Vision Research 49 (2009) 553–568

Contents lists available at ScienceDirect

Vision Research

journal homepage: www.elsevier.com/locate/visres

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

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ARTICLE INFO

Article history:
Received 21 April 2008
Received in revised form 21 October 2008

Keywords:
Contrast detection
Contour integration
Lateral interactions
Attention
Perceptual grouping
Cortex
Neural network
Dendrites

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.

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 & 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, & Fregnac, 2003; Angelucci & 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 & 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, & Westheimer, 1995; Polat, Mizobe, Pettet, Kasamatsu, & Norcia, 1998; Chen, Kasamatsu, Polat, & Norcia, 2001; Mizobe, Polat, Pettet, & 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 & 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 & Sagi, 1993, 1994).

Physiological and psychophysical experiments have shown that collinear facilitation is modulated by task context or attentional state (Ito & Gilbert, 1999; Gilbert, Ito, Kapadia, & 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, & Driver, 2001, 2004; Freeman, Driver, Sagi, & Zhaoping, 2003; Freeman & 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 & Driver, 2005).

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doi:10.1016/j.visres.2008.12.017

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 & Armstrong, 2003; Armstrong, Fitzgerald, & 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 & 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, Freeman, Sagi, & Driver, 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 & 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 & Duncan, 1995).

The paper is organised as follows: in Section 2 we introduce the model and explain how it is grounded in anatomical and physiological constraints. In Section 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. Section 4 contains simulation results replicating the psychophysical data of (Freeman et al., 2001, 2003, 2004; Freeman & Driver, 2005). Finally, in Section 5, we discuss testable predictions, potential future experiments and how the model aids theory formation.

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 & 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, & Tank, 1994; Larkum, Zhu, & 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, Spruston, Sakmann, & Hausser, 1997; Waters, Larkum, Sakmann, & 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, & Sakmann, 2001; Larkum, Waters, Sakmann, & 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 & Mel, 2003; Spruston, 2008).

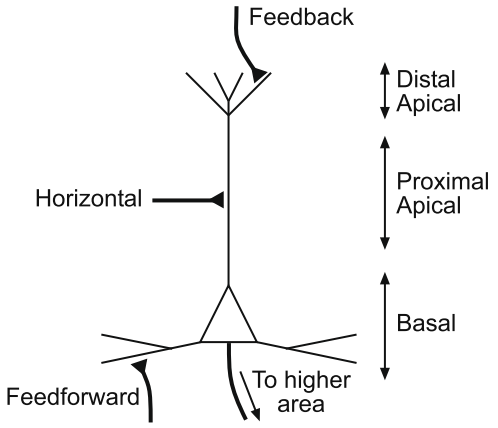

Fig. 1. Schematic of a pyramidal cell in the superficial layers (L2/3) of neocortex. Morphologically L2/3 pyramidal cells are characterised by basal dendrites that extend laterally from the soma, and by an apical dendrite that extends vertically into L1 and ends in a tuft of fine branches. Feedforward 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.

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 & 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 & Van Essen, 1991; Kapadia, Westheimer, & 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, & Wiesel, 1991; Yoshimura, Sato, Imamura, & 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