal and feedback connections linking nodes that are frequently coactive, and become zero otherwise. Top-down weights u_{ijk} were all initialised to the same small value (0.01). Horizontal weights v_{ijk} wre initialised to a small value (0.01) for nodes with neighbouring receptive fields, and to zero for distant nodes (more than 2 RF centres away) or nodes with the same receptive field.

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