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Development of spatiotemporal receptive fields of simple cells: I. Model formulation

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Abstract. A model for the development of spatiotemporal receptive fields of simple cells in the visual cortex is proposed. The model is based on the hypothesis (Saul and Humphrey, 1990) that the convergence of four types of input onto a cortical cell, viz. non-lagged ON- and OFF-inputs and lagged ON- and OFF-inputs, underlies the spatial and temporal structure of the receptive fields. It therefore explains both orientation and direction selectivity of simple cells. The response properties of the four types of input are described by the product of linear spatial and temporal response functions.

Extending an earlier model of one of the authors (Miller, 1994), we describe the development of spatiotemporal receptive fields as a Hebbian learning process taking into account not only spatial but also temporal correlations between the different inputs. We derive the correlation functions that drive the development both for the period before and after eye-opening and demonstrate how the joint development of orientation and direction selectivity can be understood in the framework of correlation-based learning.

Our investigation is split into two parts that are presented in two subsequent articles. In the first and present article the model for the response properties and for the development of direction-selective receptive fields is derived. In a second paper we present simulation results that are compared with experimental data, and also provide a first analysis of our model.

1 Introduction

Simple cells in the visual cortex of cats and many other mammals represent the first cortical processing stage of the visual pathway. Unlike neurons in the lateral geniculate nucleus (LGN) [27], which project to the visual cortex, most simple cells respond selectively to spatial and temporal stimulus parameters such as orientation [26] or the direction of stimulus motion [28]. The combined spatial and temporal response properties of cortical neurons are summarized by the notion of a spatiotemporal receptive field; for a review see [53].

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Since the first discovery of orientation selectivity by Hubel and Wiesel [26], strong experimental efforts have been made to clarify the physiological mechanisms that underlie the receptive field properties of simple cells. Much current evidence [18, 7, 54, 21] supports the classical model of Hubel and Wiesel [28] for the origin of orientation selectivity: ON- and OFF-center LGN neurons converge onto a simple cell, forming subregions within the receptive field of the cortical neuron that are aligned along its preferred orientation.

The physiological origins of direction selectivity and other temporal receptive field properties are less clear. There is an ongoing debate on the question of whether direction selectivity is mainly caused by cortico-cortical connections (for a review see [38]) or by geniculo-cortical connections [57]. In either case, direction selectivity might additionally be amplified [38, 14] or suppressed [21] by intracortical feedback.

It has been proposed by several authors that a *linear* spatiotemporal receptive field, as can be measured in reverse correlation experiments, might underlie cortical direction selectivity [1, 64, 25, 10, 12]. The discovery of non-lagged and lagged cells in the LGN of cats [42, 43] has given support to this hypothesis. Non-lagged and lagged cells that project to the cortex and are combined linearly at a simple cell could provide the temporal offset that is necessary to create a direction-selective receptive field [57, 20, 21]. Experimental support for this hypothesis comes from several sources. In extracellular measurements, an analysis of the time structure of cortical responses shows mixtures of lagged-like and non-lagged-like timing in the responses of direction-selective cells [57]. In intracellular recordings of direction-selective cells [32], analysis of the time structure of EPSPs [34] also demonstrates two underlying temporal components that match the temporal responses of lagged and non-lagged inputs, respectively. Finally, in at least a few cells, direction selectivity emerges in otherwise non-direction-tuned cells when intracortical influences are suppressed, leaving only feed-forward influences [21]. In Sect. 2 we will investigate in greater detail a model of cortical direction selectivity based on convergent lagged and non-lagged input from the LGN. This model will serve as a working hypothesis throughout this and the following article.

It is a longstanding view that the development of spatiotemporal receptive field properties of simple cells is governed by an activity-dependent learning process that determines how the synapses are modified during the critical period shortly before and after birth. In the case of orientation selectivity, there exists only indirect evidence for this hypothesis. Many simple cells of cats already show both orientation and direction selectivity before eye-opening [29, 52, 58, 3, 5], and monkeys are born with mature orientation selectivity [30]. Thus, it has not been possible to test whether the emergence of orientation selectivity in these species is activity-dependent. In ferrets, the major development of orientation selectivity depends on neural activity, but again some orientation selectivity is present before experimental manipulations of activity have been possible [6]. It appears that visual experience cannot substantially modify the preferred orientations of cortical cells [59, 60, 23].[1] In the case of direction selectivity, the evidence for activity-dependent development appears stronger: direction selectivity can be abolished by strobe-rearing during the critical period, which abolishes motion from the visual environment, while responsiveness and orientation tuning remain largely intact [4, 31]. Both orientation and direction selectivity are sharpened by normal visual experience; for reviews see [52, 58, 22, 47].

Numerous models have been formulated to describe activity-dependent learning as a self-organization process according to a Hebbian rule [63, 37, 35, 36, 51, 67, 47, 65, 16, 17]. Most of these models have exclusively concentrated on the development of spatial properties of the receptive field, such as orientation selectivity. In particular, in the model of one of the present authors [47, 16], orientation selectivity develops through a competition between ON-center and OFF-center inputs. The development of orientation selective simple cells is determined mainly by the spatial correlations of the ON- and OFF-cell inputs from the LGN that converge onto the simple cell. The remaining authors of the present paper have developed a framework [65] for extending the spatial correlation-based approach [46, 48] to the learning of spatiotemporal receptive field properties through spatio*temporal* correlations.

In this article, we combine the spatial models of [47, 16] with the spatiotemporal model of [65] to study the joint emergence of direction *and* orientation selectivity through a competition between lagged and non-lagged ON- and OFF-inputs. A more abstract model that concentrates on the development of direction selectivity alone has concurrently been explored by Feidler et al. [17]. In the same way as the spatial correlation of the input activity plays a key role during the development of orientation selectivity, the temporal correlation of the input activity proves crucial for the development of direction selectivity.

The paper is organized as follows. In Sect. 2 we introduce the linear model that is used to describe the spatiotemporal response of a simple cell and investigate the context in which such a simple model describes neuronal behavior appropriately. The developmental model itself is then formulated in Sect. 3 and the correlation functions that drive the development are derived both for the period before and after eye-opening.

In a subsequent article [66] we will present simulation results of our model for different scenarios and compare them with experimental data. Furthermore, a simple analysis of the developmental equation will be performed.

2 Spatiotemporal receptive field properties of simple cells

One of the most accurate experimental methods to measure the structure of spatiotemporal receptive fields of simple cells is the reverse correlation technique [10, 15]. Small bright and dark bars are presented on a dimly lit screen within the receptive field of the cell one records from. As a result of such a measurement one obtains two three-dimensional response profiles, corresponding to bright and dark stimuli, with two spatial dimensions x and y and one temporal dimension t. An (x, y, t)-point of such a response profile represents the probability that the cell fires t ms after a small bright or dark bar has been flashed into the cell's receptive field at a position $\boldsymbol{x} = (x, y)$. The spatiotemporal receptive field is obtained by subtracting the two response profiles. The underlying assumption of this subtraction will be discussed later on. In a spatiotemporal receptive field, positive values denote bright-excitatory subregions, whereas dark-excitatory subregions are characterized by negative values. In order to facilitate the display of these spatiotemporal receptive fields, one normally integrates along the axis of preferred orientation.

Two types of spatiotemporal receptive fields can be observed experimentally, viz. spatiotemporally separable and non-separable receptive fields [10, 12]. In the first case the response function can be written as the product of a spatial and a temporal part, whereas in the second case this is not possible; for a schematic drawing see Fig.1.a. As was pointed out by Adelson and Bergen [1] and Watson and Ahumada [64], spatiotemporally *non*-separable receptive fields may give rise to a direction-selective response of the cell. An example is shown in Figs.1.b and c. If one assumes that a simple cell acts as a linear spatiotemporal filter, its response to rightward motion can be derived from Fig. 1. A bar oriented along the y-axis (that is perpendicular to the image plane) will evoke its strongest positive response after a longer latency for small x than for large x. If the bar moves to the right at a speed v that corresponds to the slope of the ON subregion in the x-t plane, the difference in latency is compensated and a short but strong response is evoked at the cell so that the membrane potential will be above threshold and the cell will fire. Formally, the response of the cell at a particular time can be obtained from Fig. 1 by integrating along a line of slope v that has its starting point on the lower edge of the receptive field (viz. $t = 0$) at the current position of the stimulus. For rightward motion such an integration line is parallel to the ON and OFF subregions of the spatiotemporal receptive field and one obtains strong responses when the stimulus crosses the receptive field; cf. Fig. 1.b. If the stimulus moves to the left, ON and OFF subregions cancel along the integration line and the cell's response will be weak.

What are the physiological mechanisms that underlie such a spatiotemporal receptive field structure? As was already

[1] Many early results suggested that a visual environment containing only one orientation would convert all cortical cells to preference for that orientation. However, later work showed that there did not appear to be more than the normal number of cells responsive to that orientation. What had changed was that the number of unresponsive cells increased, presumably because cells that had preferred other orientations were not stimulated and so became "sick" and lost responsiveness [59, 60]. Reviews and discussions of these results can be found in [52, 58, 22].

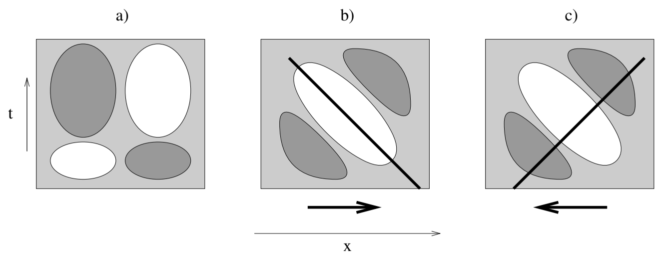

Fig. 1. Spatiotemporal receptive fields for one spatial dimension (axis of motion) and the temporal dimension are displayed schematically. A larger value of t corresponds to a larger time difference between the stimulus and the response, that is, to looking further into the past; cf. the argument of L^τ in Eq. (1). (a) A spatiotemporally *separable* receptive field can be described by the product of a spatial and a temporal response function. It responds in the same way to rightward and leftward motion. (b) and (c) A spatiotemporally *non-separable* receptive field that is most sensitive to rightward motion is shown. If a narrow bar oriented perpendicular to the image plane moves along the x-axis at a speed v, the response of the cell at a particular moment can be obtained from the figure by integrating along a line (the thick one) with slope v (towards the t-axis) that cuts the x-axis (viz. $t = 0$) at the current position of the stimulus. For a bar moving to the right, cell response is best if the (thick) integration line runs through the central ON subregion of the spatiotemporal receptive field corresponding to a current position of the stimulus on the right-hand side of the receptive field (cf. (b)). In (c) the stimulus moves to the left. In order to obtain the response of the cell at a particular time one has to integrate along a line of opposite slope as compared to (b). Since the (thick) line now crosses ON and OFF subregions and the sign of the receptive field always varies along such a line, the cell response is weak. For a similar plot cf. [25].

pointed out in the introduction, we assume that the convergence of four different types of spatiotemporal channels onto the cortical cell is responsible for the structure of the receptive field. These are ON- and OFF-type channels that come in two different temporal "flavors", namely, non-lagged and lagged. In contrast to the non-lagged channels, the lagged ones show early inhibition rather than excitation. The inhibition is followed by a delayed excitation that is weaker but broader as compared to the initial excitation of non-lagged inputs [57]. The spatiotemporal channels are modeled by the product of a spatial linear response function $R^c(\boldsymbol{\alpha}, \boldsymbol{\alpha}')$ and a temporal one, $L^\tau(t-t', \boldsymbol{\alpha})$, where the index c stands for ON- or OFF-inputs, the index τ for lagged or non-lagged inputs, and $\boldsymbol{\alpha}$ labels positions in the LGN. The product $R^c(\boldsymbol{\alpha}, \boldsymbol{\alpha}')L^\tau(t-t', \boldsymbol{\alpha})$ describes the response of a cell in the LGN to the activity of the photoreceptors. Such a description is in agreement with the experimental observation that LGN receptive fields are approximately spatiotemporally separable (ignoring the slower temporal response of surround relative to center) [12]. A spatiotemporal channel characterized by the two functions $R^c(\boldsymbol{\alpha}, \boldsymbol{\alpha}')$ and $L^\tau(t-t', \boldsymbol{\alpha})$ captures LGN responses and thus summarizes processing along the visual pathway from the photoreceptors up to the thalamo-cortical synapse. At the level of the cortex, these channels are summed and weighted by the feed-forward synapses $J^{c,\tau}(\boldsymbol{x}, \boldsymbol{\alpha})$ from the LGN to a simple cell at site $\boldsymbol{x}$ in the visual cortex. A simple cell also receives input from other cortical cells, weighted by intracortical synapses $B(\boldsymbol{x}, \boldsymbol{x}')$. For the sake of simplicity we assume that no additional time structure is introduced by the intracortical connections.

Summarizing, we obtain the following functional form for the local potential of a simple cell:

$$\begin{aligned} h(\boldsymbol{x}, t) = &\sum_{c=\mathrm{ON,OFF}} \sum_{\tau=\mathrm{nl,l}} \sum_{\boldsymbol{\alpha}} J^{c,\tau}(\boldsymbol{x}, \boldsymbol{\alpha}) \\ &\times \int_{-\infty}^{\infty} d\boldsymbol{\alpha}' \int_{-\infty}^{\infty} dt' R^c(\boldsymbol{\alpha}, \boldsymbol{\alpha}') L^\tau(t-t', \boldsymbol{\alpha}) S(\boldsymbol{\alpha}', t') \\ &+ \sum_{\boldsymbol{x}'} B(\boldsymbol{x}, \boldsymbol{x}') h(\boldsymbol{x}', t) \,. \end{aligned} \tag{1}$$

Here $S(\boldsymbol{\alpha}', t')$ denotes either the deviation of the stimulus intensity from a mean background luminance or the deviation of the photoreceptor activity from a mean noise level at a position $\boldsymbol{\alpha}'$. Therefore, S can take both positive and negative values. For example, during a reverse correlation experiment positive values of S denote a bright bar and negative values a black bar. The coordinate $\boldsymbol{\alpha}$ refers to the retinotopic position of the center of the receptive field of an LGN cell. Finally, $\boldsymbol{x}$ denotes the retinotopic position of a simple cell. It should be noted that the temporal part of the response function may also depend on the retinotopic position of the LGN cell. This is due to our assumption that the main time structure of our model is introduced by non-lagged and lagged cells in the LGN.

Since Eq. (1) is linear it can be rewritten

$$\begin{aligned} h(\boldsymbol{x}, t) = &\sum_{\boldsymbol{x}'} I(\boldsymbol{x}, \boldsymbol{x}') \sum_{c=\mathrm{ON,OFF}} \sum_{\tau=\mathrm{nl,l}} \sum_{\boldsymbol{\alpha}} J^{c,\tau}(\boldsymbol{x}', \boldsymbol{\alpha}) \\ &\times \int_{-\infty}^{\infty} d\boldsymbol{\alpha}' \int_{-\infty}^{\infty} dt' R^c(\boldsymbol{\alpha}, \boldsymbol{\alpha}') L^\tau(t-t', \boldsymbol{\alpha}) S(\boldsymbol{\alpha}', t') \end{aligned} \tag{2}$$

with

$$I(\boldsymbol{x}, \boldsymbol{x}') = \mathbf{1} + B(\boldsymbol{x}, \boldsymbol{x}') + B^2(\boldsymbol{x}, \boldsymbol{x}') + \ldots = [\mathbf{1} - B(\boldsymbol{x}, \boldsymbol{x}')]^{-1}. \tag{3}$$

For the last equation to hold, the eigenvalues of B must have absolute value less than one. The spatial kernel $R^c(\boldsymbol{\alpha}, \boldsymbol{\alpha}')$ is supposed to be shift invariant, that is $R^c(\boldsymbol{\alpha}, \boldsymbol{\alpha}') = R^c(\boldsymbol{\alpha} - \boldsymbol{\alpha}')$. The function $R^c(\boldsymbol{\alpha} - \boldsymbol{\alpha}')$ models ON-center and OFF-center receptive fields, and will be given by a Mexican hat function modeled as the difference of two Gaussians [56].

The functional form we have chosen for the temporal linear response function $L^\tau(t - t')$ for a non-lagged or lagged input is based on theoretical considerations by Dong and Attick [13]. From information-theoretic arguments they were able to derive the following power spectrum of the cell response,

$$L^\tau(\omega) L^\tau(\omega)^* = \frac{\omega^2}{[1 + (\omega/\omega_c)^2]^3}, \tag{4}$$

that agrees well with measurements of Saul and Humphrey [57] for a critical frequency $f_c = \omega_c/(2\pi) \approx 6$ Hz for non-lagged and $f_c \approx 4$ Hz for lagged cells. Because the power spectrum in Eq. (4) does not specify the phase of the Fourier transform of the response function, it does not determine a unique response function. However, if one imposes the additional constraint that the delay[2] introduced by the filter is minimal [39],

[2] By delay we mean the group delay of the filter, that is, $\partial \Phi(\omega)/\partial \omega|_{\omega=\omega_0}$ with $\Phi(\omega)$ being the phase of the Fourier transform of the filter and ω_0 being the peak frequency of the Fourier transform of a typical signal. The group delay is a measure for the temporal shift of the envelope of the signal that is introduced by the filter. Besides the requirement of a minimal group delay there are two additional formal requirements so that $L^{\mathrm{nl}}(\omega)$ is defined uniquely by its power spectrum. First, the filter function has to be stable, i.e., $\int_{-\infty}^{+\infty} |L^{\mathrm{nl}}(t)| \exp(-\epsilon|t|) dt < \infty$, and second, it has to be causal,

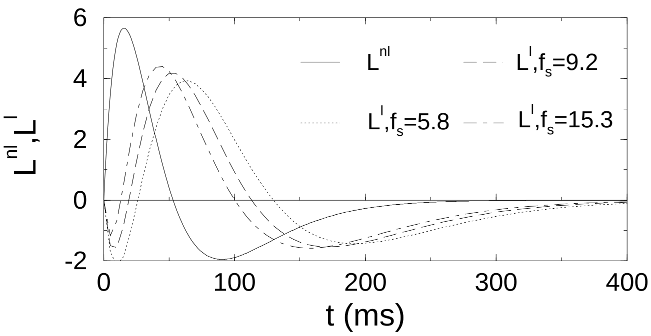

Fig. 2. The non-lagged temporal response function $L^{\mathrm{nl}}(t)$, and lagged response functions $L^{\mathrm{l}}(t)$ for the three values $f_s = 5.8$, 9.2, and 15.3 Hz, are plotted. The other parameter values are $f_c = 6$ Hz for non-lagged cells and $f_c = 4$ Hz for lagged cells. These values have been kept fixed throughout this paper.

one obtains a unique form for the Fourier transform of the temporal response function of non-lagged cells,

$$L^{\mathrm{nl}}(\omega) = \frac{\mathrm{i}\omega}{(1 + \mathrm{i}\omega/\omega_c)^3} \,. \tag{5}$$

For non-lagged cells the requirement of a minimal temporal delay seems to be a biologically plausible assumption. After an inverse Fourier transform the linear response of a non-lagged cell takes the form

$$L^{\mathrm{nl}}(t) = \begin{cases} \frac{1}{N_{\mathrm{nl}}} t(1 - \frac{1}{2}\omega_c t)\mathrm{e}^{-\omega_c t} & \text{for} \quad t \geq 0 \\ 0 & \text{for} \quad t < 0 \end{cases} \tag{6}$$

where the total power of the function L^{nl} is normalized to 1 by the pre-factor $1/N_{\mathrm{nl}}$. Figure 2 shows the response function, which is in good agreement with the linear response function derived experimentally by Saul and Humphrey [57]. The cell fires strongly during the first 50 ms and falls below the mean firing rate for about 300 ms thereafter.

According to measurements of Saul and Humphrey, the response of a lagged channel shows a markedly different form. A short drop in activity is followed by a more delayed and broader peak of the firing rate, as compared to the non-lagged case. The cell activity then falls below the mean firing rate again for another 300 ms. However, the power spectrum of a lagged cell's temporal response is well described by the same function as was found for the non-lagged case.

To obtain the lagged response function, we now, as a pure heuristic, multiply the Fourier transform of the non-lagged response function by $(1 - \mathrm{i}\omega/\omega_s)/(1 + \mathrm{i}\omega/\omega_s)$, to arrive at

$$L^{\mathrm{l}}(\omega) = \frac{\mathrm{i}\omega\,(1 - \mathrm{i}\omega/\omega_s)}{(1 + \mathrm{i}\omega/\omega_c)^3(1 + \mathrm{i}\omega/\omega_s)} \,. \tag{7}$$

In this way the form of the power spectrum (4) that is valid for both non-lagged and lagged cells remains unchanged[3]. The group delay of the response function, however, is increased as compared to the non-lagged one. By varying the shift frequency ω_s, the delay between the lagged and non-lagged response can be tuned. Performing the inverse Fourier transform, one obtains for $t \geq 0$

$$\begin{aligned} L^{\mathrm{l}}(t) = \operatorname{sgn}(\omega_s - \omega_c)\frac{1}{N_{\mathrm{l}}}\Big\{ & 2\omega_s^2(\mathrm{e}^{-\omega_s t} - \mathrm{e}^{-\omega_c t}) \\ & + \mathrm{e}^{-\omega_c t}\Big[t(\omega_s^3 + \omega_c^3 + \omega_s^2\omega_c - 3\omega_c^2\omega_s) \\ & - \frac{1}{2}t^2\omega_c(\omega_c + \omega_s)(\omega_s - \omega_c)^2\Big]\Big\} \end{aligned} \tag{8}$$

for $\omega_s \neq \omega_c$, and

$$L^{\mathrm{l}}(t) = \frac{1}{N_{\mathrm{l}}} t(-6 + 9\omega_c t - 2\omega_c^2 t^2)\mathrm{e}^{-\omega_c t} \tag{9}$$

for $\omega_s = \omega_c$, while $L^{\mathrm{l}}(t)$ vanishes for $t < 0$.

The functions L^{nl} and L^{l} have been plotted in Fig. 2, showing the form that is to be expected experimentally. The only parameter that has been varied systematically during the simulations is $f_s = \omega_s/(2\pi)$. By changing f_s, the group delay time of the system is modified. It will turn out that our model of linear response predicts that, for experimentally observed delays, the correlation between a lagged and a non-lagged channel is very weak.

The question of the degree to which a simple cell's responses can be characterized by a linear spatiotemporal filter has been subject to intense experimental investigation during the last few years. To the degree to which it can be so characterized, it should be possible to predict the response of the cell to a complicated stimulus like a stationary or a drifting sine wave grating from the reverse correlation measurements by simply convolving the input pattern with the spatiotemporal response function. As was shown by DeAngelis et al. [11], tuning curves for spatial and temporal frequency measured with sine-wave gratings agree well with linear predictions from reverse correlation measurements. Furthermore, experiments of Reid et al. [55], Albrecht and Geisler [2] and DeAngelis et al. [11] demonstrate that such a purely linear model accurately evaluates the preferred direction of motion of a simple cell. However, the direction selectivity index, which characterizes the difference in size of the responses for the preferred and the non-preferred direction, is underestimated by a linear model by a factor of 1/3 to 1/2.

It has been proposed by Heeger [25] and by Albrecht and Geisler [2] that a nonlinearity describing the spike generation mechanism can account for such discrepancies between the linear model and the experimental results. Membrane potentials are combined linearly in such a model, an idea that is supported by the intracellular recordings of Jagadeesh et al. [32].

One reason for the largely linear behavior of simple cells is that a simple cell is not only excited by a bright stimulus presented within the ON subregion of the receptive field, but is also inhibited by a dark stimulus within the ON subregion (and vice versa for OFF subregions) [19]. This makes it reasonable to subtract the spatiotemporal response profiles for a bright and a dark stimulus in a reverse correlation measurement, as described above.

In general, a linear model is always particularly appropriate if only small deviations from a mean value are considered. This is the case in our present study. When we describe the development of the receptive field structure before eye-opening, only small deviations from a mean synaptic strength level are considered, as will be sketched in the next section. Furthermore, the emerging receptive fields are compared with

i.e., $L^{\mathrm{nl}}(t) = 0$ for $t < 0$. These two requirements are full-filled for every "reasonable" filter function.

[3] Furthermore, $L^{\mathrm{l}}(\omega)$ is chosen in such a way that it is stable and causal; cf. the previous footnote.

reverse correlation measurements. These predict responses to small deviations from a mean luminance, again justifying the simplicity of our approach.

We now turn to a developmental model that aims at properly incorporating the effects of *time* in the learning equations.

3 A developmental model for spatiotemporal receptive fields

It is commonly assumed that activity-driven modifications of synapses take place according to some sort of Hebbian learning rule [24]. A Hebbian synapse is strengthened if the pre- and postsynaptic activity or depolarization are positively correlated, and is kept fixed or weakened for negative correlations.

A process of long-term potentiation involving NMDA receptors has been proposed [9, 8] as a possible mechanism that might underlie such development by correlated pre- and postsynaptic activity. Sprouting and retraction of axons and dendrites that are guided by neurotrophins could also follow a Hebbian principle, if the amount of neurotrophins available at a certain instant of time and the rate of cell growth are influenced by cell activity [62, 61].

In our model only thalamo-cortical synapses are modified by Hebbian learning, whereas intracortical synapses are kept fixed. Since we aim at describing the development of spatio*temporal* receptive fields we have to carefully consider possible temporal effects in our model. We assume that the change in synaptic strength at time t is determined by the correlation between the postsynaptic membrane potential $h(\boldsymbol{x}, t)$ according to Eq. (2) and the presynaptic cell's deviation from a mean firing rate. Then the correlation is integrated over a learning window that lasts for a time Λ so that we find [65]

$$\frac{dJ^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha},t)}{dt} = \frac{\lambda}{\Lambda}\int_0^{\Lambda} ds A(\boldsymbol{x}-\boldsymbol{\alpha})h(\boldsymbol{x},t-s) \qquad (10)$$
$$\times\left[\int_{-\infty}^{\infty}dt'\int_{-\infty}^{\infty}d\boldsymbol{\alpha}' R^c(\boldsymbol{\alpha}-\boldsymbol{\alpha}')L^{\tau}(t-s-t',\boldsymbol{\alpha})S(\boldsymbol{\alpha}',t')\right].$$

Here $A(\boldsymbol{x}-\boldsymbol{\alpha})$ denotes an arbor function that restricts the possible synaptic wirings that might emerge from the learning process. In particular, $A(\boldsymbol{x}-\boldsymbol{\alpha})$ indicates the number of synapses between a position $\boldsymbol{\alpha}$ in the LGN and a cortical position $\boldsymbol{x}$ that are modified during development. This mathematical framework can also embrace simple models involving sprouting and retraction guided by a Hebbian principle [50].

In Eq. (10) the presynaptic activity is characterized by the full spatiotemporal response function of the channel (nonlagged or lagged) that is linked to the cortical cell by the respective synapse. So the function $L^{\tau}(t,\boldsymbol{\alpha})$ that characterizes the temporal behavior of the different channels appears both in the presynaptic term and the postsynaptic membrane potential. The temporal response properties of the different channels therefore strongly influence the outcome of the learning process.

By inserting Eq. (2) into Eq. (10) we obtain the system of differential equations

$$\frac{dJ^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha},t)}{dt} = \lambda A(\boldsymbol{x}-\boldsymbol{\alpha})\sum_{\boldsymbol{x}'} I(\boldsymbol{x},\boldsymbol{x}') \qquad (11)$$
$$\times\sum_{c'=\mathrm{ON,OFF}}\ \sum_{\tau'=\mathrm{nl,l}}\ \sum_{\boldsymbol{\alpha}'} C^{c,c';\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}',t)J^{c',\tau'}(\boldsymbol{x}',\boldsymbol{\alpha}',t),$$

where $C^{c,c';\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}',t)$ denotes the correlation function

$$C^{c,c';\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}',t) =$$
$$\int_{-\infty}^{\infty}d\boldsymbol{\alpha}''\int_{-\infty}^{\infty}d\boldsymbol{\alpha}''' R^c(\boldsymbol{\alpha}-\boldsymbol{\alpha}'')R^{c'}(\boldsymbol{\alpha}'-\boldsymbol{\alpha}''')$$
$$\times\int_{-\infty}^{\infty}dt'\int_{-\infty}^{\infty}dt'' L^{\tau'}(t-t',\boldsymbol{\alpha})L^{\tau'}(t-t'',\boldsymbol{\alpha}')$$
$$\times\frac{1}{\Lambda}\int_0^{\Lambda} ds S(\boldsymbol{\alpha}'',t'-s)S(\boldsymbol{\alpha}''',t''-s). \qquad (12)$$

In the above equations, (11) and (12), we have made the additional assumption that $J^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha},t)$ changes slowly on the timescale of the learning window. In particular, we set $J^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha},t)$ constant within the learning window so that a separation of timescales is performed.

The term $1/\Lambda\int_0^{\Lambda} ds S(\boldsymbol{\alpha}'',t'-s)S(\boldsymbol{\alpha}''',t''-s)$ describes the correlation of the activity of the photoreceptors in space and time, averaged over a learning period Λ. We now consider two possible cases that might be crucial for the development of direction selectivity, and their corresponding correlation functions. In the first case uncorrelated noise in the photoreceptors drives the development, whereas in the second case development in an environment of moving gratings is considered.

Orientation and direction selectivity already develop before eye-opening [29, 52, 58, 3]. Correlations within the input activity $S(\boldsymbol{\alpha},t)$ during this period result from noise in the photoreceptors. One plausible assumption, and also the simplest one, is that a photoreceptor's deviation from the mean noise level is uncorrelated in space and time and fluctuates fast as compared to the time Λ. This gives

$$\frac{1}{\Lambda}\int_0^{\Lambda} ds S(\boldsymbol{\alpha}'',t'-s)S(\boldsymbol{\alpha}''',t''-s) = \delta(\boldsymbol{\alpha}'''-\boldsymbol{\alpha}'',t''-t'). \qquad (13)$$

Using Eq. (13), the expression for the correlation function (12) simplifies considerably, and we obtain

$$C^{c,c';\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}',t) = C^{c,c'}(\boldsymbol{\alpha},\boldsymbol{\alpha}')C^{\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}') \qquad (14)$$

with

$$C^{c,c'}(\boldsymbol{\alpha},\boldsymbol{\alpha}') = \int_{-\infty}^{\infty}d\boldsymbol{\alpha}'' R^c(\alpha-\alpha'')R^{c'}(\alpha'-\alpha''), \qquad (15)$$

$$C^{\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}') = \int_{-\infty}^{\infty}dt L^{\tau}(t,\boldsymbol{\alpha})L^{\tau'}(t,\boldsymbol{\alpha}'). \qquad (16)$$

The spatial part of the correlation function $C^{c,c'}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ is given by the convolution of two center-surround receptive fields and gives a Mexican-hat-type correlation function, as has already been pointed out by Miller [47]. For the sake of simplicity we model this correlation function by a difference of two Gaussians,

$$C^{\mathrm{ON,ON}}(\boldsymbol{\alpha},\boldsymbol{\alpha}') = C^{\mathrm{OFF,OFF}}(\boldsymbol{\alpha},\boldsymbol{\alpha}') = \qquad (17)$$
$$\exp\left(-\frac{|\boldsymbol{\alpha}-\boldsymbol{\alpha}'|^2}{\sigma_c^2}\right) - \frac{1}{\gamma_c^2}\exp\left(-\frac{|\boldsymbol{\alpha}-\boldsymbol{\alpha}'|^2}{\gamma_c^2\sigma_c^2}\right).$$

Furthermore, if $R^{\mathrm{ON}}(\boldsymbol{\alpha},\boldsymbol{\alpha}') = -R^{\mathrm{OFF}}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ it follows that

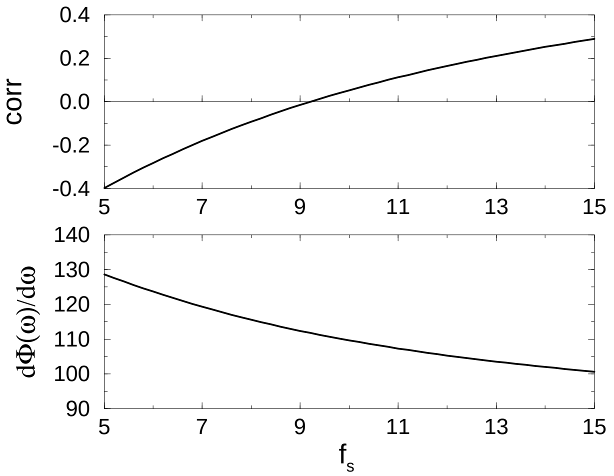

Fig. 3. The temporal correlation between non-lagged and lagged inputs $\mathrm{corr}(f_s)$ and the group delay time $\partial\Phi(\omega)/\partial\omega|_{\omega=\omega_{\mathrm{opt}}}$ of the lagged response function (in ms), for $\omega_{\mathrm{opt}} \approx 2\pi \times 2.8$ Hz (approximate peak of the power spectrum), have been plotted as a function of f_s.

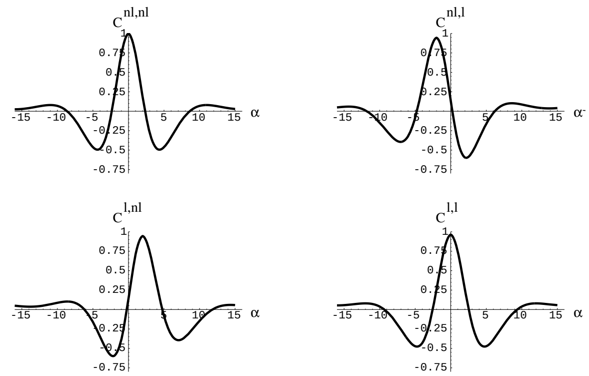

Fig. 4. The four correlation functions corresponding to different combinations of non-lagged and lagged inputs are displayed for the case of a narrow bar of light moving from left to right across the retina at a speed of 15°/s. The whole receptive field has a width of 3°. It should be noted that the correlation function is no longer rotationally symmetric as in the case of unstructured input; cf. Eq. (17). Along the α-axis one finds strong oscillations, as shown; these oscillations are smoother along the β-axis, perpendicular to the picture plane, so that excursions to negative values are small or totally missing along that axis. Note that the peaks of $C^{\mathrm{l,nl}}(\boldsymbol{\alpha}-\boldsymbol{\alpha}')$ and $C^{\mathrm{nl,l}}(\boldsymbol{\alpha}-\boldsymbol{\alpha}')$ are shifted in or opposite to the direction of motion, in contrast to the central peaks of $C^{\mathrm{nl,nl}}(\boldsymbol{\alpha}-\boldsymbol{\alpha}')$ and $C^{\mathrm{l,l}}(\boldsymbol{\alpha}-\boldsymbol{\alpha}')$.

$$C^{\mathrm{ON,OFF}}(\boldsymbol{\alpha},\boldsymbol{\alpha}') = C^{\mathrm{OFF,ON}}(\boldsymbol{\alpha},\boldsymbol{\alpha}') = -C^{\mathrm{ON,ON}}(\boldsymbol{\alpha},\boldsymbol{\alpha}')\ . \quad (18)$$

We have also performed simulations with $C^{\mathrm{ON,OFF}} = C^{\mathrm{OFF,ON}} = -0.5\, C^{\mathrm{ON,ON}}$, as used in most simulations of Miller [47]. The structure of the simulation results, however, has not been affected by this change of the correlation function.

According to Eq. (16), the temporal part of the correlation function $C^{\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ depends on the functional form of the temporal response function. In most of the simulations described in the following article [66] we make the simplifying assumption that $L^{\tau}(t,\boldsymbol{\alpha})$ does not depend on the position $\boldsymbol{\alpha}$ of the LGN neuron. Then $C^{\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ reduces to the 2×2 matrix

$$C^{\tau,\tau'} = \begin{pmatrix} C^{\mathrm{nl,nl}} & C^{\mathrm{nl,l}} \\ C^{\mathrm{l,nl}} & C^{\mathrm{l,l}} \end{pmatrix} = \begin{pmatrix} 1 & \mathrm{corr} \\ \mathrm{corr} & 1 \end{pmatrix}\ . \quad (19)$$

The diagonal elements are 1 because of the normalization of the temporal response functions. The only parameter that will be varied systematically during the simulations presented in [66] is f_s. It determines the form of the lagged response function and, in particular, the group delay time of the filter. Hence we obtain

$$\mathrm{corr}(f_s) = \int_{-\infty}^{\infty} dt L^{\mathrm{nl}}(t) L^{\mathrm{l}}(t,f_s)\,. \quad (20)$$

The dependence of the function $\mathrm{corr}(f_s)$ upon f_s and the group delay time of a lagged response function have been plotted in Fig. 3. The group delay time is given by $\partial\Phi(\omega)/\partial\omega|_{\omega=\omega_{\mathrm{opt}}}$ where $\Phi(\omega)$ is the phase of the Fourier transform of the lagged response function $L^{\mathrm{l}}(\omega)$ and $\omega_{\mathrm{opt}} \approx 2\pi \times 2.8$ Hz denotes the frequency where the power spectrum of the response of a lagged cell has its maximum.

If we vary f_s between 5 and 15 Hz, the passage time varies between 130 and 100 ms, which are typical delays for lagged cells according to Saul and Humphrey [57]. The function $\mathrm{corr}(f_s)$, on the other hand, increases from -0.4 to 0.3 and vanishes for $f_s = 9.2$ Hz.

It will turn out that for $\mathrm{corr}(f_s)$ near zero, that is, for weak correlations between lagged and non-lagged cells, strongly direction-selective receptive fields emerge, as observed experimentally.

We now turn to a second case and derive the correlation function that corresponds to an environment of moving gratings. Such a structured input might arise either from patterned vision after eye-opening or from spindle waves traversing the LGN during sleep [33, 44].

For simplicity we consider an infinitely narrow line/bar that moves into the α-direction at a speed v. For this case the activity of a photoreceptor at time t' and at point α'' is correlated with the activity of the photoreceptor at time t'' and at point $\alpha''' = \alpha'' + (t''-t')v$, since the bar has moved from α'' to α''' during the time interval $t''-t'$. Hence, the correlation of the activity of the photoreceptors takes the form

$$\frac{1}{A}\int_0^A ds S(\boldsymbol{\alpha}'',t'-s)S(\boldsymbol{\alpha}''',t''-s) = \delta(\alpha'''-\alpha''-(t''-t')v) \quad (21)$$

with $\boldsymbol{\alpha} = (\alpha,\beta)$.

Inserting (21) into (12) and performing the integration in Eq. (12) one obtains complicated functional forms for the four spatial correlation functions corresponding to the different combinations of non-lagged and lagged inputs. We do not exhibit these functions explicitly but display them in Fig. 4 for the case of $v = 15°$/s and a width of a receptive field of about 3°. While $C^{\mathrm{nl,nl}}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ and $C^{\mathrm{l,l}}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ have a central peak, the maximum of $C^{\mathrm{nl,l}}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ and $C^{\mathrm{l,nl}}(\boldsymbol{\alpha},\boldsymbol{\alpha}')$ are shifted in or opposite to the direction of motion.

The general case of bars with different directions of motion is either modeled by averaging the correlation function in Fig. 4 over different directions of motion or by rotating the correlation function during a simulation run.

We would like to stress that our approach of deriving the correlation functions from a model of linear response for the different channels can only serve as a kind of plausibility argument for the form of the correlation functions. It would be more desirable to obtain these functions directly from cross-correlation measurements in the LGN. There exists some experimental data about the correlations in activity between retinal ganglion cells [40, 41, 45] that are consistent with the form of the spatial correlation function as given in Eq. (17). However, data on temporal correlations between non-lagged and lagged cells in the LGN is not available yet.

The arbor function $A(\boldsymbol{x}-\boldsymbol{\alpha})$ and the intracortical interaction function $I(\boldsymbol{x}-\boldsymbol{x}')$ have been chosen in agreement with Miller [47]. Here $A(\boldsymbol{x}-\boldsymbol{\alpha})$ is proportional to the overlap in area of a circle of radius r_A and a circle of radius $c_A r_A$ with centers separated by $|\boldsymbol{x}-\boldsymbol{\alpha}|$. It is zero for $|\boldsymbol{x}-\boldsymbol{\alpha}| > D_A/2$, where D_A is the arbor diameter, with $r_A = (D_A - 1)/2$ and $c_A = 0.5$. The function $I(\boldsymbol{x}-\boldsymbol{x}')$ is a Mexican hat of the form

$$I(\boldsymbol{x}) = a(\boldsymbol{x}) \left[\exp\left(-\frac{|\boldsymbol{x}|^2}{\sigma_I^2}\right) - \frac{1}{\gamma_I^2} \exp\left(-\frac{|\boldsymbol{x}|^2}{\gamma_I^2\sigma_I^2}\right) \right]. \qquad (22)$$

As mentioned above only the parameter f_s that determines the form of the lagged response is varied systematically in the simulations described in the second article [66]. The other parameters have been chosen in such a way [47] that stable orientation selectivity emerges for the purely spatial case. In particular, for the case of a noise-driven development we assign the values $\sigma_c = r_c D_A/2$ with $r_c = 0.25$ and $\gamma_c = 3$ to the parameters of the spatial correlation function. For the scenario in which moving gratings determine the development we choose a speed of $v = 15°/\mathrm{s}$ for the patterns moving across the retina. Furthermore, we take for the parameters of the intracortical interaction function the values $\sigma_I = r_I D_A/2$ with $r_I = 0.25$ or $r_I = 0.4$, $\gamma_I = 3$, and $a(0)$=1 for $\boldsymbol{x} = 0$ and $a(\boldsymbol{x}) = 0.5$ otherwise. So the width of both the correlation function and the intracortical interaction function scale with the diameter of the arbor function. For a more detailed discussion of the different parameter values see Miller [47].

If synapses are modified according to Eq. (11), they will grow or decrease without bound [49]. To avoid such biologically implausible behavior, upper and lower bounds of the couplings have been introduced. Since all afferent thalamocortical couplings are assumed to be excitatory, we set

$$0 \le J^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha}) \le J_{\max} A(\boldsymbol{x}-\boldsymbol{\alpha}) \qquad (23)$$

with a default value $J_{\max} = 4$. Moreover, Eq. (11) is completed by a constraint that ensures that the sum of the synaptic weights received by one cortical cell is kept fixed. In this way a competitive process between afferent axons is modeled.

The full learning equation then takes the form

$$\begin{aligned}\frac{dJ^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha},t)}{dt} &= \lambda A(\boldsymbol{x}-\boldsymbol{\alpha}) \sum_{\boldsymbol{x}'} I(\boldsymbol{x},\boldsymbol{x}') \\ &\quad \times \sum_{c'}\sum_{\tau'}\sum_{\boldsymbol{\alpha}''} C^{c,c';\tau,\tau'}(\boldsymbol{\alpha},\boldsymbol{\alpha}'') J^{c',\tau'}(\boldsymbol{x}',\boldsymbol{\alpha}'',t) \\ &\quad - \epsilon(\boldsymbol{x}) A(\boldsymbol{x}-\boldsymbol{\alpha}), \end{aligned} \qquad (24)$$

where

$$\epsilon(\boldsymbol{x}) = \frac{\left[\sum_{\boldsymbol{\alpha}'} \sum_{c=\mathrm{ON,OFF}} \sum_{\tau=\mathrm{nl,l}} \frac{d}{dt}\big|_{\mathrm{u}} J^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha}',t)\right]}{\sum_{\boldsymbol{\alpha}',c,\tau} A^{c,\tau}(\boldsymbol{x}-\boldsymbol{\alpha}')} \qquad (25)$$

and $\frac{d}{dt}\big|_{\mathrm{u}} J^{c,\tau}(\boldsymbol{x},\boldsymbol{\alpha}')$ is given by Eq. (11). Here $A^{c',\tau'}(\boldsymbol{x}-\boldsymbol{\alpha}'') = A(\boldsymbol{x}-\boldsymbol{\alpha})$ for all c' and τ' (the arbor function is identical for all input types); explicitly noting the input type will be useful when we consider inactivation of synapses that reach the maximum or minimum allowed values. The effects of the constraint on the dynamics of the learning equation have been discussed in great detail elsewhere [49].

4 Outlook

In a subsequent paper [66] we study the developmental model derived above in numerical simulations and present a first analysis of the model. We consider different scenarios that cover the development both before and after eye-opening. A thorough discussion of our model and a comparison with experimental data will also be presented there.

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