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Spontaneously emerging direction selectivity maps in visual cortex through STDP

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Abstract It is still an open question as to whether, and how, direction-selective neuronal responses in primary visual cortex are generated by feedforward thalamocortical or recurrent intracortical connections, or a combination of both. Here we present an investigation that concentrates on and, only for the sake of simplicity, restricts itself to intracortical circuits, in particular, with respect to the developmental aspects of direction selectivity through spike-timing-dependent synaptic plasticity. We show that directional responses can emerge in a recurrent network model of visual cortex with spiking neurons that integrate inputs mainly from a particular direction, thus giving rise to an asymmetrically shaped receptive field. A moving stimulus that enters the receptive field from this (preferred) direction will activate a neuron most strongly because of the increased number and/or strength of inputs from this direction and since delayed isotropic inhibition will neither overlap with, nor cancel excitation, as would be the case for other stimulus directions. It is demonstrated how direction-selective responses result from spatial asymmetries in the distribution of synaptic contacts or weights of inputs delivered to a neuron by slowly conducting intracortical axonal delay lines. By means of spike-timing-dependent synaptic plasticity with an asymmetric learning window this kind of coupling asymmetry develops naturally in a recurrent network of stochastically spiking neurons in a scenario where the neurons are activated by unidirectionally moving bar stimuli and even when only intrinsic spontaneous activity drives the learning process. We also present simulation results to show the ability of this model to produce direction preference maps similar to experimental findings.

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1 Introduction

The development of direction-selective neuronal responses in visual cortex is widely believed to be based on activity-dependent Hebbian learning (Wimbauer et al. 1997; Feidler et al. 1997; Senn et al. 2002) early in an animal's life. It is, however, still much debated (Livingstone 1998; Clifford and Ibbotson 2003) what kind of neuronal circuit underlies the generation of directional responses and where it is located. With the primary visual cortex being the first level at which these selectivities are found, it is evident that either thalamocortical and/or intracortical processing must be responsible for direction selectivity. In addition, the realization through feedforward or recurrent circuits, the role of inhibition, the spatiotemporal summation of channels of different temporal response characteristics, and contributions of short- and long-term plasticity have been considered in a multitude of experimental and theoretical investigations (Suarez et al. 1995; Wimbauer et al. 1997; Mineiro and Zipser 1998; Livingstone 1998; Sabatini and Solari 1999; Rao and Sejnowski 2001; Senn et al. 2002; Roerig et al. 2003; Shon et al. 2004).

A mechanism suggested by Mehta and Wilson (2000) attributes direction selectivity to a simple asymmetrical thalamocortical connection scheme that had originally been proposed for hippocampal place cells in rats. The latter encode the position of a rat in its environment in an experience-dependent fashion. When the rat traverses a region of space several times in the same direction the synaptic connections to a neuron with a corresponding place field will shift in a direction opposite to the direction of motion and thus polarize the initally isotropic synaptic input pattern. This has the effect that, after learning, the neurons show anticipatory responses for the future location of the rat. Mehta suggests that the same principle of asymmetric integration fields might be utilized in visual cortex neurons which could then 'anticipate a moving stimulus'. The underlying asymmetry, as in the hippocampus, could be achieved by spike-timing-dependent learning with an asymmetric learning window.

Here we investigate the idea of learning an asymmetric coupling structure for intracortical connections in a recurrent network of stochastically spiking neurons. We have taken into consideration the finding of Bringuier et al. (1999) that the integration field of a cortical neuron is not homogeneous with regard to the delay at which inputs are registered in its membrane potential. Probably due to slow axonal propagation the action potential of an input cell has to travel the longer, the larger the distance between source and target cell is. Accordingly, the delay of inputs increases with their distance resulting in an isotropic latency basin. As we will show, this specific temporal structure within the integration field can be utilized beneficially to extract spatiotemporal correlations from activity in the network.

Only until recently theoretical models have been based almost exclusively on *rate* descriptions of the neuronal dynamics and learning rules were assumed to be sensitive only to rate correlations. With the experimental finding that the learning process in a synapse depends on the relative timing of the spiking activity of its pre- and postsynaptic neuron (Markram et al. 1997; Bi and Poo 1998; Zhang et al. 1998; Feldman 2000; Froemke and Dan 2002), models were quickly adapted to incorporate the so-called spike-timing-dependent plasticity (STDP): Only neuronal connections from inputs that have emitted a spike shortly before their postsynaptic target are strengthened whereas all input neurons that send a spike after their target cell get weakened resulting in a temporally asymmetric learning rule. Because synaptic changes occur only *within* a short period of time centered around the postsynaptic spike (ca. ± 50 ms) it is often referred to as a 'learning window' (Gerstner et al. 1996). Kempter et al. (1999) have analyzed the main differences between STDP and rate-based models.

Bartsch and van Hemmen (2001) and also Yao et al. (2004) have indicated how orientation selectivity can develop in a recurrent network model of primary visual cortex by STDP forming smooth maps of orientation preferences. Extending the purely spatial to spatio-*temporal* fine-tuning of synapses by an asymmetrical learning window we show how maps of direction preference may emerge in a similar scenario, much like the maps found experimentally in optical-imaging studies (Weliky et al. 1996; Shmuel and Grinvald 1996).

By considering two activity scenarios we demonstrate that both highly correlated, structured visual input and cortex-intrinsic spontaneous activity is able to drive the learning of synapses with an asymmetrical learning window to produce direction selective neurons. Whereas spatiotemporal correlations in visual input arise from, e.g., moving contrast edges within the sensory input relayed from the retina, the correlations in the cortical network's spontaneous activity without external input are generated solely by the processing through its already existing connections. The simulation results show that even without structured input the formation of typical direction preference maps is possible.

Part of this work has already been presented in Noll et al. (2002).

2 Model Description

As already pointed out, the learning processes in a neural network depend on the exact timing of the spiking of its neurons. We therefore start with a network of neurons described by the Spike-Response Model (SRM, Gerstner and van Hemmen 1994), a stochastic spiking neuron model which is similar to the common integrate-and-fire models and well suited to investigate large network simulations but can still capture many biological details. In this ansatz the state of a neuron i is determined by its instantaneous membrane potential $h_i(t)$ where inputs from other neurons j at times t_j^f are summed up as postsynaptic response functions $\epsilon(t)$ weighted by the momentary synaptic coupling strength $J_{ij}(t)$. In addition, the neuron's refractoriness due to its own spike emission is taken into account by an exponentially decaying kernel $\eta(t)$, which artificially reduces a neuron's membrane potential for a certain period of time after a spike, making it less likely to spike again in short succession,

$$h_i(t) = \sum_j \sum_{t_j^f \le t} J_{ij}(t_j^f)\epsilon(t - t_j^f) - \sum_{t_i^f \le t} \eta(t - t_i^f).$$

For simplicity we chose $\epsilon(t) = \exp(-t/\tau_{\mathrm{EPSP/IPSP}})\theta(t)$ for excitatory and inhibitory inputs, respectively, and $\eta(t) = \eta_0 \exp(-t/\tau_{\mathrm{ref}})\theta(t)$, where $\theta(t)$ refers to the Heaviside step function. After transforming the membrane potential by a rectifying activation function we use the resulting value as the instantaneous rate function of an inhomogeneous Poisson process to compute the spike times t^f of the neuron as the simulation goes along. Accordingly, the spiking probability of a neuron during the interval $[t, t + \mathrm{d}t)$ with $\mathrm{d}t = 1$ ms is given by the sigmoidal $P_i^f(t)\mathrm{d}t = \mathrm{d}t/\{1 + \exp[-\beta(V(t) - \theta)]\}$ with a noise parameter β and θ the sigmoid's threshold. Following each spike a new refractory kernel $\eta(t)$ is subtracted from the membrane potential. For parameter values used in the simulations see Table 1.

The asymmetrical learning window has been chosen as shown in Fig. 1. It consists of an exponentially decaying half for long-term potentiation (LTP) and an α-function shaped half for depression (LTD). The width of the depressing part has been chosen considerably longer than that of potentiation to weaken connections from massively delayed inputs. We have assumed plasticity only for excitatory synapses and kept inhibitory coupling strengths constant during the simulation runs.

Table 1 Parameters used in simulations

Spike Generation		Synaptic Response	
β	0.5	τ_{EPSP}	6 ms
θ_{exc}	2.0	τ_{IPSP}	30 ms
θ_{inh}	3.0	τ_{ref}	10 ms
Learning Window		η_0	+40.0
τ_{LTP}	11 ms	synapse growth limits	
τ_{LTD}	20 ms	$0 \le J_{\mathrm{ij}} \le 0.8$	
$\Delta t_{\mathrm{ij}}^{\mathrm{max}}$ in integr. field	20 ms	v_{axon}	0.2 m/s

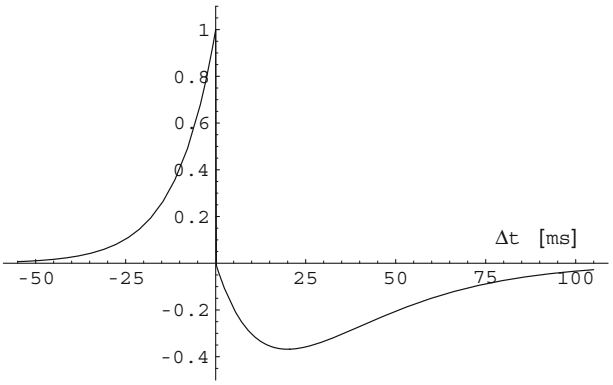

Fig. 1 Asymmetric learning window with an exponentially decaying part for LTP and a longer-lasting half for LTD . The horizontal axis represents the delay between pre- and postsynaptic spike $\Delta t = t_{\text{pre}} - t_{\text{post}}$ in ms. Vertical axis with arbitrary units

To model the primary visual cortex we take a two-dimensional network (Fig. 2) consisting of one layer of excitatory and another layer of inhibitory neurons. Neurons are arranged in a grid, e.g., 64x64 in size, where each neuron receives recurrent inputs from neighboring neurons of both polarities up to a certain radius (typically 7 grid positions). We will refer to this area of inputs as the neuron's *integration field*. The inputs from positions within the integration field are attenuated by an arbor function of Gaussian shape which is used to account for a reduced number of synaptic contacts at larger distances. By choosing $\sigma_{\text{exc}} = 1.0$, $\sigma_{\text{inh}} = 2.24$ inhibitory inputs are stronger than excitatory ones in the peripheral part of the input fields.

As has been found by Bringuier et al. (1999), the transmission delay Δt_{ij} between two synaptically connected neurons can be described by an isotropic function of their separation $d_{\text{ij}} = |\mathbf{x_i} - \mathbf{x_j}|$, .i.e., $\Delta t_{\text{ij}} = d_{\text{ij}}/v$ with v on the order of 0.2 m/s for thin (and thus slowly conducting) axons. We have therefore delayed inputs from the integration field from 0 ms at the same grid position to 20 ms for the most distant neurons. This corresponds to an axonal propagation velocity of about $50^0/s$ for an integration field extending over 2 mm in cortex, equivalent to roughly 2^0 in the visual field. In our setup we make the simplifying assumption that the whole integration field is able to drive the target cell to spiking, whereas in reality the integration field consists of a central discharge field and a surrounding area from which only subthreshold responses can be elicited. Furthermore, we assume dendritic latencies, synaptic delay and EPSP rise time to contribute only a small additional constant delay offset of 3 ms. Although this value, in some cases, may be significantly higher (cf. Senn et al. 2002), we focus for the moment on axonal delay selection in this study and therefore treat dendritic influences as constant and small as compared to axonal propagation times; see also Anderson et al. (1999) for evidence that dendritic asymmetry cannot account for directional responses.

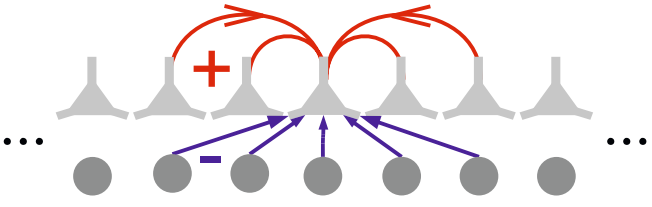

Fig. 2 Scheme representing a 1D slice through the two-dimensional recurrent network built from excitatory and inhibitory neuron layers that are used to model the primary visual cortex. Excitatory neurons are depicted by templates of pyramidal shape in light gray, inhibitory cells as discs in darker gray. For the excitatory neuron in the center the excitatory (+) and inhibitory (-) inputs from neighboring cells are shown to illustrate the spatially restricted range of connections between neurons in the network, which also represents the area of the neuron's integration field. One has to keep in mind that longer connections also imply a larger delay due to slow action potential propagation along the underlying axons. This connection scheme is to be imagined for every neuron, supplemented with periodic boundary conditions in two dimensions

In Fig. 3 the principle of direction selective neuronal responses based on asymmetric coupling of inputs within a neuron's integration field is illustrated in terms of the membrane potential of a cell with strong synapses for inputs from its left half of the integration field. The cell therefore responds preferentially, i.e., most strongly to rightward-moving activity waves.

Given the ability of asymmetric integration fields to produce directionality, how can it be "learned" by spike-timing-dependent plasticity? Looking at Fig. 4 we can see how the asymmetric learning window changes the coupling strengths within the integration field when learning is driven by an activity wave. It is evident that repeated stimulation by a wave coming from the same direction will increase weights more and more in that half of the input field pointing to the approaching wave. Eventually, only these synapses will remain whereas all others will have become silent or have disappeared.

3 Results

As a proof of concept we show in a first scenario how direction-selective weight profiles within the integration fields of the neurons can develop through STDP when spiking activity in the recurrent network is mainly driven by an external stimulus, namely, a bar moving unidirectionally from left to right over the cortical network.

3.1 Scenario 1–Learning driven by structured input with unidirectional motion

To simplify and speed up the simulations the lateral geniculate nucleus (LGN) cells relaying retinal input to the cortex have not been explicitely modeled but an appropriately preprocessed stimulus is added directly to the cortical cells' membrane potential. In Fig. 5 one can get an impression of the excitatory and inhibitory spiking activity in the network caused, on the one hand, by the bar stimulus and, on the other

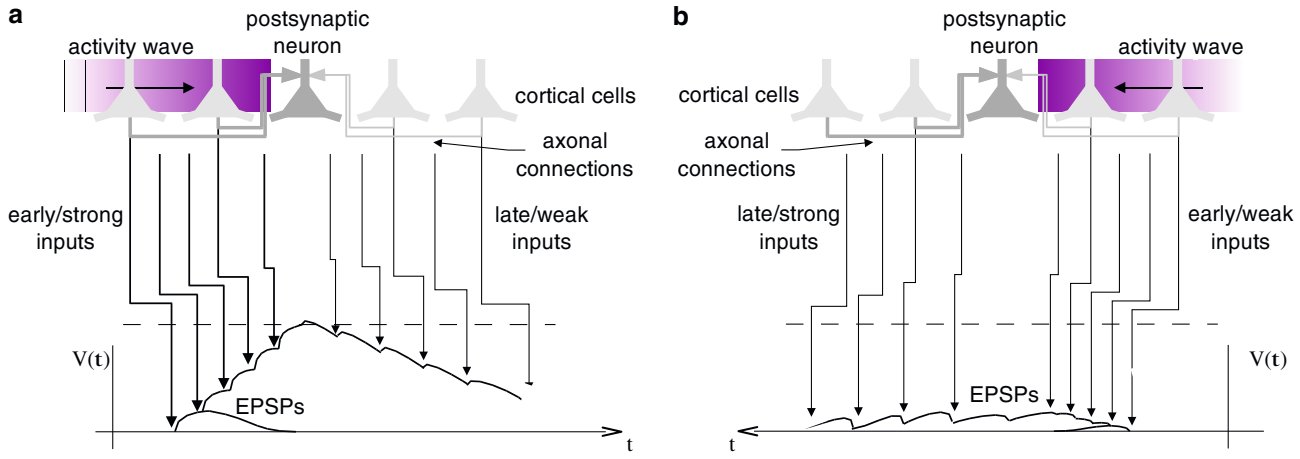

Fig. 3 Principle of direction-selective neuron responses based on a spatially asymmetric arrangement/weighting of axonal delay lines. The two schemes show the time course of the membrane potential (bottom graphs) of a target cell with strong synapses from inputs within the left half of its integration field for the case of stimulation by an acitivty wave moving through the input field (a) from left to right and (b) moving in the opposite direction. Only excitatory contributions are shown. The neurons depicted in dark gray in the centers of the two figures represent the target neurons onto which inputs from laterally displaced neighbors converge via slowly conducting axons. The latter are depicted as lines of different thickness to visualize strong and weak coupling strengths at the corresponding synapses. When the activity wave enters the integration field from the left where strong synapses are delivering their signals to the target with shorter and shorter delays, the input EPSPs arrive very closely in time and thus sum synergistically. This will lead the neuron to fire because the decreasing axonal delays from distant to closer inputs provide highly coincident input for the target cell. For movement from right to left, however, the weaker synapses cannot sufficiently elevate the membrane potential even though they arrive in rapid succession. The stronger inputs arrive too late and do not overlap much due to the more and more increasing delays which disperse the incoming signals. In this way, a cell with an asymmetric weight structure responds with different strength in the two cases of stimulus movement and is thus direction selective

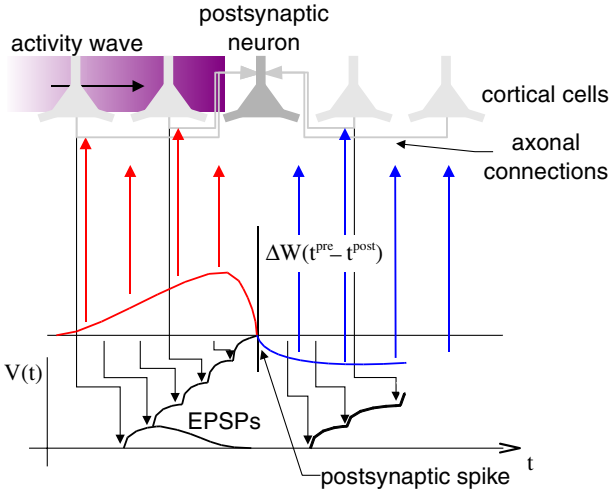

Fig. 4 How can spike-timing-dependent learning account for the emergence of spatially asymmetric input weights to form a direction-selective neuron? Here a snapshot of a neuron is depicted that sums input from a wave of elevated spike activity produced, e.g., by a bar stimulus moving through the neuron's input field. The axonally delayed inputs are registered (black vertical arrows at EPSP onsets) in the membrane potential of the target cell, which will spike when the membrane potential crosses the spike threshold. The temporally asymmetric learning window $W(\Delta t)$ with $\Delta t = t^{\text{presyn}} - t^{\text{postsyn}}$ (Fig. 1) can now be imagined to be centered at the spiking time of the postsynaptic target cell and all input lines that participated in bringing the neuron to fire get potentiated (red arrows for 'early' or 'driving' inputs) according to the value of the learning window for the time difference of the corresponding pre- and postsynaptic spike. Further inputs that are activated by the activity wave but after the postsynaptic spike ('late inputs') will get depressed by the negative part of the learning window (blue arrows). If this process is repeated several times, the weights of inputs coming from the direction of the moving wave will get strengthened and those on the opposite side will decrease even further. Ultimately, this leads to an asymmetric and thus direction-selective weight profile

hand, by spontaneous spiking due to the stochasticity of the neuron model.

Starting with a rotationally isotropic connection pattern between neighboring neurons in the recurrent network the cells respond equally strongly to all stimulus directions. In what follows, the stimulation will be restricted to a bar moving repeatedly from left to right over the simulated cortex. As soon as a neuron is activated to spike the symmetry in

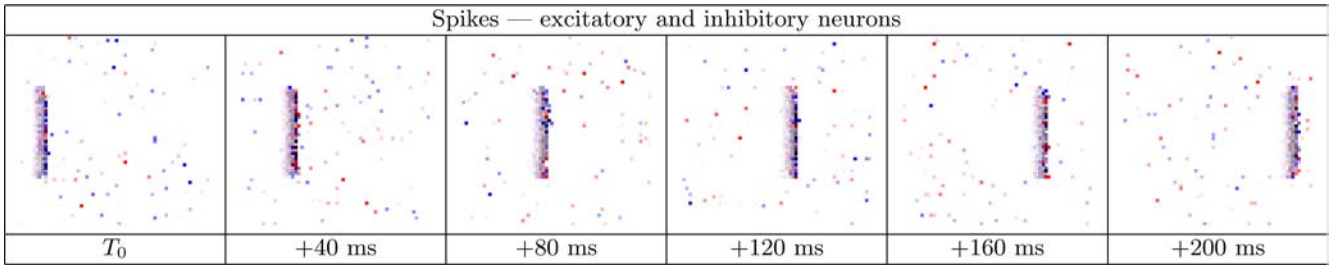

Fig. 5 In this series of pictures the evolution of the spiking activity within the two-dimensional recurrent network (64x64 cells) is depicted for a period of 200 ms simulated real time with the activity wave due to the cortical representation of a bar stimulus moving from left to right and several spontaneous spike events distributed over the cortex. Spikes are coded in red for excitatory and in blue for inhibitory cells, black is used when both types of cell are active simultaneously

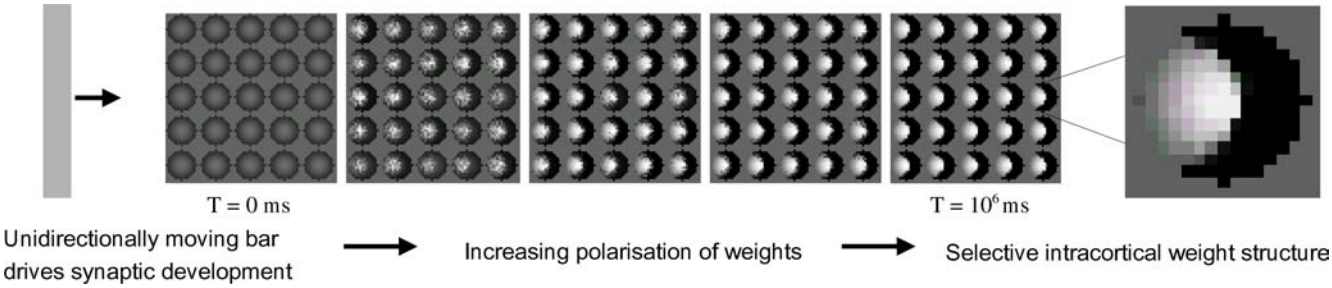

Fig. 6 This figure shows the changing of lateral synaptic coupling patterns for 5 by 5 cells when stimulated by a bar moving repeatedly from left to right over the integration fields while learning with an asymmetric learning window. The neurons' coupling strengths to their neighbors are shown as circular profiles in which weight values are coded by a grey scale with white representing maximal weight. At the beginning, the weight profiles are initialized as rotationally symmetric with attenuated coupling strengths for distant inputs. During persistent periods of stimulation and synaptic plasticity (the picture series shows snapshots every 250 seconds of computed realtime), the learning rule selects appropriately delayed inputs to best activate each cell by the stimulus and thus grows a more and more asymmetric spatial distribution of synaptic couplings. It is evident that exactly those weights are strengthened that couple a neuron to its neighbors in the direction into which the stimulus moves over the network. At the end, the initially rotation-symmetric weight profile has turned into a highly asymmetric one, which we have seen to allow for direction-selective responses

the connection pattern will be broken up by the learning window, i.e., a small seed of asymmetry will be introduced in the weighting of inputs pointing in the direction the bar has come from. As can be seen in figure 6 the repetition of learning events stimulated by the moving bar more and more polarizes the connection pattern until only a restricted range of inputs within the receptive field remains. This arrangement of inputs represents a direction-selective neuron (Fig. 3).

3.2 Ongoing spontaneous activity in a recurrent network

Next we consider a cortical activity scenario without external input to the network. By adjusting the strength, balance, and time constants of synaptic potentials for excitatory and inhibitory recurrent inputs a configuration can be found where neurons fire at a low rate of only a few spikes per second most of the time. Because of fluctuations due to the stochastic activation of neurons this ongoing baseline activity is occasionally intermitted by short excursions of excitation which appear at random locations in the network. They start out as small excitation seeds gaining size rapidly and, after a short period, fade out as soon as inhibition catches up with the excitatory activity. As the return to baseline usually takes several tens of milliseconds this allows the rise in activity to spread out in a random direction and move over the cortex in the form of small activity wavelets or bubbles. A typical example of this kind of activity fluctuation is given in Fig. 7 below.

3.3 Scenario 2 — Learning driven by spontaneous network-intrinsic activity

The spontaneous bubble- or wavelet-like network activity seems intriguingly well-suited to drive synaptic plasticity with an asymmetric learning window in a way similar to the external bar stimulus (section 3.1). One might therefore wonder whether it could serve an instructive role for the development of direction-selective weight patterns when spike-timing-dependent learning is 'switched on'.

Small waves of activation break the symmetry of the initial coupling patterns introducing a slight asymmetry through the learning window. It turns out that this asymmetry in itself is not strong enough to withstand synaptic changes elicited by other waves moving in different directions. By introducing, however, a sliding learning threshold dependent on the recent activity of the neuron, it is possible to stabilize asymmetries that have already been learned. Due to the learning threshold only strong activations that drive the activity of the neuron across a certain level determined by the recent activation of

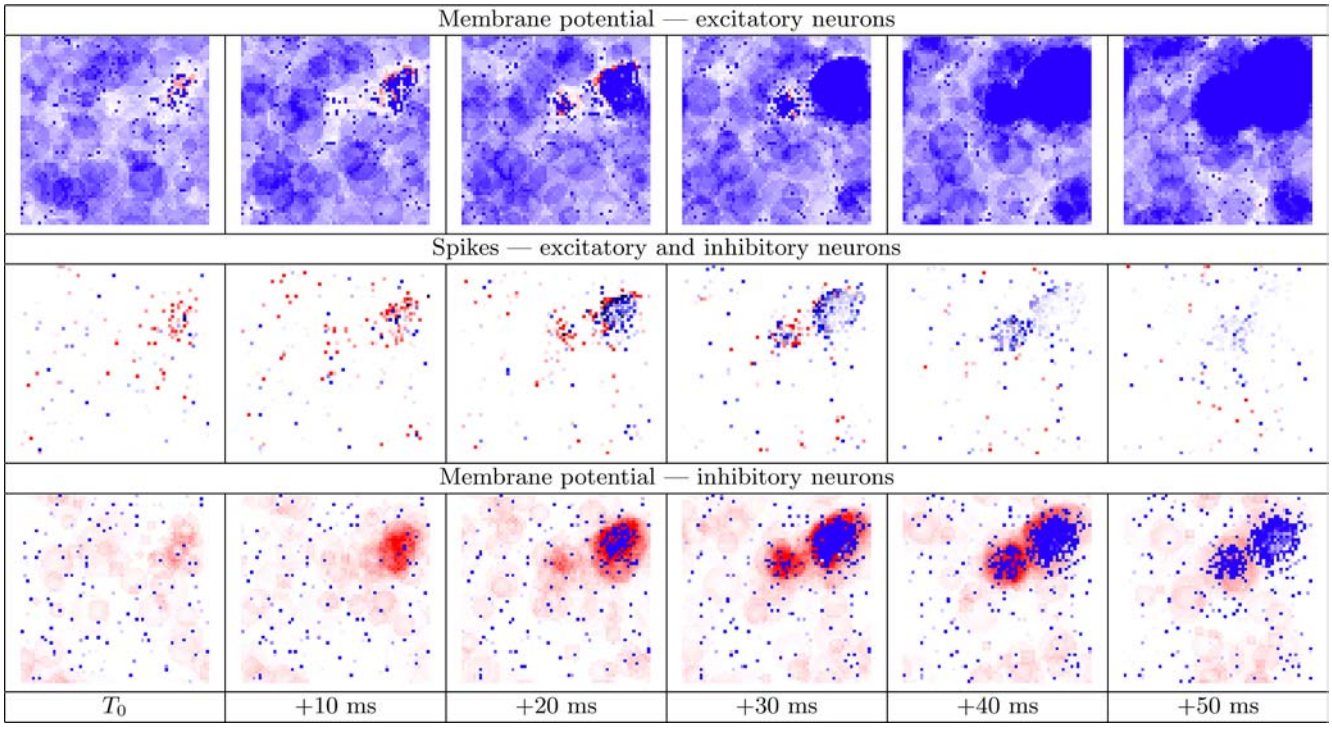

Fig. 7 In this series of pictures the evolution of the membrane potential and the spiking activity within the two-dimensional recurrent network is depicted for cortical spontaneous activity during a period of 50 ms simulated real time. In the top row the excitatory cells' membrane potential is shown with blue representing cells having hyperpolarized and red cells having depolarized membrane potential; white areas stand for cells near their resting potential. Given the membrane potential the probability of spiking has been computed — the corresponding distribution of spikes is shown in the second row. Here spikes are coded in red for excitatory and in blue for inhibitory cells; black is used when both types of cell are active simultaneously. The fading out of the colors of some of the spikes represents that these action potentials are older with respect to the interval between the activity snapshots than the ones that are more saturated. In the bottom row the membrane potential of the inhibitory cells is shown; the same color scheme as for the excitatory neurons' potential applies. As can be seen clearly, a small excitatory activity fluctuation at T_0 entrains more and more neighboring neurons forming a small wave (wavelet) which propagates over the cortex until it is canceled by inhibitory activity. The unstructured spontaneous background spiking has been adjusted to about 2-3 Hz

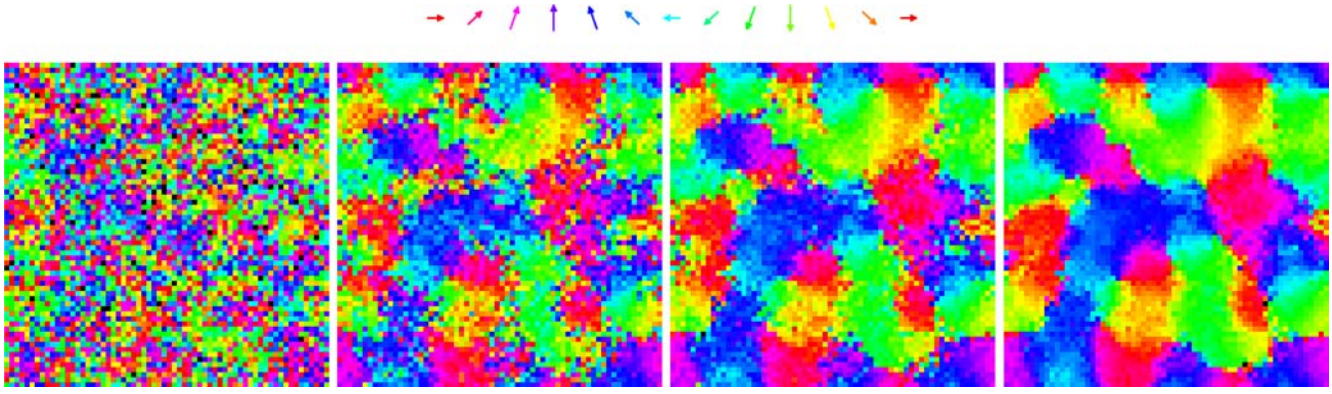

Fig. 8 These color-coded maps depict the arrangement of direction preferences as developed by spike-timing-dependent learning driven by cortex-intrinsic spontaneous wavelet activity. The colors correspond to the direction preferences as represented by the colors of the arrows in the legend above the maps. Starting from an unselective random map at $T = 0$ the spontaneous activity drives development of direction-selective synaptic coupling patterns building small patches of cells with similar preferences. The final map ($T = 10^7$ ms) resembles smoothly changing direction preference maps as found experimentally, e.g., by Weliky et al. (1996) and Shmuel and Grinvald (1996)

the cell can still alter the synaptic inputs' coupling strengths (Ngezahayo et al. 2000).

Starting with an isotropic rotationally symmetric lateral coupling pattern in the synapses on a neuron (without self-couplings) the wavelet activity in the network can drive a learning process that yields smoothly changing asymmetric coupling patterns and stable direction preference maps. The final outcome of this kind of simulation is shown in Fig. 8.

To verify that the developing map arises from the particular composition of wavelet directions in which activity waves have run over the network we show in Fig. 9 a series of snapshots of the spatial distribution of the statistics of

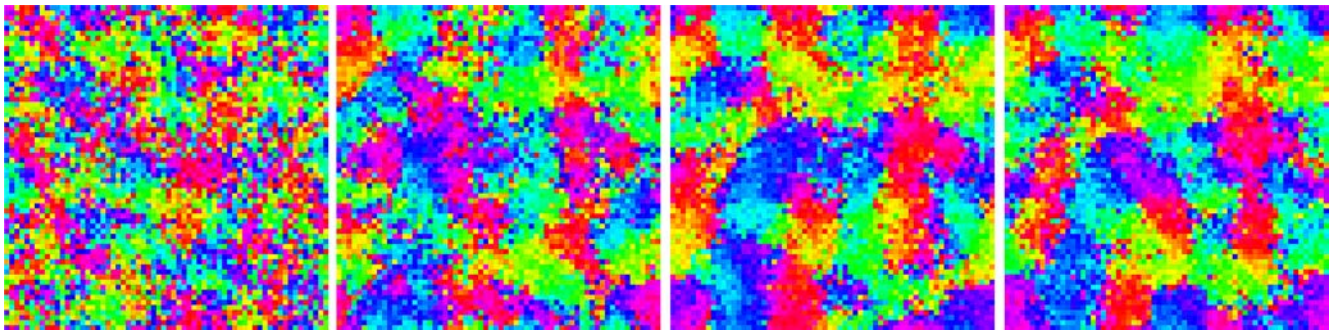

Fig. 9 In these direction statistics maps the color at each pixel corresponds to the most common direction in which wavelets have moved over the corresponding network location during the simulation run (colors as in Fig. 8). The statistics of wavelet motion in each frame have been collected over a period of 1 second simulated real time. In the beginning, the spatial distribution of the statistics of wavelet motion is randomly distributed. As learning proceeds and weight profiles get increasingly polarized, patches with similar preferences start to grow and the learned direction preference maps (Fig. 8) more and more resemble the corresponding direction statistics map

wavelet directions. By averaging over 1 second real time of spiking network activity we have computed statistics maps at several instances. Obviously, the statistics of the direction ensemble of wavelets within the first second of the simulation run does not have a structured distribution of direction preference, i.e., the initial spontaneous wavelets are directionally unbiased. From the sequence of direction-statistics maps one can clearly see that the wavelets become increasingly deflected into directions that have been learned by STDP polarizing the lateral synaptic connection patterns. To be more precise, the propagation of activity waves into directions that have passed a given region within the map not so often will die out faster than for directions that have passed more frequently already. In this way, a once-established directional asymmetry at one location will continue to be potentiated and, in just the same way, also influence its neighborhood (and direction statistics) accordingly.

The above result demonstrates that the correlations in the network-generated wave-like activities are sufficient to drive the learning process of spike-timing-dependent plasticity in such a way as to select those inputs to a neuron that increase the synchronicity of the cell's activation by these wavelets and thus produce synaptic coupling patterns that render the cells direction selective.

In a simulation study Bartsch and van Hemmen (2001) have shown that under conditions similar to the ones in the present investigation maps of *orientation* selectivity could emerge. These may start as asymmetrically deformed intracortical connection patterns that afterwards (or even simultaneously) impose an orientation seed in their structure onto the developing thalamocortical synapses. That is to say, the lateral connection pattern is able to influence the feed-forward connections, which could also be true with regard to direction selectivity.

4 Discussion

We have shown how direction-selective neuronal responses can develop by means of spike-timing-dependent plasticity with an asymmetric learning window. It is possible to drive the learning process not only by structured (i.e., explicitely spatio-temporally correlated) external stimulation like a moving bar but also by a special activity scenario of correlated intrinsic spontaneous activity in a recurrent network of spiking neurons. In the optic tectum of Xenopus, for example, an asymmetric modification of receptive fields consistent with spike-timing-dependent learning has already been demonstrated for moving visual stimuli (Engert et al. 2002). A series of other studies also recognized and attributes phenomena like predictive coding, anticipatory activation and motion-induced mislocalization to asymmetric coupling patterns; cf. Mineiro and Zipser (1998); Fu et al. (2004). Furthermore, because with the kind of asymmetric connectivity that, as we have shown, emerges under the learning paradigm, the excitatory inputs to a neuron will preferentially come from cells with similar direction preferences, i.e., inputs will be iso-direction-tuned. This has also been confirmed by experiment (Roerig and Kao 1999).

Recently, Shon et al. (2004) have suggested a related approach regarding the emergence of direction selectivity in a feedforward architecture which is supplemented by intracortical connections. In their model, however, the intracortical asymmetry in the pattern of connectivity is more supportive rather than generative to direction selectivity as we propose here.

Taking into account the temporal structure of a cell's integration field due to delayed axonal inputs the learning process enhances those inputs that contribute to the cell's firing. This increases the synchronicity of activation by future stimuli because input delays are selected in such a way that the signals evoked by an approaching wave of activity arrive coincidently. As has been shown in Shon et al. (2004) and also corresponding to our simulations (data not shown), axonal delays are not essential to the development of the directional asymmetries of the coupling pattern. It is straightforward to see, however, that a neuron sensing a moving activity wave (such as from a bar stimulus) can be improved by delayed inputs because the approaching wave is temporally squeezed (and, conversely, spread out when having passed the target) and coincident input at the integrating neuron is enhanced and thus easier and more certain to be detected. Hereby a delay

in transmission, which might normally be interpreted as a limiting aspect of processing, can be used in a positive way.

As already stated, the symmetry breaking of the initial synaptic coupling patterns is not stable enough to withstand repeated learning influences opposing the patterns "seen" so far if not supplemented by a learning threshold. In contrast to Buchs and Senn (2002) who have also pointed out the need for a (fixed) threshold nonlinearity to stabilize development, we have assumed a sliding learning threshold that averages recent postsynaptic activation and thus automatically adapts to the rise in mean network activity from about 1 Hz to 3 Hz during the simulation. Especially in view of the experimental finding of activation-dependent plasticity by Ngezahayo et al. (2000) a dynamic threshold seems generally applicable.

Finally, the connection pattern of nearby cells in a network driven by intrinsic activity turns out to differ only slightly within a small patch of cortex, in this way giving rise to smooth changes of direction preference. This results in direction maps that resemble maps found in optical imaging experiments. Schütt et al. (2001) have found evidence that the change of the map structure by artificial stimulation is also guided by a learning rule similar to spike-timing-dependent learning and may directly result from the learning rule at the cellular level.

Trying to explain the emergence of direction selectivity with purely lateral intracortical connection schemes (or pure thalamocortical feed-forward architectures) would be an oversimplification. Presumably circuits and mechanisms at both levels contribute to cortical direction selectivity (see also Mineiro and Zipser 1998; Bartsch and van Hemmen 2001; Yao et al. 2004; Shon et al. 2004) and our approach is to be understood as a hypothesis for and crude approximation of the intracortical part of a more complex mechanism. The finding of orientation selectivity being already present in newborn and thus visually inexperienced animals (Banton et al. 1999; Issa et al. 1999; Crair et al. 1998; Katz and Shatz 1996; Chapman et al. 1996; Sur and Leamey 2001; Ferster and Miller 2000) renders the scenario of cortex-intrinsic spontaneous activity (section 3.3) an imaginable model for the formation of direction selectivity and its corresponding map by spike-timing-dependent synaptic plasticity in early postnatal animals, possibly even before eye-opening.

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