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Spiking Dynamics during Perceptual Grouping in the Laminar Circuits of Visual Cortex

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Spiking dynamics during perceptual grouping in the laminar circuits of visual cortex

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Abstract

Grouping of collinear boundary contours is a fundamental process during visual perception. Illusory contour completion vividly illustrates how stable perceptual boundaries interpolate between pairs of contour inducers, but do not extrapolate from a single inducer. Neural models have simulated how perceptual grouping occurs in laminar visual cortical circuits. These models predicted the existence of grouping cells that obey a bipole property whereby grouping can occur inwardly between pairs or greater numbers of similarly oriented and co-axial inducers, but not outwardly from individual inducers. These models have not, however, incorporated spiking dynamics. Perceptual grouping is a challenge for spiking cells because its properties of collinear facilitation and analog sensitivity to inducer configurations occur despite irregularities in spike timing across all the interacting cells. Other models have demonstrated spiking dynamics in laminar neocortical circuits, but not how perceptual grouping occurs. The current model begins to unify these two modeling streams by implementing a laminar cortical network of spiking cells whose intracellular temporal dynamics interact with recurrent intercellular spiking interactions to quantitatively simulate data from neurophysiological experiments about perceptual grouping, the structure of non-classical visual receptive fields, and gamma oscillations.
**Introduction: perceptual grouping in the laminar circuits of visual cortex**

Grouping of local image contrasts is an important step in the perceptual organization process that leads to the emergence of 3D object boundary representations. Such boundaries delimit object borders and surfaces, allowing the brain to build meaningful perceptual units in response to complex scenes, and thereby contributing to global form perception. While perceptual grouping has long been studied in psychology, and models of perceptual grouping have been proposed, it remains to fully characterize its mechanisms and functions in laminar cortical circuits. In particular, although models exist of how spiking neurons within laminar circuits of visual cortex support certain perceptual and cognitive processes, spiking laminar models of perceptual grouping remain to be characterized.

**Figure 1** Perceptual grouping by bipole cell interactions: (A) The four pac man figures induce the percept of a Kanizsa square whose sides are delimited by illusory contours. (B) Input from a single pac man is
insufficient to induce illusory contours. (C) Input from a pair of collinear pac man edges creates an illusory contour by activating bipole cells at all positions between them.

Illusory contour stimuli illustrate particularly well the requirements that must be satisfied by an adequate grouping mechanism. Illusory contours show that perceptual boundaries are completed only in regions enclosed by properly aligned boundary inducers. In particular, Figure 1A shows a Kanizsa square stimulus whose four pacmen inducers lead to the percept of a bright square bounded by illusory contours. A parsimonious explanation is that neural signals corresponding to almost collinear pairs of edge inducers complete over the gap that separates them, as reported by von der Heydt et al. (1984) and Peterhans and von der Heydt (1989), among others (see Table 1). It is then necessary to explain why illusory contours do not propagate from a single image inducer. In other words, how does inward boundary completion between pairs or greater numbers of inducers, on opposite sides of a target cell, occur without causing uncontrollable outward boundary propagation from a single inducer? This has been called the bipole grouping property in the Boundary Contour System, or BCS, model that has been developed by Grossberg and his colleagues (e.g., Cohen and Grossberg, 1984; Grossberg, 1984; Grossberg and Mingolla, 1985a, 1985b). Psychophysical experiments on association fields (Field, Hayes, and Hess, 1993) and contour interpolation (Kellman and Shipley, 1992), among others, have supported the bipole grouping concept. The more recent 3D LAMINART model has refined the analysis of bipole grouping by predicting how it takes place in laminar cortical circuits, and has thereby explained much larger psychophysical and neurobiological data bases that depend upon perceptual grouping, including properties of cortical development, perceptual learning, attention, 3D vision, and figure-ground separation (e.g., Grossberg and Raizada, 2000; Grossberg and Williamson, 2001; Grossberg and Swaminathan, 2004; Yazdanbakhsh and Grossberg, 2004; Cao and Grossberg, 2005; Grossberg and Yazdanbakhsh, 2005; Fang and Grossberg, 2009; Grossberg, Yazdanbakhsh, Cao, and Swaminathan, 2008). Neurons in the aforementioned laminar cortical models use rate coding, rather than spikes, to represent intercellular signals.

<table>
<thead>
<tr>
<th>Model connection</th>
<th>Functional interpretation</th>
<th>Selected references</th>
</tr>
</thead>
<tbody>
<tr>
<td>LGN → 4</td>
<td>Strong LGN input</td>
<td>Blasdel and Lund (1983); Ferster et al. (1996); Thomson et al. (2003)</td>
</tr>
<tr>
<td>4 → 2/3 pyramidal</td>
<td>Feedforward stimuli with bottom-up support</td>
<td>Fitzpatrick et al. (1985); Callaway and Wiser (1996); Shmuel et al. (2005)</td>
</tr>
<tr>
<td>V1 2/3 pyr. → 2/3 pyr.</td>
<td>Long-range collinear integration</td>
<td>Bosking et al. (1997); Schmidt et al. (1997); Chisum et al. (2003)</td>
</tr>
<tr>
<td>V2 2/3 pyr. → 2/3 pyr.</td>
<td>Long-range collinear integration</td>
<td>Levitt et al. (1994)</td>
</tr>
<tr>
<td>2/3 inhib. int. → 2/3 pyr.</td>
<td>Keep outward grouping subthreshold (bipole property)</td>
<td>Lund et al. (2001)</td>
</tr>
<tr>
<td>2/3 inhib. int. → 2/3 inhib. int.</td>
<td>Normalize 2/3 inhibition (2-against-1 principle)</td>
<td>Tamas et al. (1998); Fukuda et al. (2006)</td>
</tr>
</tbody>
</table>

Table 1: Model connections and supporting anatomical data

The present article models how perceptual grouping of boundaries emerges in a laminar cortical model of spiking neurons. Spiking neurons challenge the bipole property because communication using temporally discrete, not necessarily coincident, spikes increases the
difficulty of computing whether two or more inducers are contributing to the formation of grouping. How the brain overcomes this difficulty must be understood since the biological neurons that subserve perceptual grouping are spiking neurons, and many cortical processes are known to depend on spike timing; for example, spike-timing-dependent-plasticity (STDP; Markram et al., 1997).

What cortical mechanisms allow the bipole property to be realized in a spiking milieu? It is not sufficient to base such an analysis on the possibility that individual spikes may be coincident if only because outputs from multiple cells at multiple distances and time lags input to each bipole grouping cell from each side of its receptive field, and because the time scale of conscious perceptual grouping is orders of magnitude slower than the time scale of individual spiking coincidences. How the brain may synchronize temporally dispersed signals due to different axonal and synaptic delays has been demonstrated using a rate-based description of laminar cortical grouping within the 3D LAMINART model (Yazdanbakhsh and Grossberg, 2004), but not yet in a spiking grouping model.

Our study extends the 3D LAMINART model to explain how spike-based cortical grouping may occur. This extended model represents a synthesis of the 3D LAMINART model and the Synchronous Matching ART (SMART) model of Grossberg and Versace (2008). The SMART model has simulated how spiking dynamics in laminar cortical circuits can explain data bases other than perceptual grouping. In particular, SMART clarifies how bottom-up adaptive filtering and top-down learned expectation processes undergo match/mismatch operations that attentively regulate perceptual and cognitive processes, notably how multiple stages of laminar cortical processing interact with specific and nonspecific thalamic nuclei to control category learning and recognition, and how gamma and beta oscillations may be triggered in match and mismatch states, respectively. The current study focuses on the horizontal interactions that support perceptual grouping, rather than the bottom-up/top-down processes that regulate attention. A future study will synthesize both types of processes into a more comprehensive cortical model of how spiking dynamics are regulated in bottom-up, horizontal, and top-down laminar interactions.

Previous 3D LAMINART modeling work simulated how cortical layer 2/3 pyramidal cells respond to inputs from deeper cortical layers to group together (almost) collinear image features via long-range recurrent excitatory interactions between the pyramidal cells. This long-range oriented interaction limits contextual contributions to a restricted set of neighboring collinear layer 2/3 cells with a similar orientation preference and (almost) collinear positional alignment, as has been experimentally found (Table 1). Figure 1C illustrates how a pair of collinear pacman figures can activate bipole cells located between them to form an illusory contour. Similar grouping kernels have been reported by several authors (e.g. Field et al., 1993; Li, 1998). If only excitatory recurrent connections existed, run-away excitation could easily occur. This is prevented by balanced recurrent interactions between layer 2/3 long-range excitatory pyramidal cells and short-range inhibitory interneurons which together ensure that single-sided input, however strong, does not lead to horizontal activation (Figure 1B). An excitatory-inhibitory balance also ensures that inhibition is not too strong, thereby preventing maladaptive suppression of network activation. Earlier modeling has shown how such an excitatory-inhibitory balance can self-organize during cortical development and give rise to a laminar perceptual grouping circuit whose properties match perceptual data from adult human observers (Grossberg and Williamson, 2001). This analysis illustrates how adult perceptual properties may emerge from the dynamics that govern stable brain development.
Feedback from higher cortical areas to lower ones enables interaction of grouping circuits at different spatial scales. For example, activation of smaller-scale bipole cells in V1 is modulated by feedback from larger-scale bipole cells in V2, such that contextual elements at a larger spatial scale may sharpen the activity at the level of V1 bipole cells (Kisvárday et al., 1997; Grossberg and Raizada, 2000; Raizada and Grossberg, 2001; Grossberg and Swaminathan, 2004; Shmuel et al., 2005; Anzai et al., 2007). Although this article models data about V1 circuitry, the similarity of V1 and V2 circuits with respect to the presence of anisotropic horizontal collaterals suggests that variants of our results may also apply to V2. Table 2 lists supporting evidence from various species for the presence of bipole-type kernels in both V1 and V2.

<table>
<thead>
<tr>
<th>Species</th>
<th>Area</th>
<th>Horizontal extent [mm]</th>
<th>Selected references</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td>Area 17</td>
<td>8</td>
<td>Gilbert et al. (1983)</td>
</tr>
<tr>
<td></td>
<td>Area 18</td>
<td>6.5*</td>
<td>Kisvárday et al. (1997)</td>
</tr>
<tr>
<td>Tree shrew</td>
<td>V1</td>
<td>8</td>
<td>Bosking et al. (1997)</td>
</tr>
<tr>
<td>Monkey</td>
<td>V1</td>
<td>7</td>
<td>Stetter et al. (2002)</td>
</tr>
<tr>
<td></td>
<td>V2</td>
<td>8</td>
<td>Levitt et al. (1994)</td>
</tr>
</tbody>
</table>

Table 2: Maximal bilateral extent of horizontal connections from selected studies

The current Spiking LAMINART model (sLAMINART) uses Hodgkin-Huxley (1952) dynamics to represent realistic neuronal biophysical membrane constraints (cf. Gautrais and Thorpe, 1998). The model depends upon intracellular dynamics to temporally average across irregularities in individual spike timing, and to thereby enable bipole grouping to occur in response to approximately coincident bursts of spikes. The prevention of outward spreading of activation in response to individual inducers also exploits spiking mechanisms. Hodgkin-Huxley type cells can behave as threshold units due to the presence of a stable attractor state during a period with little or no input (FitzHugh, 1955; Carpenter, 1979; Izhikevich, 2007). Thresholds help to minimize noise propagation, which is critical for robustness in networks with multiple layers (Sarpeshkar, 1998). Previous rate-based bipole models used an explicit rectification, or threshold, to prevent boundary propagation in response to individual inducers. In sLAMINART, such rectification is implicit and derives from Hodgkin-Huxley membrane dynamics within each cell.

Each cell’s intracellular dynamics supports a graded, or analog, activation profile. A critical property of a properly designed network is that it retains analog sensitivity even as it binds multiple cell activations together into emergent groupings through recurrent interactions.

* Although the estimated horizontal extent appears smaller for area 18 than for area 17, the lower cortical magnification factor in area 18 (0.75 mm²/degrees² for area 18 Vs 3.6 mm²/degrees² for area 17 near area centralis; Tusa et al., 1979) means that horizontal connections in that area span a wider range of visual angle, and thus can be considered as functioning at a larger scale.
Such a coexistence of analog sensitivity with coherent binding is called *analog coherence*, and helps to explain effects of contrast magnitude on perceptual grouping (Grossberg et al., 1997; Grossberg, 1999). sLAMINART successfully simulates a range of single-cell recording data about analog-sensitive perceptual grouping, and hereby shows how digital spiking dynamics can induce analog coherence. In particular, sLAMINART quantitatively simulates data about short-range grouping (Kapadia et al., 2000), long-range modulation (Polat et al., 1998; Crook et al., 2002), contrast sensitivity (Polat et al., 1998), horizontal summation (Chisum et al., 2003), and gamma-range oscillations (Gray et al., 1989). The next section describes the sLAMINART model, followed by a section that summarizes model simulations.

**Method**

The sLAMINART model defines and simulates a layer 2/3 spiking bipole grouping circuit, fed by inputs from deeper cortical layers, described for simplicity as layer 4 in Figure 2. Each spatial location along the horizontal axis roughly corresponds to one hypercolumn. Pyramidal cells mutually excite each other via long-range horizontal connections. Inhibitory interneurons in each hypercolumn are divided into two populations. As shown in Figure 2, one population receives excitatory long-range horizontal input from layer 2/3 pyramidal cells in hypercolumns located to their left. The other population receives excitatory long-range horizontal inputs from layer 2/3 pyramidal cells located to their right. Henceforth we refer to the two populations of interneurons as *Left* and *Right*, respectively. Each layer 2/3 inhibitory interneuron inhibits the layer 2/3 pyramidal cell and the antagonist interneuron in the same hypercolumn. This inhibitory scheme is designed to realize the bipole property within a laminar cortical circuit (Grossberg and Raizada, 2000; Raizada and Grossberg, 2001).

For example, when a pyramidal cell receives input from a single excitatory pyramidal cell to its left, it also receives a balanced inhibitory input from its Left inhibitory interneuron (“one against one”). Hence, individual pyramidal cells cannot cause run-away excitation across the network due to the way in which the pyramidal cell temporally averages the excitatory and inhibitory spikes. When a pyramidal cell receives collinear inputs from excitatory pyramidal cells to its left and its right, these flanking pyramidal cells activate the corresponding Left and Right inhibitory interneurons. Both of these interneurons inhibit the target pyramidal cell, as well as one another. The mutual inhibition of the inhibitory interneurons acts to normalize their total activity (Grossberg, 1973). As a result, the total excitation to the target pyramidal cell increases, but the total inhibition remains similar to the level of inhibition from an individual inhibitory interneuron (“two against one”). The target pyramidal cell can therefore fire, again due to the way in which the pyramidal cell temporally averages the excitatory and inhibitory spikes.

![Figure 2 Bipole cell circuit: Input is clamped at each spatial location at the level of Layer 4 pyramidal cells. Each layer 4 pyramidal cell projects to the dendrite of a single layer 2/3 pyramidal cell. The pyramidal cell dendrite also receives horizontal excitatory connections from neighboring layer 2/3](image-url)
pyramidal cells, including projections from itself. Layer 2/3 horizontal connections also excite the dendrites of left or right inhibitory interneurons, which inhibit neighboring layer 2/3 pyramidal cell bodies and right or left inhibitory interneuron cell bodies. See Appendix equations (11)-(17).

Each layer of the model is composed of a one-dimensional array of 51 neurons. Layer 4 cells are implemented with a single somatic compartment governed by Hodgkin-Huxley dynamics. External input to the model is provided via current injection in the soma of layer 4 cells. Pyramidal cells and interneurons in layer 2/3 have an additional passive dendritic compartment, which is consistent with pyramidal cell anatomy and enables smoother temporal integration of excitatory inputs from other pyramidal cells. This smoother integration results from the cable equation, whose leaky integrator dynamics ensure that both the temporal integration of inputs from pre-synaptic afferents into the dendritic compartment, and the transfer of current from the dendrite to the soma, are much slower than individual spike events.

Smooth integration is needed to obtain stable grouping in the presence of pre-synaptic spikes that are not coincident due to distance-dependent axonal delays and the presence of noise. Besides providing better stability, the presence of a dendritic compartment also allows selective enhancement of inhibition by inhibitory synapses on the soma, while excitatory synapses terminate on the dendrite (Megías et al., 2001; Spruston, 2008). This anatomy allows inhibition to reach the soma faster than excitation, thereby helping to prevent spurious outward propagation of activity. Another consequence of this specific placement of synapses is to make the spike trains of bipole cells look like that of bursting cells, although they are not intrinsic bursters. Indeed, the occurrence of an inhibitory spike to the soma of an excited bipole cell is strong enough to induce a pause in that cell’s otherwise constant firing activity, yielding spike trains similar in appearance to various types of intrinsic bursters (Carpenter, 1979).

Synaptic interactions are implemented as double exponentials with parameters corresponding to $\alpha$-amino-3-hydroxy-5-methylisoxazolepropionic acid (AMPA) and $\gamma$-Amino-Butyric Acid (GABA) receptors for excitatory and inhibitory synapses, respectively. The location of synaptic contacts is on the passive dendrite for AMPAergic synapses and on the soma for GABAergic synapses. The model includes realistic distance-dependent axonal delays (Hirsch et al., 1991; Bringuier et al., 1999; Girard et al., 2001). Mathematical equations and parameters, as well as details pertaining to the simulation protocol, are included in the Appendix. All simulations were conducted using KDE Integrated Neurosimulation software (KInNeSS; Versace et al., 2008).

Results

Illusory contour

The combination of the above factors, notably the bipole-organized balance of excitation and inhibition, intracellular temporal spike averaging, and the differential locations of excitatory and inhibitory contacts on soma and dendrite, respectively, supports stable firing of layer 2/3 spiking bipole cells during grouping of an illusory contour (Figure 3). The bottom plot of Figure 3 shows a 1D stimulus pattern corresponding to two collinear flanking stimuli (each 3 spatial locations wide) separated by a wide gap (5 spatial locations). Inducers were simulated by injecting 0.03nA current inputs into layer 4 pyramidal cells. The middle plot shows the firing rate of layer 4 pyramidal cells, where completion does not occur. Note, unless noted otherwise, reported firing rates are computed over 500ms of simulation following an initial transient. The upper plot shows the firing rate of layer 2/3 bipole cells, where inward completion is present. Adding small
amounts of noise in layer 2/3 dendrites leads to negligible activity in a few cells located just outside of the stimulus pattern.

Figure 3 Long-range completion in layer 2/3 spiking bipole cells: Two flanking stimuli (bottom) separated by a wide gap (5 hypercolumns) give rise to corresponding activity in layer 4 pyramidal cells (middle). The layer cell activities generate inputs activity pattern to vertically aligned layer 2/3 bipole cells (top), whose activation leads to inward completion of activity. Note the absence of outward propagation of activity. See text for further details.

Short-range grouping
Spatially short-range grouping is illustrated in Figure 4, where model simulations are plotted together with monkey V1 data from three different conditions: flankers-only, target-only, and target-with-flankers (Kapadia et al., 2000). The size of the illusory contour gap and of the inducers in these simulations was determined by taking into consideration the cortical magnification factor and details about the original experimental protocol. In particular, the gap and inducers both cover one spatial location in the simulations; see the Appendix. In the flankers-only condition, two bars were presented adjacent to the *classical receptive field* (CRF) of a recorded cell.

Note that the firing rate in the flankers-only grouping simulation of Figure 3 is higher than that shown in Figure 4. This is due to several interacting factors: First, the simulated illusory contour gap is wider in Figure 3 than in Figure 4, but the inducer length is also wider. Second, the horizontal projections are weaker than the bottom-up projections (in Table 3, $g_{\text{max}}$ equals 0.003 and 0.049 for horizontal and bottom-up projections, respectively). Thus, although the amount of current input in Figure 3 was the same as in the high contrast conditions of Figure 4 (0.03nA in both cases, denoted as 50% contrast in Figure 4), the amount of support is greater in Figure 3, resulting in a higher firing rate in that case due to horizontal summation of wider bottom-up inputs.
<table>
<thead>
<tr>
<th>Pre-Synaptic</th>
<th>Post-Synaptic</th>
<th>Post-synaptic Site</th>
<th>E [mV]</th>
<th>g max [mS/cm²]</th>
<th>τᵣ [ms]</th>
<th>τᵣ [ms]</th>
<th>W</th>
<th>δAx [ms]</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 pyr.</td>
<td>2/3 pyr.</td>
<td>dendrite</td>
<td>0</td>
<td>0.049</td>
<td>2</td>
<td>4</td>
<td>0.2</td>
<td>3</td>
</tr>
<tr>
<td>2/3 pyr.</td>
<td>2/3 pyr.</td>
<td>dendrite</td>
<td>0</td>
<td>0.003</td>
<td>2</td>
<td>6.5</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>2/3 pyr.</td>
<td>2/3 int.</td>
<td>dendrite</td>
<td>0</td>
<td>0.037</td>
<td>1</td>
<td>4</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>2/3 int.</td>
<td>2/3 pyr.</td>
<td>soma</td>
<td>-60</td>
<td>1.8</td>
<td>2</td>
<td>7</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>2/3 int.</td>
<td>2/3 int.</td>
<td>soma</td>
<td>-70</td>
<td>1</td>
<td>2</td>
<td>7</td>
<td>1</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Table 3: Synaptic connection parameters. Weights (W) and delays (δAx) of horizontal connections are determined by equations (19) and (20) respectively.

The model explains the three cases depicted in Figure 4 as follows. In the flankers-only condition, summation of horizontal inputs leads only to weak increments in firing rates, compared to the other two conditions, as noted above. Just as excitatory horizontal input is weak, inhibitory input to the target bipole is also weak, since interneurons in the target hypercolumn inhibit each other through recurrent connections. This balance of excitation and inhibition enables the target bipole to emit spike trains, resulting in short-range grouping. In the target-only condition, bottom-up input from the layer 4 cell activates the layer 2/3 bipole across a wide dynamic range. The reason why the range of firing rate is greater in this condition than in the other two is that it is the only one where inhibitory interneurons in the hypercolumn of the recorded bipole cell do not receive excitatory inputs from neighboring hypercolumns. Given the graded output profile of single Hodgkin-Huxley cells modeled here (see Figure 11), the target bipole cell is thus free to span a wide range of firing rates. Finally, in the target-with-flankers condition, strong bottom-up input coupled with horizontal interactions results in a somewhat reduced dynamic range. The bottom-up and flanker inputs activate horizontal excitatory connections to all bipole cells in between. These recurrent excitatory horizontal interactions cause a larger activation of the target cell even at low contrast. In addition, inhibitory interneurons in the target hypercolumn receive bilateral horizontal inputs. As a result, the interneurons inhibit each other through recurrent connections, as in the flankers-only condition. As contrast increases, the activity of the excitatory recurrent interactions and the inhibitory interneurons increases until the inhibitory interneuron activities reach the self-normalizing range, thereby helping to limit the increasing firing rate of the target bipole.
Figure 4 Data from Kapadia et al. (2000) (dashed lines, reprinted with permission) and model simulations (solid lines). Error bars for the data represent variations among different cells/trials. Error bars for simulations represent variations among different parameter settings (see Appendix). The stimulus pattern in each case is indicated in the insets. See text for further details.

**Long-range modulation: Two sides**
At greater stimulus separations, contrast-dependent long-range modulation has been reported in pyramidal cells of area 17 (V1) in the cat (Polat et al., 1998). In that study, the activity of a target cell whose receptive field was stimulated with an optimally oriented bar was monitored in the absence or presence of flanking collinear stimuli. The stimulus bars were made long enough to cover the entire classical receptive field (CRF) of the recorded cell. In simulations, the bars span 3 spatial locations. At low stimulus contrast, flanking inputs increased the activity of the target cell located between them, relative to when flankers are absent (facilitation condition), whereas at high stimulus contrast, they resulted in depression of activity relative to the no-flanker case. In the experiment, the separation between the flankers was determined by successively widening the gap until no grouping was observed. In the simulations, this occurred when the gap was widened to the point that it spanned 7 spatial locations, whereas grouping with gaps of width 5 and 1 have been obtained in Figures 3 and 4, respectively. Figure 5 shows that the model simulates these data.
This result may be explained as follows: As in the target vs. target-with-flankers conditions of Figure 4, target bipole firing is higher at low current input in the target-with-flankers. As current input to the target site increases, the inhibition on the target cell body from flanking interneurons limits the combined influence of bottom-up and horizontal excitatory inputs from further increasing target bipole activity. Figure 5 shows that, as current input increases, this limiting influence eventually results in greater target bipole activity in the target-only condition than in the target-with-flankers condition. Such reversal of activity is not observed in the simulations of Figure 4, where current input is not sufficiently high (the maximum current input is 0.03nA in Figure 4 whereas it is 0.12nA in Figure 5).

**Long-range modulation: One side**
The bipole property can also enable one pyramidal cell to modulate the activity of a horizontally displaced cell, even though an individual pyramidal cell cannot cause significant suprathreshold grouping activity. Figure 6 reports simulation results and supporting evidence for this property from a cat V1 pyramidal cell (data from Crook et al., 2002). In the target-only condition, a single optimally oriented bar was presented in the CRF of the target cell. In simulations, this bar length spans a single spatial location. In the flanker-only condition, a single collinearly oriented bar was presented in a location adjacent to the CRF of the cell. In the target-with-flanker condition, the
target and flanker bars were presented simultaneously. The contrast of the flanker bar was 10 times higher than the contrast of the target bar. In simulations, current input to the target and flankers was 0.0096\(\text{nA}\) and 0.06\(\text{nA}\), respectively, in order to approach this ratio. Finally, in the \textit{GABA} condition, both the flanker and target bars were presented but with a simultaneous injection of GABA at the cortical activation site of the flanker stimulus.

As expected, cell activity was high in the \textit{target-only} condition. In the model, this was due to bottom-up activation of the target layer 2/3 pyramidal cell. Activity of the target pyramidal cell was decreased to near zero in the \textit{flanker-only} condition, reflecting the absence of outward completion from the flanker. In simulations, activity decreased to zero since, although flanker input to the target hypercolumn simultaneously excited the bipole cell and one inhibitory interneuron, the synaptic weights on the path leading to inhibition of the target bipole are stronger. Indeed, the maximal conductance \(g_{\text{max}}\) of layer 2/3 horizontal projections between model pyramidal cells is 0.003\(\mu\text{S}\), whereas the maximal conductances for the pyramidal-to-interneuron and interneuron-to-pyramidal projections are 0.037\(\mu\text{S}\) and 1.8\(\mu\text{S}\), respectively (see Table 3). In comparison, in the flankers-only condition of Figure 4, \textit{bilateral} excitatory input caused the interneurons to inhibit each other due to the recurrent connectivity, thereby leaving the bipole cell free to spike at a low level in that case.

Firing rates in Figure 6 nearly doubled in the \textit{target-with-flanker} condition, relative to the target-only condition. As in Figure 4, there is super-additive excitation in the target-with-flankers case. In Figure 6, this is due to recurrent horizontal excitatory connections between the single flanker and the target cell, after bottom-up excitatory input from layer 4 to the target bipole cell allows it to overcome inhibition from the flanker site and to emit spikes. If GABA inhibits the flankers, then the target cell response returns to the level in the target-only condition.

\textbf{Figure 6} Crook et al. (2002) neurophysiological data (empty bars) is matched with simulations (dark bars) in four conditions. Note the absence of outward propagation in the flanker-only condition. Error bars for the data represent the variation observed across different cells/trials, whereas they represent variations across different parameter settings for the simulations (see Appendix). [Data reprinted with permission from Crook et al. (2002).]
Horizontal summation

The salience and strength of perceptual groupings depend upon the amount of support present in the image inducers (Lesher and Mingolla, 1993; Soriano et al., 1996). Support is the ratio of the length of inducers to the total stimulus length. A correlate of this psychological observation is shown in physiological recordings of a layer 2/3 pyramidal cell in the tree shrew (Chisum et al., 2003). In this experiment, cell activity was monitored during presentation of stimulus bars of different lengths and at a predefined contrast level. Activation was reported as the ratio of firing activity obtained for the various stimulus lengths divided by the activity obtained for a wider bar of higher contrast. Figure 7 shows model bipole cell activity as a function of the length (in degrees of visual angle) of such stimulus presented at different contrast levels (the contrast levels tested correspond to current inputs of 0.0084, 0.0126 and 0.03nA) and overlaid on a subset of the Chisum et al. (2003) data. Horizontal summation is indicated by the fact that, as the array width gradually increases from one spatial location to seven, relative activity with respect to a long high contrast stimulus bar (13 spatial locations wide, current input of 0.048nA), tends toward 1. See the Appendix for details on how the degrees of visual angle from the experimental study were matched to the number of locations spanned in present simulations.

This result may be explained by noting that the target bipole cell receives excitatory input from a progressively larger number of flanking bipole cells as the stimulus bar gets wider. At low contrast, excitation from horizontal projections is slightly stronger than inhibition, which results in an increase in target bipole firing rate as a function of inducer length. However, at the highest level of contrast tested, the influence of inhibition becomes more pronounced, such that further increasing the inducer length does not increase the activity of the target bipole. Note in particular how the relative activity plotted for this contrast level approximates 1, meaning that it approaches the activity obtained in the case of the 13-units wide bar at even higher contrast. The model thus predicts that horizontal summation exerts a significant effect at lower contrasts but less so at high contrast. This is compatible with the simulations of short-range grouping of Figure 4 and long-range modulation of Figure 5, where the impact of inhibition on the target bipole cell is accentuated at similarly high contrast levels (i.e., for current inputs of 0.03nA), resulting in a reduction in analog sensitivity.

Figure 7 Activity of a layer 2/3 cell in area V1 of a tree shrew (continuous thick curve) is shown here as the ratio of activity (firing rate) obtained at a particular inducer length with respect to the activity in response to a longer stimulus at high contrast. In the simulations, this baseline stimulus spans 13 hypercolumns and corresponds to a current input of 0.0408 nA. Horizontal summation in the data is consistent with model simulations at lower current inputs (0.0084 and 0.0126 nA), whereas saturation is
observed at the high current input simulated (0.03 nA). Error bars in the data represent variations across different cells/trials. They represent variation across different parameters (see Appendix) in the simulations. [Data reprinted with permission from Chisum et al. (2003).]

**Gamma band oscillations, synchrony and perceptual grouping**

A number of investigators have reported synchronous oscillations during perceptual grouping, among other brain processes; e.g., Eckhorn et al. (1988) and Gray et al. (1989). Bipole grouping in rate-based models is capable of fast synchronization of boundary groupings, including illusory contours and fast resynchronization after inputs change. Synchronization occurs both in non-laminar models (Grossberg and Somers, 1991; Grossberg and Grunewald, 1997) and laminar cortical models (Yazdanbakhsh and Grossberg, 2004).

To study whether and how resynchronization occurs in the spiking laminar cortical sLAMINART model, simulations were conducted by presenting a random pattern of activation for the first 100ms, thereby randomizing the firing phases of layer 2/3 pyramidal cells, and then switching to either a real contour (Figure 8A) or an illusory contour (Figure 8B). The resulting layer 2/3 membrane potential traces were then low-pass filtered to preserve only burst oscillations. This is consistent with the observation that the oscillations found in the study of Gray et al. (1989) were mostly due to bursts, rather than to single-spike coincidence. Figure 8A shows resynchronization occurring in the 100ms following input change. Figure 8B shows resynchronization to occur around 300ms later. The faster synchronization observed for real contours is due to the fact that layer 4 activity, which drives the activity of layer 2/3, is synchronous along the simulated contour. This result is consistent with data showing that illusory contours take longer to be perceived than real contours (Meyer and Ming, 1988; Francis, Grossberg and Mingolla, 1994).

**Figure 8** (A) Fast resynchronization of bursts in the presence of a real contour. A random pattern was presented for the first 100ms and followed by a static real boundary contour stimulus. Oscillations shown here correspond to the middle 5 units along the contour, which was 9 units wide. (B) Slower resynchronization of bursts in the presence of an illusory contour. The gap between inducers was 3 units.

During synchronization, the bipole grouping network exhibits oscillatory synchrony in the low gamma range (>20Hz) along a grouped contour (Figure 9), a claim supported by a wide range of electrophysiological evidence (e.g. Gray et al., 1989; Samonds et al., 2006). This result is related to, but differs in an important way, from the binding-by-synchrony hypothesis (Milner, 1974; von der Malsburg, 1981). In the sLAMINART model, it is more proper to describe a synchrony-by-binding hypothesis, since synchronization, when it occurs at all in a bipole network, is an emergent property of how recurrent network interactions bind cells together during perceptual grouping. According to the experimental study of Gray et al. (1989), phase locking between a pair of pyramidal neurons is strongest in area 17 of the cat when a single contour spans the collinear CRF of the two recorded cells (Figure 9, upper left row). Synchrony is reduced at locations on an illusory contour (middle left row), and is non-existent during presentation of
uncorrelated moving bars (bottom left row). Simulations (middle column) agree qualitatively with the empirical cross-correlograms.

The single contour condition was simulated as a single inducer, 11 spatial locations wide. For the illusory contour condition, inducer bar and gap length were set to 3 and 5 spatial locations, respectively, as in Figure 3. The uncorrelated bars were simulated by alternately presenting each inducer from the illusory contour condition. Inducers were simulated by injecting 0.03nA current input. A phase randomizing signal was further included by injecting time-varying noise of amplitude up to 0.003nA in the dendrite of layer 2/3 pyramidal cells.

The exact nature of synchronous oscillations in the simulated spike trains is further probed in the right column, where points above the horizontal dashed line indicate significant coherence at a particular frequency (cf. Rosenberg et al., 1989). This analysis confirms the presence of low-range gamma oscillations (20-40Hz) along boundary groupings. Comparison of the single contour and illusory contour conditions reveals that phase-locked layer 4 activity transmitted via layer 2/3 bipoles located within a pair of nearby layer 2/3 bipoles helps to synchronize activity along the represented contour. This is to be expected from the symmetry of the bipoles: bipoles located in the middle project equally strongly to bipoles on the left and right of the contour (see Golubitsky and Stewart (2006) for a discussion of synchrony-inducing symmetry). The spectral analysis in the right column further shows that the shape of the frequency spectrum remains roughly similar across the two conditions. The main difference resides in the magnitude of the spectrum in each frequency bin, which again reflects the influence of excitation and inhibition from bipoles in the middle. In other words, both real and illusory contours produce oscillations mainly in the gamma range, but illusory contours display lesser power overall. However, oscillatory synchrony in this condition remains stronger than in the alternating contour condition, where horizontal excitatory signals from one side of the contour are insufficient to trigger reliable spiking in bipoles on the other side in the absence of concurrent bottom-up input.

Figure 9 (Left) Data of Gray et al. (1989) showing that synchrony between pyramidal cells in cat area 17 is strongest for a continuous bar stimulus, weaker at an illusory contour induced by a part of collinear bars, and absent between non-collinear bars. (Middle) Model simulations replicate these data. (Right) The synchronous coupling in the model is statistically significant at a confidence level of 5% in the full bar
and coherent bar cases and absent in the incoherent bar case, as indicated by the coherence index $|R|^2$. [Data reprinted with permission from Gray et al. (1989).]

The gamma oscillations depicted in Figure 9 remain when measured at a larger scale. In the experimental literature, gamma synchronization of evoked potentials has been found during viewing of grouping stimuli. In particular, Tallon et al. (1995) reported a 30Hz component over areas covering visual cortices in response to a Kanizsa triangle stimulus. In order to probe the synchronous dynamics of the network measured at a larger scale, cell activity across the 1D array of layer 2/3 cells obtained during simulations of an illusory contour (8 units wide flankers separated by a 5 units wide gap) was combined into a single estimate of the local field potential (LFP) according to the methodology described in Versace et al. (2008). The resulting signal was Fourier transformed (FFT) to obtain the power spectrum displayed in Figure 10. This result suggests that the large-scale gamma oscillations observed in response to grouping stimuli may either originate in the bipole circuit, or at least be partially supported by it.

![Figure 10](image)

**Figure 10** Local field potential (LFP) spectrum power in model layer 2/3. A clear peak is observed in the low gamma range. This result reflects the pattern of LFP spectrum power obtained using various stimulus configurations. See text for details.

**Discussion**

This article demonstrates for the first time how a network of layer 2/3 recurrently interacting spiking neurons with multiple compartments that obey Hodgkin-Huxley dynamics may perform stable, analog coherent, and synchronous perceptual grouping of both real and illusory contours in a manner that quantitatively reproduces key neurophysiological data from multiple laboratories. Whereas neural spikes can be implemented using relatively simple mechanisms (e.g., integrate-and-fire neurons in point neurons), the sLAMINART model is defined by more realistic assumptions such as Hodgkin-Huxley cell dynamics, the existence of multiple cellular compartments, the termination of excitatory and inhibitory recurrent interactions on cell dendrites and somata, and the existence of distance-dependent axonal delays. In addition, the selective grouping properties that exemplify the bipole property are realized without the use of explicit thresholds, since the latter are implicitly included in Hodgkin-Huxley dynamics for the combination of parameters used here.

The emergence of stable grouping despite the presence of distant-dependent axonal delays and the addition of noise in our simulations implies that perfect coincidence in spike timing is not necessary for grouping to occur. Rather, an instantaneous spike event exerts a temporally persistent influence on bipole cells principally due to the time course of EPSPs and
IPSPs (see equation (10)), and of inter-compartmental currents. These slower processes allow for
temporal summation of multiple pre-synaptic spike trains, thereby enabling the network
dynamics as a whole to exhibit properties like analog coherence and sensitivity to the extent of
stimulus support.

The differential localization of excitatory and inhibitory terminals on bipole cells, with
excitatory synapses on the dendrites and inhibitory synapses on the soma, means that inhibition
can act much faster on the soma than excitation does, thereby preventing the unwanted outward
propagation of signals outside of the inducers’ locations. This is further guaranteed by the larger
conductance of inhibitory synapses than excitatory synapses in the model.

The balance of inhibition and excitation is critical to explain the data on long-range
modulation (Figure 5). These data were also simulated in a previous non-spiking laminar cortical
model of perceptual grouping by Grossberg and Raizada (2000). That simulation required
modulatory feedback from V2 bipole cells to the V1 network. The current study shows how a
properly balanced network of spiking layer 2/3 cells within V1 is sufficient to generate this
result.

The simulations of fast synchronization highlight the relationship between boundary
grouping and gamma oscillations that has been reported in the neurophysiological literature.
Gamma oscillations have also been simulated during matching between bottom-up feature
patterns and top-down learned expectations during attentive category learning and recognition
(Grossberg and Versace, 2008). These two examples show that gamma synchronization can be
the result of quite different brain mechanisms. These results argue against a binding-by-
synchrony hypothesis. Rather, they illustrate how gamma synchronization may result due to
qualitatively different types of “binding”. Moreover, synchronization need not occur only in the
gamma frequency range, even in the same brain network. Grossberg and Versace (2008) have
predicted, for example, that slower beta oscillations can occur when a mismatch occurs between
bottom-up and top-down signal patterns, and at least three laboratories have reported data that
are consistent with this prediction (Buffalo et al., 2004; Berke et al., 2008; Buschman and Miller,
2008). Grossberg and Versace (2008) have predicted why more beta oscillations may be found in
the deeper layers of visual cortex, as reported by Buffalo et al. (2004), and Grossberg (2009) has
proposed an explanation of why beta oscillations occur in the hippocampal place cell learning
data of Berke et al. (2008).

**Comparison with other spiking models of grouping**
The fact that exact coincidence of spikes is not required in order to represent boundary contours
in the model (cf. Figure 8) illustrates that the grouping mechanism is robust. A related
mechanism of asynchronous spike-based contour completion – employing long-range anisotropic
excitatory connections but lacking inhibitory interneurons – was proposed in VanRullen et al.
(2001). Runaway excitation in the model was prevented by artificially limiting each neuron to
emit no more than one spike. Simulations showed that their network can account for cooperation
among neighboring collinear inducers (i.e., it can perform contour integration). However, the
authors did not test necessary computational properties such as the bipole property, stability of
the emergent grouping over time, analog sensitivity, and horizontal summation.

Yen et al. (1999) simulated a more elaborate network of compartmental neurons in which
a kernel of long-range anisotropic excitatory connections was used to promote synchronous
spiking among populations coding for collinear edges, with the intent of showing how spike
timing could be used as an index of which edge a given neuron represents. However, the stimuli
used in that study consisted only of real boundary contours. It is not clear whether the network
could perform boundary completion, notably illusory contour completion. In addition, the authors did not test for the presence of spurious outward propagation and did not address the requirement of analog coherence.

Yen and Finkel (1998) proposed a model of contour grouping based on synchronization of neural oscillators. However, suprathreshold activation of a cell required direct bottom-up input, implying that their model could not do boundary completion, notably formation of illusory contours. Moreover, in our model, gamma oscillations are an emergent property of cells which, left alone, do not oscillate.

Domijan et al. (2007) implemented a one-dimensional network of non-spiking compartmental bipole cells based on the original Boundary Contour System model of Grossberg et al. (1985a). The dendritic tree of bipole cells in the model was restricted to two branches: one that collects horizontal inputs from the left and the other one from the right. Their simulations replicated the bipole property, analog sensitivity, and horizontal summation. The crucial mechanism consisted in the explicit multiplication of the contribution of both dendritic branches (an AND-gate that preserves analog sensitivity). The model therefore embodies the following two assumptions: (1) the presence of multiplicative interactions between separate dendritic branches, and (2) the anisotropic distribution of postsynaptic sites on the dendrite tree, as dictated by the location of the presynaptic cell (i.e., pre-synaptic inputs from the left target the dendrite on the left, and vice-versa). The first assumption is contradicted by recent experimental findings that supralinear summation occurs only between synaptic sites on a single branch, whereas inputs from different branches sum only linearly (Polsky et al., 2004). The second assumption would be supported if the shape of dendritic trees were anisotropic. However, anatomical evidence suggests that dendritic trees, at least in layer 3 of visual cortices, are isotropic (Elston et al., 1996). Thus it remains to be shown how such an anisotropic distribution of synaptic inputs on isotropic dendritic trees could result from experience-induced development. This model does not incorporate spiking dynamics nor conduction delays, and does not strictly implement the cable equation (e.g., there are no bidirectional interactions between compartments and temporal integration is not present in the dendrites). In other words, the model does not address the problem of how neurons integrate asynchronous spikes to achieve perceptual grouping. Thus, this model does not incorporate several neurophysiological constraints that support sLAMINART quantitative simulations of key neurophysiological data.
Appendix

Model

First the mathematical equations for single cell dynamics are described, followed by network equations.

Hodgkin-Huxley dynamics and compartmental equations

Neurons are implemented as either one or two compartments governed by cable equations (Segev, 1998). Compartmental membrane potential $V$ is governed by an equation of the form:

$$C_m \frac{dV}{dt} = -\sum I_k,$$

where $I_k$ refers to either of synaptic, axial, injected or membrane channel currents, as explained below. Membrane capacitance $C_m$ is a function of compartment diameter ($d$) and length ($l$), and specific capacitance $C_M$:

$$C_m = \pi dl C_M.$$  \hspace{1cm} (2)

In accordance with general practice, we set $C_M=1\mu F/cm^2$ (Koch, 1999). Compartment dimensions $d$ and $l$ are the same for all neurons of a given layer. For clarity, $dV/dt$ is noted $dS/dt$ when the compartment is the soma, and by $dD/dt$ when the compartment is a dendrite.

Somatic compartments are governed in part by Hodgkin-Huxley (1952) equations. To simplify notation, the collective influence of the leak, K+, and Na+ currents is denoted $f(S^i_j)$, where $S^i_j$ stands for the somatic membrane potential of unit $i$ in layer $j$:

$$f(S^i_j) = -g_L(S^i_j + |E_L|) - g_K n^4(S^i_j + |E_K|) + g_{Na} m^3 h (E_{Na} - S^i_j),$$

where $g_L$, $g_K$ and $g_{Na}$ are the maximal conductances of the leak, K+, and Na+ channels respectively. $E_L$, $E_K$ and $E_{Na}$ represent the reverse potentials of the three respective currents, with specific values shown in Tables 3 and 4. The short form in (3) is not used for dendritic compartments since, for simplicity, the latter only have a passive leak current term $g_L (D_i + |E_L|)$. Gate variables $n$, $m$ and $h$ stand for the $K^+$ and $Na^+$ activating gates, and the $Na^+$ deactivating gate, respectively. The dynamical behavior of these gates is governed by the differential equation (where $k = \{n,m,h\}$):

$$\frac{dk}{dt} = \alpha_k(S^i_j)(1-k) - \beta_k(S^i_j)k.$$ \hspace{1cm} (4)

The rate constants $\alpha_k$ and $\beta_k$ for the $n$, $m$ and $h$ gating variables are given in equations (5)-(7), respectively. The parameters for these equations were adapted from Traub and Miles (1991) such that cells transition to spiking through a supercritical Andronov-Hopf bifurcation (Izhikevich, 2007) and have a stable attractor for small external input:

$$\alpha_n(V) = 0.032 \frac{15-V}{\exp(\frac{15-V}{5})-1}$$ \hspace{1cm} (5)
\begin{align*}
\beta_n(V) &= 0.5 \exp\left(\frac{-13.7 - V}{40}\right), \\
\alpha_m(V) &= 0.32 \frac{13.0 - V}{\exp\left(\frac{13 - V}{4}\right) - 1}, \\
\beta_m(V) &= -0.28 \frac{40 - V}{\exp\left(\frac{40 - V}{40 - V}\right) - 1},
\end{align*}

\text{and}

\begin{align*}
\alpha_h &= 0.128 \exp\left(\frac{17 - V}{18}\right), \\
\beta_h &= \frac{4}{\exp\left(\frac{40 - V}{5}\right) + 1}.
\end{align*}

Setting \( n(t) + h(t) \approx 0.84 \) and \( m = m_\infty(V) \), the \((V,n)\)-phase plane (Izhikevich, 2007) resulting from this choice of parameters is shown in Figure 11. It can be seen that, with these parameters, model neurons behave as threshold units, firing only for sufficiently depolarizing input.

\textbf{Figure 11} (A)-(C) Parameters used for Hodgkin-Huxley equations allow model neurons to lose stability through a supercritical Andronov-Hopf bifurcation with increasing current input strength \( I \). The presence of a stable attractor at low \( I \) ensures limited outward propagation of signals in the bipole network. (D) These settings also render model neurons analog sensitive in terms of firing rate (Hz).

For inter-compartmental currents, the \textit{actual} axial conductance of neurons within layer \( j \) is denoted \( q_j^f \) and is defined by:
\[
q_j = \frac{\pi d_j^2}{4l_jR_j^2}.
\]

Note that the diameter \(d_j\) and length \(l_j\) are the dimensions of the compartment \(c\) towards which the current appears to be directed in the relevant equation. Thus, \(c\) is replaced by \(S\) in the case of the soma and by \(D\) in the case of dendrite. Parameter \(R_j^a\) denotes specific axial resistance for neurons in layer \(j\).

Synaptic input \(I_{ij}^{kl}\) from unit \(i\) of layer \(k\) to unit \(j\) of layer \(l\) is modeled by:

\[
I_{ij}^{kl} = w_{ij}^{kl} \left( \sum_{t_n \in S_{ij}^{kl}} g_{ij}^{kl} (t - \delta_{ij}^{kl}, t_n) - \prod_{t_n \in S_{ij}^{kl}} g_{ij}^{kl} (t - \delta_{ij}^{kl}, t_n) \right),
\]

where \(\delta_{ij}^{kl}\) stands for axonal delay for that particular synaptic connection. Function \(g_{ij}^{kl} (t - \delta_{ij}^{kl}, t_n)\) is a double exponential (e.g. Köhn and Wörgötter, 1998):

\[
g_{ij}^{kl} (t - \delta_{ij}^{kl}, t_n) = \begin{cases} 
\frac{p}{\tau_f - \tau_r} & \text{if } \tau_r \neq \tau_f \\
\frac{t}{\tau_r} e^{-\frac{t-\delta_{ij}^{kl}-t_n}{\tau_r}} & \text{if } \tau_r = \tau_f 
\end{cases}.
\]

Constants \(\tau_r\) and \(\tau_f\) represent the rise-time and fall-time, respectively, of \(g_{ij}^{kl} (t - \delta_{ij}^{kl}, t_n)\), which closely determines excitatory and inhibitory postsynaptic potentials (EPSP/IPSP) shape and duration (see Table 3). Constant \(p\) is set to ensure that \(g_{ij}^{kl}(\cdot) \in [0, 1]\). The summation and multiplication in (9) are taken over the set \(S_{ij}^{kl}\) of the last two spike times \(t_n\) from unit \(i\) of layer \(k\) to unit \(j\) of layer \(l\). Thus, keeping in mind that \(g_{ij}^{kl}(\cdot) \in [0, 1]\), the multiplicative term ensures that the aggregated conductances remain between 0 and 1, which implies that synaptic current \(I_{ij}^{kl}\) varies between 0 and \(w_{ij}^{kl}\). In the case of one-to-one connections, such that \(i = j\), we abbreviate \(I_{ij}^{kl}\) by \(I_i^{kl}\) in equation (9).

The synaptic current resulting from the use of equation (9) is obtained by multiplying \(I_{ij}^{kl}\) with the voltage difference \((E - V_j^l)\). Here, \(V_j^l\) is the membrane voltage of the post-synaptic compartment of cell \(j\) in layer \(l\), and \(E\) is the driving potential specific to the type of synapse under consideration. For excitatory connections, \(E\) is set to a depolarizing value (0 mV). For inhibitory connections, \(E\) is set to a hyperpolarizing value (-60 or -70 mV).

**Network equations**

The neural network is composed of 1-dimensional arrays of layer 4 and layer 2/3 pyramidal cells and *Left* and *Right* interneurons. Each of the four layers contains 51 neurons. Figure 2 illustrates a representative diagram of the network where layer 2/3 cells and interneurons span 7 spatial locations. External input is provided at each layer 4 pyramidal cell. Each layer 4 cell projects to a single layer 2/3 cell. Horizontal excitatory connections originating from layer 2/3 cell span a total of 7 spatial locations. *Left* interneurons receive such horizontal connections only from layer
2/3 cells on their left. The reverse holds for Right interneurons. Left and Right interneurons at a given spatial location inhibit each other via recurrent connections. Both interneurons also inhibit the layer 2/3 cell at the same spatial location. These connections are described more precisely below.

All network equations are written with endogenous currents (ionic channels, inter-compartmental) on the left-hand side and exogenous currents (synaptic, injections) on the right-hand side.

**Layer 4 pyramidal cells**

Each layer 4 pyramidal cell is modeled as a single compartment (a soma) $S^4_i$ that receives externally injected input $X_i$, where the latter is a scalar value for each neuron that is determined according to the simulation (see below):

$$C_m \frac{dS^4_i}{dt} - f(S^4_i) = X_i.$$  \hspace{1cm} (11)

**Layer 2/3 pyramidal cells**

Each layer 2/3 pyramidal cell is composed of one soma ($S^2_i$) and one dendrite ($D^2_i$) compartment. The dendritic compartment receives bottom-up excitatory input from one layer 4 pyramidal cell ($I^{42}_i$) and recurrent excitatory input from a Gaussian neighborhood of layer 2/3 pyramidal cells ($\sum_{m=-3}^{i+3} I^{22}_{mi}$):

$$C_m \frac{dD^2_i}{dt} - q^D_2 (S^2_i - D^2_i) + g_L (D^2_i + |E_L|) = (E_{AMPA} - D^2_i) I^{42}_i + (E_{AMPA} - D^2_i) \sum_{m=-3}^{i+3} I^{22}_{mi}. \hspace{1cm} (12)$$

The Gaussian distributed weight kernel and the axonal delay kernel implicit in $I^{22}_{mi}$ are determined by equations (13) and (14), respectively:

$$v^{kl}_{mi} = g_{max} e^{(m-i)^2/\sigma^2}, \hspace{1cm} (13)$$

$$\delta^{kl}_{mi} = 1 + 3 \cdot |i-m|, \hspace{1cm} (14)$$

where $m$ and $i$ are the indices of the pre- and post- synaptic units respectively, and $\sigma=4.47$. The maximal conductance, $g_{max}$, is specific to the projection and values used for it are in Table 3.

The somatic potential is defined as follows: Input from layer 2/3 pyramidal cells is significantly delayed in time, reflecting the presence of slow conduction delays in layer 2/3 horizontal connections. The soma receives convergent inhibitory input from the Left and Right interneurons at the same spatial location ($I^{L2}_i + I^{R2}_i$):

$$C_m \frac{dS^2_i}{dt} - q^S_2 (D^2_i - S^2_i) - f(S^2_i) = -(S^2_i + |E_{GABA}|) [I^{L2}_i + I^{R2}_i]. \hspace{1cm} (15)$$

Equation (15) implies that inhibitory interneurons have a decisive effect on the layer 2/3 pyramidal cell they innervate due to their direct action on the soma.
Layer 2/3 inhibitory interneurons

Layer 2/3 interneurons are divided into two groups according to whether they are to the Left or to the Right of the layer 2/3 pyramidal cell they innervate. Since the form of the differential equations is similar and all parameters are the same, only equations for Left interneurons are explicitly given here. Each Left layer 2/3 interneuron is composed of one soma \( S_i^L \) and one dendrite \( D_i^L \) compartment. The dendritic compartment receives excitatory input from a half-Gaussian neighborhood of Layer 2/3 pyramidal cells located to its left:

\[
\sum_{m=i-3}^{i-1} f_{mi}^{2L} \]

The synaptic weight and axonal delay parameters that define \( I_{mi}^{2L} \) are determined by equations (13) and (14), respectively, with the additional constraint that it is set to 0 for \( m>i \) in the case of Left interneurons and for \( m<i \) in the case of Right interneurons.

Equation (17) governs the somatic membrane potential. The soma of a Left inhibitory interneuron only receives inhibitory input originating from the Right interneuron at the same spatial location:

\[
C_m \frac{dS_i^L}{dt} - q_L^D (S_i^L - D_i^L) + g_L (D_i^L + |E_L|) = (E_{AMP} - D_i^L) \sum_{m=i-3}^{i-1} f_{mi}^{2L}.
\]  (16)

Local field potential (LFP) calculations

Local field potentials are calculated in the KinNeSS software package according to the methodology described in Versace et al. (2008). Emplacement of the electrode is determined by choosing a random location within \([10-200]\)\(\mu\)m of the middle spatial location of the array of pyramidal layer 2/3 cells and aligning the electrode shank with the orientation of the cells. The distance of that electrode to the remaining 50 cells in the layer was uniformly random within the \([10-1000]\)\(\mu\)m interval. The electrode is composed of five equally spaced electrode tips covering the entire length of layer 2/3 cells (i.e., sum of the dendrite and soma lengths). The LFP output did not differ significantly across electrode tips, so the respective outputs were averaged together in order to get a global estimate. The Fast Fourier Transform (FFT) was calculated on the averaged LFP thereby obtained.

Synchronization measure

The synchronization measure described in Rosenberg et al. (1989) was used to quantify the significance of the coupling in a selected pair of layer 2/3 pyramidal cells in Figure 9 (right column). Each spike train is divided into \( L \) segments of length \( T \). Let \( \tau_j \) represent spikes times, then the finite Fourier transform of the \( i^{th} \) segment at frequency \( \omega \) is given by:

\[
d^T_i(\omega,l) = \sum_{(l-1)T < \tau_j < lT} e^{-i\omega \tau_j}.
\]  (18)

The cross-spectrum between two spike trains (denoted \( a \) and \( b \)) is further given by:

\[
\hat{f}_{ab}(\omega) = \frac{1}{2\pi LT} \sum_{l=1}^{L} d^T_a(\omega,l) \overline{d^T_b(\omega,l)}.
\]  (19)
where the bar indicates the complex conjugate. The squared magnitude of the estimated coherency between the two processes is defined as:

\[
| R_{ab}(\omega) |^2 = \left| \frac{\hat{f}_{ab}(\omega)}{\hat{f}_{aa}(\omega) \hat{f}_{bb}(\omega)} \right|^2. \tag{20}
\]

An upper 95% confidence limit to test for the presence of synchrony is given by \(1 - (0.05)^{1/(L-1)}\). This limit is plotted as a horizontal dashed line in Figure 9. Values above the line indicate significant coupling in the frequency range indicated on the x-axis. Here \(L=50\) and simulations were run for 25000ms, such that \(T=500\)ms, yielding a frequency resolution of 2Hz.

**Parameters**

**Network**

Biophysical parameters for synaptic connections and cells are given in Tables 3 and 4.

<table>
<thead>
<tr>
<th>Population</th>
<th>Compartments</th>
<th>Diameter [mm]</th>
<th>Length [mm]</th>
<th>(g_{\text{leak}}) [mS/cm²]</th>
<th>(E_{\text{leak}}) [mV]</th>
<th>Axial resistance [K·Ω·cm]</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 pyr.</td>
<td>soma</td>
<td>0.001</td>
<td>0.005</td>
<td>0.01</td>
<td>-60</td>
<td>--</td>
</tr>
<tr>
<td>2/3 pyr.</td>
<td>soma</td>
<td>0.001</td>
<td>0.012</td>
<td>0.001</td>
<td>-60</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>dendrite</td>
<td>0.001</td>
<td>0.032</td>
<td>0.005</td>
<td>-60</td>
<td>10</td>
</tr>
<tr>
<td>2/3 int.</td>
<td>soma</td>
<td>0.001</td>
<td>0.01</td>
<td>0.01</td>
<td>-60</td>
<td>--</td>
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</tr>
</tbody>
</table>

Table 4: Cell compartment dimensions, passive leak and axial conductivity parameters

**Weights and conduction delays**

Horizontal weight kernels have a Gaussian shape as in equation (13) (half-Gaussian for connections reaching interneurons). The extent of the kernels is designed by dividing the maximum extent of horizontal connections by the width of a V1 hypercolumn (Yazdanbakhsh and Grossberg, 2004). Assuming a 7mm wide kernel and a 1mm wide hypercolumn, the kernel size is set to 7 spatial locations.

Horizontal axonal conduction delay kernels are linearly dependent on distance, as in equation (14). The delay between neighboring hypercolumns is calculated by dividing the hypercolumn width by horizontal conduction speed. Recent estimates of horizontal conduction speed in both monkey V1 and cat area 17 put it at approximately 0.3m/s (Hirsch et al., 1991; Bringuier et al., 1999; Girard et al., 2001). Using a hypercolumn width of 1mm, the conduction delay between neighboring hypercolumns is set to 3ms; see equation (14).

**Simulation protocol**

All simulations were performed with KinNeSS (Versace et al., 2008). Unless mentioned otherwise, firing rates are measured once the network reaches a steady state, by counting the number of spikes in the last second of simulation. An integration time-step of 0.05ms, 0.02ms, or 0.01ms was used to obtain numerically accurate results.
The results displayed in Figures 4, 6 and 7 represent averages (and standard deviation for error bars) from nine different parameter settings where the maximal conductance of AMPA connections were varied along two dimensions. Specifically, maximal conductance of the layer 4-2/3 connection was varied within the set \{0.048, 0.049, 0.05\} mS/cm\(^2\) and maximal conductance of the layer 2/3-interneuron connection was varied within the set \{0.036, 0.037, 0.038\} mS/cm\(^2\). Results appeared qualitatively similar in all cases. The simulations in Figure 5 correspond to the \textit{middle} parameter configuration (i.e., values of 0.049 and 0.037 for respective parameters).

In simulations where this is relevant, stimulus contrast is defined as \(X_i/0.0006\), where \(X_i\) is the current injected into layer 4 pyramidal cells. Thus, nonlinearities between stimulus contrast and current input to cortical cells are not included in the simulations, implying that the simulations of contrast-dependent data may arise totally due to properties of the bipole network.

**Simulation of bipole property (Figure 3)**
Nonzero input was applied to units at spatial locations 21, 22, 23, 29, 30 and 31 in the ID input array. Input strength at those locations was \(X_i = 0.03\) nA. The simulation was conducted for 2000ms and firing rates were calculated by monitoring spikes in the last 500ms in order to ensure that the network has settled into a stable firing mode.

**Short-range completion simulations (Figure 4)**
Each stimulus bar is represented as an input \((X_i)\) to a single location in Layer 4. This is motivated by considerations of recent estimates of the cortical magnification factor (cmf) at 4° of eccentricity (Polimeni et al., 2006). Accordingly, a cmf of 2.7mm/° gives approximately 1mm of cortical extent to a 30’ stimulus bar as was used in the original study of Kapadia et al. (2000). However, 1-2mm is the approximate diameter of one hypercolumn. Thus, a single stimulus bar is presented to a single location. This is also consistent with their adjustment of the length of the bars to the size of the CRFs. Stimulus contrasts of 20, 30 and 50 percent were simulated by adjusting input strength to 0.012, 0.018 and 0.03 nA, respectively.

**Simulations of long-range modulation (Figure 5)**
The size of the bar stimuli in Polat et al. (1998) match CRF size which is here mapped to 3 adjacent columns. The distance between flanking bars is set to 7 locations, which is the shortest distance for which no inward completion occurred for the set of parameters considered. This particular constraint is in accordance with the method used in the original paper of Polat et al. (1998), and serves the purpose of studying long-range modulation instead of short-range completion. The current inputs simulated were (in nA): 0.0036, 0.006, 0.0072, 0.0126, 0.021, 0.03, 0.06, 0.09 and 0.12.

**Outward propagation simulations (Figure 6)**
In the original study of Crook et al. (2002), each stimulus bar was simulated as input to a single location. In the original study, the separation between the target cell recorded and the cell whose CRF receives the flanking stimulus was \(~2\)mm. This corresponds to the approximate size of the cat’s hypercolumn width (Lund, 2003). Thus, in the simulations the target and flanking bars were presented at adjacent locations. The target input strength was set to 0.0096 nA and that of the flanker line was set to 0.06 nA in order to approach the 1-to-10 contrast ratio in the original study.
**Horizontal summation simulations (Figure 7)**

Arrays of collinear Gabor patches are represented by low contrast stimuli in the simulations. This is meant to represent the fact that the patches used in this study are of smaller diameter than the measured CRF size, such that they do not produce maximal activation. The length of stimuli used is determined by using the cmf (21 mm/°) reported by the authors multiplied by the length (in degrees) of the original stimuli (here 5, 10, 15, 18, 23, 28 and 33 degrees). The corresponding array lengths are: 1, 2, 3, 4, 5, 6 and 7 mm, where each mm corresponds to one location in our simulations, which is in the order of the size of a hypercolumn in the tree shrew (Bosking, 1997). For completeness, input stimuli are simulated at three contrast magnitudes by setting input strength to values of 0.0084, 0.0126 or 0.03 nA. The firing rate obtained is divided by the firing rate obtained for a 13-units long bar of high contrast (input strength set to 0.048 nA) simulated for the central parameter configuration in order to report quantities as relative activation with respect to a continuous bar of high contrast, consistent with the original study of Chisum et al. (2003).

**Fast resynchronization simulations (Figure 8)**

The simulations of Figure 8A and 8B were constructed by inserting a fixed random frame with input values ranging between 0 and 0.018 nA across spatial locations for the initial 100 ms and then switching to a homogeneous input stimulus for the remaining 900 ms. In the case of the full contour simulation (Figure 8A), homogeneous input of magnitude 0.03 nA was applied to units 22 to 30. In the case of the illusory contour simulation (Figure 8B), the same homogeneous input was applied to units 22, 23, 24, 28, 29 and 30. Membrane potential traces of layer 2/3 bipoles were low-pass filtered with a Butterworth filter of order 4 to remove single spikes but preserve slow oscillations, which simplifies detection of phase synchrony. Visual inspection of traces revealed that bursts occurred during ups and silent periods during troughs of the resulting filtered signals. The oscillations displayed therefore reliably represent burst occurrences.

**Oscillatory synchrony simulations (Figure 9)**

Simulations were run for 25000 ms. The full bar consisted of a 9-units wide stimulus. Separate bars consisted of 3-units wide stimuli, separated by a 3-units wide gap. The pair of cells selected for recording had their CRF located in the middle of each bar and were thus separated by 5 hypercolumns. This reflects the arrangement in the experimental recordings by Gray et al. (1989) where the pair of cortical cells was separated by approximately 7mm. Input strength was set to 0.03 nA. For the full bar and coherent bars condition, stimuli were presented with simultaneous injection in the layer 2/3 dendrites of 10 ms sub-threshold white noise frames of amplitude varying in the interval [0 0.003] nA. For the incoherent bar condition, each short bar was presented for a randomly determined period of 300-500 ms. Each bar presentation was followed by a noise-only period of 300-500 ms, whose purpose was to attenuate the periodicity artificially induced in the delta band by the slow alternation of stimulus bars. Note that, exclusion of these periods from the simulation did not change the synchronization profile of Figure 9 (right) in the frequency bands of interest (mostly beta and gamma). Furthermore, the cross-correlograms reported in the middle column were shifted 100 ms in time to magnify the signal strength for the incoherent bar condition. Indeed, without this shift, the cross-correlogram output remains at 0 in the time frame considered for that condition, due to the inclusion of noise-only frames. However, the pattern of result remains the same when removing the shift. The coherence index $|R|^2$ was calculated according to equations (18)-(20).
LFP spectrum simulations (Figure 10)
Simulations were run for 25000 ms with 10 ms noise frames. Static input was set to 0.012 nA and time-varying noise magnitude spanned the interval [0 0.006] nA, such that the signal-to-noise ratio varied from 0 to 50%. The stimulus pattern simulated – i.e. a long bar, short bar, flankers-only bar, etc. – did not affect significantly the LFP power spectrum significantly, varying between the upper 30 Hz to about 50 Hz. In the particular simulation shown in Figure 10, the stimulus pattern consisted of two 8 units wide flankers separated by a 5 units wide gap, and noise magnitude was set to 0 (no noise condition).

Threshold and analog sensitive single-cells (Figure 11)
The plots A, B and C were generated from equations (5)-(7) using the phase plane technique described in Izhikevich (2007), and for three representative current input levels (no current, low current, large depolarizing current). Plot D was generated by measuring the firing rate of a single neuron as the current input to that neuron was increased from 0 nA.
References


Buschman T.J. and Miller E.K. 2008. Covert shifts in attention by frontal eye fields are synchronized to population oscillations. Submitted for publication.


Fang, L. and Grossberg, S. 2009. From stereogram to surface: How the brain sees the world in depth. Spat Vis, 22: 45-82.


