AN EMERGENT MODEL OF VISUAL CORTICAL ORIENTATION SELECTIVITY

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ABSTRACT

We demonstrate a cortical circuit based on known anatomical details and cellular properties which achieves sharp orientation tuning despite poorly tuned thalamocortical input. Sharp tuning arises emergently from excitatory interactions between similarly tuned neurons, which also receive broadly tuned inhibition. This model accounts for data obtained from intracellular recording and pharmacological blockade studies, which had previously appeared to conflict over the role of inhibition. We suggest that inhibition acts non-specifically and indirectly to maintain the selectivity of individual neurons by balancing strong intracortical excitation at the columnar level.

INTRODUCTION

The means by which visual cortical neurons obtain their orientation selectivity was first addressed by Hubel & Wiesel [1] who proposed that LGN cells covering an oriented region of visual space might provide converging input onto single cortical simple cells. However, simple cell subfields appear to have insufficient length to width ratios to account for the sharp tuning observed physiologically[2, 3] (Full width at half-amplitude $\leq 40°$ [4]). In addition, anatomical studies indicate that cortical neurons receive only a small percentage (5-20%) of synapses from LGN neurons, while they receive 50-70% of their synapses from cortical excitatory neurons and 10-25% from cortical inhibitory neurons[5, 6, 7].

Others have suggested that intracortical inhibition plays the dominant role in orientation selectivity, perhaps by suppressing responses to roughly orthogonal or "cross" orientations[8]. This is supported by experiments in which pharmacological blockade of inhibition over a small region of cortex disrupted orientation selectivity[9]. However, intracellular recordings suggest that cross-orientation inhibition is weak at best[10, 11]. Furthermore, our laboratory has recently reported that intracellular blockade of inhibition in single cortical neurons has a negligible effect on orientation selectivity of those neurons[13]. Here we present a model which reconciles these apparently conflicting data, by utilizing recurrent, iso–orientation cortical excitation as the dominant source of orientation selective responses.

METHODS

Our model circuit consists of three layers: an image/retina layer, a retinotopic patch of LGN, and a small patch of layer IV of visual cortex. Image/retina components provide feedforward inputs to LGN cells. LGN neurons, in turn, provide feedforward inputs to both excitatory and inhibitory cortical neurons. To create an initial orientation bias to cortical cells, thalamocortical connections were chosen according to a weak form of the Hubel-Wiesel model[1]. LGN inputs to a *cortical column* were chosen randomly from a 3 by 1 oriented region of visual space; however, inputs to *individual cortical cells* varied substantially (1:1 – 3:1, mean 2:1) and were hence either weakly oriented or unoriented. This thalamocortical wiring scheme is consistent with the report [12] that the LGN afferents to a cortical column occupy a roughly 3:1 oriented region of visual space and with reports that subfield aspect ratios vary greatly among simple cells[2, 3]. Cortical neurons also received input from both cortical inhibitory and cortical excitatory neurons. Both sets of inputs came most densely from the same or nearby orientation columns with the inhibition from a somewhat broader set of orientations.

Cortical neuron representations: Cortical neurons have been implemented as "improved integrate–and–fire" neurons (e.g.,[14]) with time–varying firing thresholds. Each cell is modeled as a single compartment whose membrane potential, V_i is given by:

$$\begin{aligned} C_m \frac{dV_i(t)}{dt} &= \sum_{j=1}^{k} g_{ji}(t-t_{ji})(V_i(t)-E_{ex}) + \sum_{j=k+1}^{k+l} g_{ji}(t-t_{ji})(V_i(t)-E_{inh}) \\ &\quad + g_{leak}(V_i(t)-E_{leak}) + g_{AHP}(t-t_{spike})(V_i(t)-E_{AHP}). \end{aligned}$$

where the time–dependent conductances are given by:

$$g_{ji}(t) = w_{ji} \sum_{p}^{S_j} (t-t_p) e^{-\frac{t-t_p}{t_{peak}}}.$$

E_{ex}, E_{inh}, E_{leak}, and E_{AHP} correspond to the reversal potentials for Na^+, Cl^-, Cl^-, K^+, respectively. t_{ji} and w_{ji} are the synaptic delay and synaptic strength,

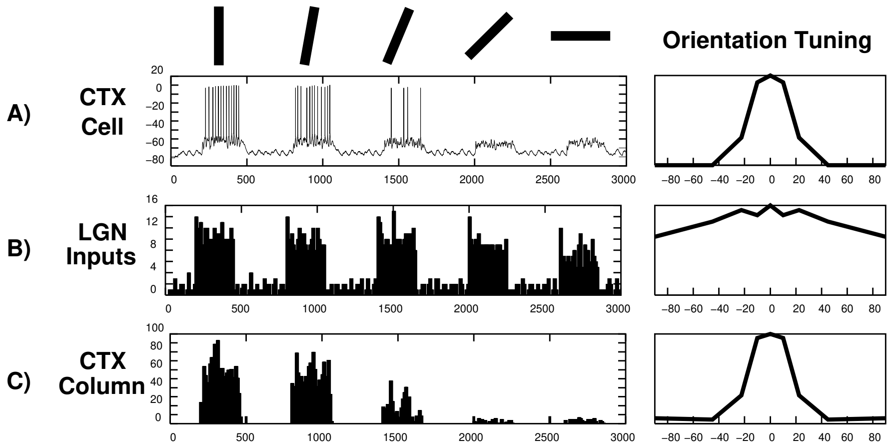

Figure 1 Orientation selectivity in the model. A) Simulated intracellular response of a cortical neuron to flashed bars oriented at 0, 10, 22.5, 45, and 90 degrees. This cell exhibits sharp orientation tuning (full-width at half-amplitude $< 40°$) despite receiving poorly tuned LGN inputs (shown as spike histogram in B). The tuning of this cell is typical of cortical neurons within this orientation column (shown in C).

respectively, between cell j and cell i. t_p is the time of each recent spike (described by the set S_j) of pre–synaptic cell j. When the membrane potential reaches threshold a spike is recorded for cell i and g_{ahp} is activated. Excitatory and inhibitory cortical neurons were modeled separately using experimentally reported input resistances, membrane time constants, and firing characteristics of regular–spiking (RS) and fast–spiking (FS) neurons [15]. LGN neurons were modeled as Poisson processes with center-surround receptive fields and parameters chosen to approximate physiological responses to flashed stimuli[14]. Computer simulations were run on the CM-5 Connection Machine using the SPIKETIME simulation package (Somers, unpublished). Model networks included up to 4000 neurons and 300,000 synapses.

RESULTS

The LGN inputs (with no intracortical synapses) produced broad tuning (mean 120° full–width) which varied substantially across cells in a column. Our model utilized inhibitory inputs primarily from nearby orientation columns, and hence produced IPSPs which were strongest at the preferred orientation [10, 11]. Iso–orientation inhibition (with no cortical excitation) enhanced selectivity, but only at the cost of decreasing response amplitudes ("iceberg" effect) [14].

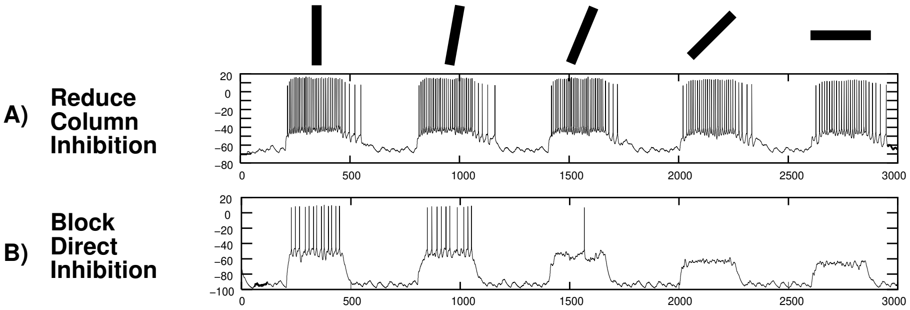

Figure 2 Inhibitory blockade simulations. A) Iontophoresis of the GABA A antagonist, bicuculline disrupts orientation tuning. With reduced columnar inhibition, all orientations yield robust responses. B) Intracellular blockade of direct inhibitory inputs has little effect on orientation selectivity.

The combination of intracortical excitation and inhibition produced both *robust responses* and *sharp orientation tuning*[16, 17]. This is demonstrated in Figure 1. Cortical connection strengths were usually chosen so that the response amplitude to the preferred orientation was equal to that for thalamocortical connections alone. Robust orientation tuning ($40°$ full-width at half-amplitude) was observed over a wide range of network parameters provided that there was an an approximate balance of excitation and inhibition and that inhibition was more broadly tuned than cortical excitation. Intracortical excitation generated a similar degree of orientation selectivity in all cells in the column, roughly independent of their direct thalamocortical connections (in agreement with [3]). Even "unoriented" cells (full-width $> 180°$), whose feedforward–only response to the orthogonal orientation did not fall below half of the preferred orientation response, exhibited 40 degree full–width tuning under the feedback conditions. Thus intracortical excitation integrates and amplifies the oriented pattern distributed across the column. This significantly reduces the degree of specificity in the thalamocortical wiring necessary to achieve a given degree of orientation selectivity.

In order to investigate further the role of cortical inhibition in orientation selectivity, we simulated two key physiological experiments by manipulation of inhibitory connection strengths. These simulations (see Figure 2) show the same model cell in the same network for which sharp tuning was exhibited in Figure 1. Sillito [9] demonstrated that orientation selectivity could be disrupted by blocking GABA A inhibition over a small region of cortex (by iontophoresis of bicuculline). To simulate these inhibitory blockade experiments, we reduced g_{inh} by 80% for all cells in an orientation column. Consistent with [9] this greatly disrupted orientation selectivity. The reduction of inhibition within the column permitted recurrent cortical excitation to generate strong responses to all orientations. Response levels were high, but were not saturated; cells could fire twice as fast in response to strong injected current.

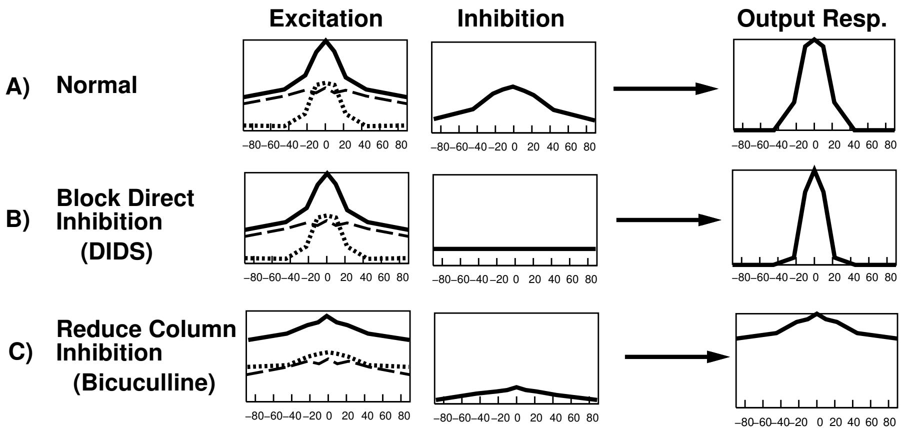

Figure 3 Summary of model function. A) Sharply tuned cortical excitatory inputs (dotted line) combine with poorly tuned LGN inputs (dashed line) to yield well oriented excitatory input (solid line). Cortical inhibitory inputs (middle) are strongest at the preferred orientation and thus do not simply act to "sculpt out" a well oriented receptive field. B) Blockade of the direct inhibitory inputs to this cell has only a mild effect on tuning for which a fixed (orientation non–specific) hyperpolarizing current can compensate. C) However, inhibitory neurons have a more important *distributed* effect on orientation selectivity. When the inhibitory inputs to all neurons of a cortical column are partially blocked, cortical excitatory inputs (dotted line) lose their sharp tuning and the circuit non-selectively amplifies stimulus responses.

In contrast to the effects of blocking cortical inhibition in *a cortical column*, blocking the inhibitory (100%) and after-hyperpolarizing (80%) currents in *a single cell* had little effect on selectivity, as observed experimentally [13]. EPSP tuning was unaffected and was sufficiently sharp to produce well oriented responses in the absence of inhibition. Injection of hyperpolarizing current into a blocked cell (as done by [13]) to reduce the maximal response to pre-blockade levels yielded selectivity matching that observed prior to blockade. The dramatic difference in the effects of the two types of inhibitory blockade reflects the difference in the tuning of cortical excitatory inputs in the two cases (see Figure 3). Blockade of inhibition in a single cell had a negligible effect on the tuning of other neurons, thus cortical EPSPs remain sharply tuned. However, when inhibitory inputs to a population of cells were reduced, cortical EPSPs lost their sharp tuning and further contributed to the disruption of tuning by pushing cell responses into their higher and more compressing output ranges.

SUMMARY AND CONCLUSIONS

These simulations suggest that recurrent, iso-orientation cortical excitation is the dominant source of robust, sharply oriented responses. In the absence of direct inhibition to single cells, well tuned cortical EPSPs are sufficient to generate sharply tuned responses. The well tuned cortical excitation reflects selective amplification of responses to preferred stimulus orientations and is an emergent property of the recurrent cortical circuitry. We call these sharply tuned cortical responses *emergent* since they are both cause and effect, and they are not generated by afferent inputs alone. Intracortical inhibition plays a critical, but distributed role in regulating the gain of cortical amplification. The pattern of inhibition is less important since either iso–orientation inhibition or unoriented hyperpolarizing input is sufficient to permit sharp tuning. This argues against a critical role for strong cross–orientation inhibition, and suggests a reconciliation of apparently contradictory data from extracellular inhibitory blockade studies [9] and intracellular studies [10, 11, 13].

In summary, all 3 types of synaptic input in our model contribute essential components to the generation of orientation selectivity. LGN inputs provide the initial orientation bias to a column. This is consistent with a weak, distributed form of the Hubel & Wiesel model [1, 12]. Inhibitory inputs to a cortical neuron sharpen its selectivity directly, but non–specifically. Even more importantly, however, inhibition acts indirectly by keeping recurrent excitation sharply tuned. Short-range cortical excitation amplifies responses and integrates the pattern of LGN input to a column. This columnar integration reduces the degree of specificity in afferent connections required to generate highly selective responses. The combination of cortical excitation and inhibition achieves both sharp selectivity and strong response levels.

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