Chapter 7
On the Emergence of Orientation Biases in V1
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Abstract Though the site of the emergence of orientation biases in the primary visual cortex (V1) is still debated, it seems that the consensus is converging on the idea originally suggested by Hubel and Wiesel in early 1960’s; the idea that the convergence of spatially-arranged geniculate inputs on single cortical cells is the source of orientation biases in V1. But, is the Hubel–Wiesel type of geniculocortical connectivity is the only choice V1 has to generate orientation biases?

Introduction
Intracellular recordings [1] provided strong evidence that the excitatory centers of the RFs of the studied orientation-selective simple cells were nearly circular. In addition, experimental studies based on blocking the cortical GABAergic inhibition by intracortical administration of bicuculline [2] demonstrated that the orientation selectivity of the studied simple cells were reversibly abolished to a degree that their RFs became virtually circular, or that even in some cases, the cells’ original orientation preferences were reversibly changed. These pieces of experimental evidence, firstly, imply the existence of non-thalamic source of orientation biases in the responses of cortical cells, and secondly indicate that the convergence of spatially-arranged non-oriented geniculate inputs is by no means the only possible mechanism that enables V1 to generate orientation biases.

Research Goal
The main goal is to construct a spiking neuromorphic model in order to demonstrate how foveal V1’s neural circuitry could generate orientation biases from non-oriented thalamic inputs without resorting to a Hubel–Wiesel type of geniculocortical-connectivity mechanism.

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Method

In my previous article [3], I developed a physiologically-plausible computational model for showing how a mature foveal V1 could, in principle, create cortical excitatory/inhibitory orientation biases from non-oriented thalamic inputs. To develop the model, I adopted the Marr’s information-theoretic approach. According to Marr’s prescription, in order to understand the intracortical information processing that underlies the emergence of orientation biases in V1, we need to answer the following interrelated questions: (1) what is the computational goal of V1? (2) how does V1 achieve its computational goal? and (3) how does the V1’s hardware implement its computational goal? In that article, I provided an answer to the first question by formulating a probabilistic computational theory for the emergence of orientation selectivity in V1. The formulated theory comprised: (1) a computational scenario, (2) a two-layer hierarchical Markov random field, which was assumed to generate the the activity patterns of the lateral geniculate nucleus (LGN) cells, and (3) a Maximum-A-Posteriori (MAP) estimation of the activity pattern of the orientation-selective cortical cells for a given LGN activity pattern, which was envisaged as the computational goal of V1. There, an answer was also provided for the second question by developing a physiologically-plausible parallel algorithm that enables V1 to achieve its computational goal. In this article, I introduce, very briefly, a spiking neuromorphic model as an answer to the last question.

The Orientation-selective Computational Models

For the sake of concreteness, let’s consider the following system of local updating rules and doubleton-clique energies which describe a horizontally-tuned physiologically-plausible Bayes–Markovian computational model.

The Local Updating Rules

\[
\tilde{x}_{ij}(n+1) = \arg \max_{x_{ij} \in \{X, \tilde{X}\}} \left\{ - \sum_{C \in C'_{ij}} \mathcal{E}(y_{ij}, y_{C'} | x_{ij}; \beta, T) - \max \left\{ - \sum_{C \in C'_{ij}} \mathcal{E}(y_{ij}, y_{C'} | x_{ij}; \beta, T), \sum_{C \in C'_{ij}} \mathcal{E}(y_{ij}, y_{C'} | x_{ij}; \beta, T) \right\} - \sum_{C \in C'_{ij}} \mathcal{E}(x_{ij}, \tilde{x}_{C'}(n); \gamma(n)) \right\}
\]

(7.1)

for all \((i, j) \in \mathcal{L}\), where \(\mathcal{L}\) denotes an \(N \times N\) rectangular lattice.