The Caltech Computer Cat: a fine-grain simulator of the primary visual system of cat

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Abstract

Visual cortex shows a high degree of functional specificity, which is not based on a strict anatomical structure of this part of the brain: intracortical connections seem to be arranged rather uniformly. We use a computer simulation to study how this apparent contradiction between low structural and high functional specificity can be resolved for one known functional property of primary visual cortex, their selectivity for the orientation of bars presented within their receptive fields. It is found that long-range lateral inhibition (a uniform intracortical connection scheme) does lead to sharpening of orientation tuning. Furthermore, a strong directional bias emerges. Such anisotropic behavior is an inherent feature of the columnar organization of orientation preferences and is not introduced by noise induced symmetry breaking. Since already isotropic connections lead to a directional bias it is likely that a more specialized convergence pattern will also influence both orientation and directional tuning simultaneously. This suggests that the mechanisms underlying direction and orientation specificity are not independent features [1]. We believe that fine-grain simulations, like the one we use, will prove to be valuable tools in the study of neural systems.

1 Introduction

Over the last years, a realistical computer model of a part of the primary visual system of the cat has been developed. The cat was chosen in view of the considerable amount of anatomical and physiological data which are available for this animal in the literature. A monocular patch of about 5° by 5° of the primary visual pathway was simulated at a retinal excentricity of about 4.5°. Because the dominant input to area 17 in cat comprises X geniculate cells, we chose to simulate only ON and OFF retinal ganglion cells of the X type, corresponding to the anatomical β ganglion cell class. The projection of these cells was traced to the LGN and finally to the corresponding population of neurons in layer IV of the visual cortex (see Fig. 1). The model contains more than 16,000 cells and 4,000,000 synapses. Originally developed for UNIX machines, the simulator was recently implemented also on the massively parallel Connection Machine CM-2.

2 Single cell Model

A detailed description of the model has been given elsewhere [2, 3] and will not be repeated here. We find it nevertheless interesting to show in some detail the model for the building blocks of the simulator, the single neurons. Each cell is modeled as a single passive compartment corresponding to the cell body, a capacitance C_m in parallel with a membrane leak conductance, g_{leak}, and a leak battery, E_{leak}. Every synapse is described by a synaptic reversal potential (E_{ex} or E_{inh} for excitatory and inhibitory synapses, respectively) in series with a transient conductance increase $g_{ex}(t)$ and $g_{inh}(t)$. The time-course of these induced conductance changes follows an α function, i.e. $g(t) = const \cdot te^{-t/t_{peak}}$, with $t_{peak} = 1\ msec$. All synaptic input is added in parallel. The generation of action potentials is mimicked by a simple threshold; that is, at the time t_{spike} where $V(t) > V_{Thres}$, an action potential is assumed to be generated and relayed with an appropriate delay to all postsynaptic cells. The voltage threshold, V_{Thres}, varies from cell to cell and is uniformly distributed between -45 and $-35\ mV$. An absolute and relative refractory period is modeled by a transient potassium conductance $g_{AHP}(t)$, in series with a potassium reversal battery, $E_{AHP} = -90\ mV$. Thus, each time an action potential is generated, $g_{AHP}(t)$ becomes activated, leading to an afterhyperpolarization and preventing the cell from firing for a time dependent on t_{peak} of the associated α function. The equation describing the evolution of the state of every neuron is given by

$$\begin{aligned} C\frac{dV_i(t)}{dt} &= \sum_{i=1}^{k} g_{ex}(t-t_i)(V_m(t)-E_{ex}) \\ &\quad + \sum_{i=1}^{l} g_{inh}(t-t_i)(V_m(t)-E_{inh}) + g_{leak}(V_m(t)-E_{leak}) \\ &\quad + g_{AHP}(t-t_{spike})(V_m(t)-E_{AHP}), \end{aligned} \tag{1}$$

where k and l are the total numbers of excitatory and inhibitory synapses, respectively. Action potentials arrive at times t_i at the respective synapses and t_{spike} is the time the cell generated the last action potential before t. A propagation delay (which includes synaptic delay) is incorpoated into the times of arrival of action potentials, depending on the locations of pre- and postsynaptic cells. We refer the reader to ref. [3] for the numerical values of the parameters (see Table 1 there) in eq. 1. The temporal dynamics of our single cell model is obviously not very realis-

tic, but we believe that it is adequate for the study of the kind of question we are interested in, namely the interaction between cortical orientation column structure and intracortical connectivity.

Details of the structure of model retina and lateral geniculate nucleus (LGN) and of their implementation in the simulator are also given in ref. [3]. One of the main results of that paper was that a certain type of specific intracortical long range inhibition (cross-orientation inhibition, [4, 5, 6]) resulted in an unequal average orientation tuning for different populations of cortical cells. This finding triggered our interest in the functional implications of the geometrical arrangement of the functional elements (the receptive fields).

3 Are Large Scale Simulations doomed to success?

We believe that this is *not* the case! In fact, we found that our simulation did not yield the result we expected beforehand, and we feel that the emergence of this kind of surprise is where at least part of the value of simulations lies. This should be considered together with the canonical reasons why simulations are done, like the possibility to do impossible (or nearly impossible) "Gedanken experiments".

In our work, we focused on the interaction between the geometrical arrangement of the orientation columns and the synaptic connections. An orientational bias is induced in cortical cells by the organization of the thalamo-cortical afferents [7]. It is assumed that receptive fields of cortical cells are sharpened by intracortical inhibition. A given cell, called "target cell" receives the strongest inhibition for the *stimulus* orientation which is orthogonal to it's own preferred orientation. This is what we call "functional cross-orientation inhibition". An apparently efficient connection scheme to accomplish this is "structural cross-orientation inhibition". This term denotes the following geometrical arrangement of receptive fields: A target cell, receives inhibitory input from cells called "source cells" with an orientation preference which is orthogonal to that of the target cell.

Although these definitions seem to describe the same situation, we have shown that the "classical" cross-orientation inhibition scheme is subjected to functional limitations introduced by the cortical column structure [8, 9]. We demonstrated that the problems observed for cross-orientation inhibition do not occur for a less

specific connection scheme, which we called "circular inhibition". In this model, inhibition arises isotropically from cells lying on a circle around the target cell.

We introduced circular inhibition as an unspecific connection scheme allowing us to investigate the limits of specificity necessary to achieve functional order. When we analyzed the temporal behavior of the model system in more detail, we found that circular inhibition also introduces a directional bias which is comparable to experimentally obtained values. Similar to cross-orientation inhibition, circular inhibition cannot generate orientation tuning without an initial orientation bias. Directional tuning, however, arises without any preexisting directional bias. Very little connection information (radius and annulus diameter) is necessary to establish circular inhibition, which can be considered as a particular type of long-range lateral inhibition. Thus, circular inhibition represents a rather unspecific and broadly tuned connection scheme which appears easy to implement developmentally. In fact, weak tuning of inhibition compatible with this scheme has been observed recently [10].

It seems plausible that such a readily available directional bias was used in phylogeny and strengthened by enhancing those mechanisms that add to its performance. Such an interpretation is supported by the experimental finding that during development, only a small number of cells are initially directional selective. Many more cells are only weakly biased early in development and their directional selectivity increases only after the development of orientation tuning [11]. This supports our notion that directional tuning follows the emergence of orientation selectivity.

What can we conclude from these experiences for the value of fine-grained computer simulations? When structural cross-orientation inhibition was implemented in the simulation code, its failure to yield functional cross-orientation inhibition first came as a surprise. When the situation was analyzed subsequently, we gained a deeper understanding of the problem by making a simpler model, in which we stripped the model from all properties (like temporal dynamics, stochastic variations, ...) except the orientation selectivity of the neurons. In retrospect, for this particular project, the qualitative insight (but not the quantitative support) could have come without the detailed simulation. We believe, however, that the necessity to quantify the qualitative (and plausibly looking) ideas was a necessary step in the process of understanding.

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Figure Caption

Structure of the model. The simulator comprises models of a patch in the retina (approx. 1 $mm \times 1$ mm) and the projections to the LGN. The central part of the corresponding area in primary visual cortex (shown in black) is simulated. For all details of the simulation see refs. [2, 3].