Combined Hebbian development of geniculocortical and lateral connectivity in a model of primary visual cortex

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Received: 20 December 1999 / Accepted in revised form: 9 June 2000

Abstract. We present a network model of visual map development in layer 4 of primary visual cortex. Our model comprises excitatory and inhibitory spiking neurons. The input to the network consists of correlated spike trains to mimic the activity of neurons in the lateral geniculate nucleus (LGN). An activity-driven Hebbian learning mechanism governs the development of both the network’s lateral connectivity and feedforward projections from LGN to cortex. Plasticity of inhibitory synapses has been included into the model so as to control overall cortical activity. Even without feedforward input, Hebbian modification of the excitatory lateral connections can lead to the development of an intracortical orientation map. We have found that such an intracortical map can guide the development of feedforward connections from LGN to cortical simple cells so that the structure of the final feedforward orientation map is predetermined by the intracortical map. In a scenario in which left- and right-eye geniculocortical inputs develop sequentially one after the other, the resulting maps are therefore very similar, provided the intracortical connectivity remains unaltered. This may explain the outcome of so-called reverse lid-suture experiments, where animals are reared so that both eyes never receive input at the same time, but the orientation maps measured separately for the two eyes are nevertheless nearly identical.

1 Introduction

How does a cortical map arise? This is a longstanding question that has stimulated many experimental and theoretical investigations. On the one hand, it has been proposed that the layout of any map may be genetically coded. On the other hand, cortical maps may form through a Hebbian learning process driven by spontaneous or sensory-induced neuronal activity. Here we suppose that the basic network structure is made available by genetic coding and show how most of the orientation map in primary visual cortex can emerge from Hebbian plasticity of intracortical and geniculo-cortical interactions. Since the discussions concerning the origin of cortical maps are intense, we start with a short review.

Measurements of neuronal activity in primary visual cortex of cat have shown that cortical cells respond well to stimulation within a certain receptive field on the retina. Many cells respond preferentially to bar-like stimuli of a specific orientation and are activated predominantly through one of the two eyes. The location of the receptive field, the preferred orientation, and the ocular dominance of recorded cells change gradually as the recording site is moved tangentially to the cortical surface (Hubel and Wiesel 1962). The global organization of these cortical response properties has been mapped by anatomical, electrophysiological, and optical imaging methods for cat (Tusa et al. 1978; Bonhoeffer and Grinvald 1991; Bonhoeffer and Grinvald 1993), monkey (LeVay et al. 1975; Hubel et al. 1977; Blasdel and Salama 1986; Blasdel 1992a,b), ferret (Law et al. 1988; Chapman and Stryker 1993; Chapman et al. 1996; Weliky and Katz 1997), and tree shrew (Humphrey and Norton 1980; Humphrey et al. 1980; Bosking et al. 1997).

It is, however, still a matter of debate how cortical orientation selectivity is set up (Freerster and Miller 2000). Hubel and Wiesel (1962) originally proposed that geniculo-cortical connections are arranged so that the receptive field centers of thalamic cells projecting onto a single cortical simple cell cover an elongated region in the visual field. While there are many experimental studies claiming that the response properties of simple and complex cells are mainly determined by feedforward projections, as suggested by this model (Freerster 1987, 1988; Reid and Alonso 1995; Freerster et al. 1996; Chung and Freerster 1998), others find that intracortical links provide the main contribution (Sillito 1979; Sillito et al. 1980; Crook and Eysel 1992; Nelson et al. 1994). }
present it seems most likely that both feedforward and recurrent intracortical processes both excitatory and inhibitory in nature participate in the formation of orientation selectivity (Vidyasyagar et al. 1996).

Theoretical studies (von der Malsburg 1973; Linsker 1986a,b; Kammen and Yuille 1988; Stetter et al. 1993; Miller 1994; Wimbauer et al. 1997a,b) have proposed a Hebbian development of geniculocortical synapses that is driven by correlated feedforward input from thalamic neurons (for a recent review see Miller et al. 1999). In these correlation-based approaches, the intracortical connectivity is usually assumed to be rotationally symmetric and fixed. Under relatively general conditions they predict the emergence of an orientation map for cortical simple cells which is formed by the resulting arrangement of feedforward connections.

As such, the above explanation of cortical map formation has been challenged by work of Gödecke and Bonhoeffer (1996) and Sengpiel et al. (1998). In their experiments, cats were raised so that both eyes never received visual input at the same time, which was achieved by reverse lid suture. If geniculocortical refinement were driven by activity correlations in the lateral geniculate nucleus (LGN) and these correlations were mainly determined by the correlations of the visual input, then the left-eye orientation map would form independently of the right-eye map and the two maps could be expected to be different. Optical imaging of area 18, however, showed them to be nearly identical. The authors concluded that each map's layout was fixed by some internal mechanism either a priori or during the period when the first eye was open. They proposed long-range horizontal projections within primary visual cortex as a potential substrate of this mechanism.

Recently, however, Erwin and Miller (1998) have demonstrated that the emergence of ocularly-matched orientation maps can be well explained within the framework of correlation-based development, if an appropriate amount of thalamic inter-eye activity correlations is assumed. Experimental findings of Weliky and Katz (1999) indicate that strong inter-eye correlations are indeed present in the ferret's LGN before eye opening. Nevertheless, it is still unclear whether this model can actually account for the outcome of the reverse lid-suture experiments.

Wolf et al. (1996) have pointed out that cortical area 18 of the cat is shaped as a narrow band on the cortical surface, so that pattern formation within this region is subject to strong confinement. In computer simulations they have shown that different feedforward orientation maps developing under this constraint are always very similar – in accordance with reverse-suturing experiments. The authors argued that experimental results should be qualitatively different in a larger area, e.g. area 17, because boundary conditions are less important. As Bonhoeffer and Gödecke (1996) have explained: “Unfortunately this idea is difficult to test, as in cats the main part of area 17 lies buried in the medial bank and is therefore inaccessible to optical imaging”.

In this paper we propose a model of layer 4 of primary visual cortex consisting of laterally interconnected spiking neurons of both excitatory and inhibitory type. It combines the idea of correlation-based learning of geniculocortical afferents with Hebbian development of short-range intracortical synapses. Inhibitory interneurons and plastic inhibitory synapses have been included in the model so as to control overall network activity. Large-scale computer simulations show that in this kind of network it is possible to obtain an intracortical orientation map from a Hebbian learning process driven by cortical activity alone. The process does not depend on the presence of feedforward input and could therefore occur at early stages of visual development, when thalamic axons have not yet entered cortical layer 4. The resulting map structure resembles that typical of orientation maps obtained from optical imaging experiments. This might indicate that intracortical circuitry does contribute significantly to the orientation selective response properties of cells in the primary visual cortex.

Experiments by Ferster (Ferster 1987, 1988; Ferster et al. 1996) do indicate, however, that feedforward input from the LGN is relevant as well. Consistently with these data, we demonstrate that correlation-based development of geniculocortical projections can interact with emerging intracortical connectivity so as to give a matched feedforward and intracortical orientation tuning for each cortical cell. As a consequence, the developing pattern of feedforward projections is more or less predetermined once the intracortical connectivity is fixed. This provides a very natural explanation for the remarkable stability of orientation maps that has been found experimentally (Kim and Bonhoeffer 1994; Weliky and Katz 1997; Gödecke and Bonhoeffer 1996; Sengpiel et al. 1998, 1999). In contrast to the proposition of Wolf et al. (1996), our model predicts strong correlation between orientation maps in reverse-suturing experiments not only for small but also for larger visual areas such as area 17 of the cat.

This paper is organized as follows. In Sect. 2 we introduce the model of spiking neurons that we have used in our simulations. We then turn to a description of the full network, explain the learning rules that govern synaptic development, and finally we present our data-analyzing procedure. In Sect. 3 we show that intracortical orientation maps can develop from spontaneous cortical activity alone, without feedforward input. We demonstrate how this intracortical development can be combined with plasticity of geniculocortical connectivity and show that such a combined development can explain the outcome of the reverse-suturing experiments. We end with a summary and a short discussion.

Part of this work has been presented in preliminary form – see Bartsch and van Hemmen (1999).

2 Methods

2.1 Spiking neurons

To keep our network as close to biology as possible, we decided to build it up from spiking neurons. A neuron model that is able to reproduce many biological features