

Mean-field theory of a neural network

(visual cortex/synaptic modification)

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ABSTRACT A single-cell theory for the development of selectivity and ocular dominance in visual cortex has been generalized to incorporate more realistic neural networks that approximate the actual anatomy of small regions of cortex. In particular, we have analyzed a network consisting of excitatory and inhibitory cells, both of which may receive information from the lateral geniculate nucleus (LGN) and then interact through cortico-cortical synapses in a mean-field approximation. Our investigation of the evolution of a cell in this mean-field network indicates that many of the results on existence and stability of fixed points that have been obtained previously in the single-cell theory can be successfully generalized here. We can, in addition, make explicit further statements concerning the independent effects of excitatory and inhibitory neurons on selectivity and ocular dominance. For example, shutting off inhibitory cells lessens selectivity and alters ocular dominance (masked synapses). These inhibitory cells may be selective, but there is no theoretical necessity that they be so. Further, the intercortical inhibitory synapses do not have to be very responsive to visual experience. Most of the learning process can occur among the excitatory LGN-cortical synapses.

A single cell theory for the development of selectivity and ocular dominance in visual cortex has been presented previously by Bienenstock, Cooper, and Munro (1). This has been extended to a network applicable to layer 4 of visual cortex (2). In this paper we present a mean field approximation that captures in a fairly transparent manner the qualitative, and many of the quantitative, results of the network theory.

Visual cortex has been extensively investigated (3, 4). We summarize some of the dominating experimental facts very briefly. Neurons in the primary visual cortex of normal adult cats are sharply tuned for the orientation of an elongated slit of light and most are activated by stimulation of either eye. Both of these properties—orientation selectivity and binocularity—depend on the type of visual environment experienced during a critical period of early postnatal development extending from approximately 3 weeks to 3 months. For example, deprivation of patterned input during this critical period leads to loss of orientation selectivity, while monocular deprivation (MD) results in a dramatic shift in the ocular dominance of cortical neurons such that most will be responsive exclusively to the open eye. The ocular dominance shift after MD is the best known and most intensively studied type of visual cortical plasticity. The consequences of binocular deprivation (BD) on visual cortex stand in striking contrast to those observed after MD. While 7 days of MD during the second postnatal month leave few neurons in striate cortex responsive to stimulation of the deprived eye, most cells remain responsive to visual stimulation through either eye after a comparable period of BD. However, prolonged periods of BD lead to a loss of orientation selectivity, an effect

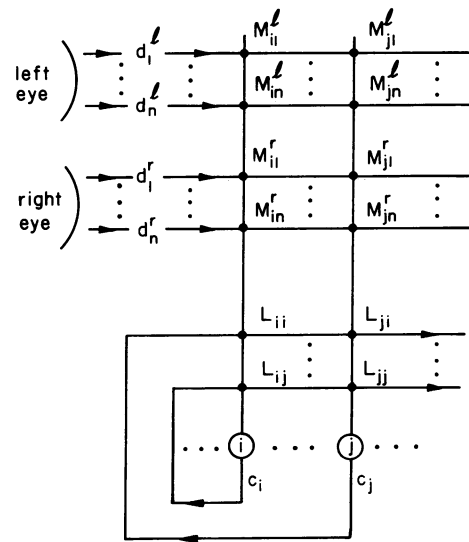


FIG. 1. Network with inputs from left and right eyes, with LGN-cortical and cortico-cortical synapses.

not observed in the response to the open eye after comparable periods of MD. The theory we discuss is concerned primarily with the explanation of these and related facts.

Definitions and Notation

We focus attention on the input from the lateral geniculate nucleus (LGN) and intercortical interactions. Inputs from other regions of cortex are considered part of a background excitation or inhibition contributing to the spontaneous activity of the cell. In addition, the various time delays that result in structure in the poststimulus time histogram are assumed to be integrated over periods of the order of a second for purposes of synaptic modification. This leads to a circuit as shown in Fig. 1.

The output of the cells of the full network can be written

$$c = c^*(Md + Lc), \quad [1]$$

where c^* is a sigmoidal response function,

$$c = (c_1 \dots c_N)^T, \quad [2a]$$

c_i is the output firing rate of the i th cortical cell, and

$$M = (M_{is}^l, M_{is}^r), \quad [2b]$$

Abbreviations: MD, monocular deprivation; BD, binocular deprivation; LGN, lateral geniculate nucleus; GABA, γ -aminobutyric acid. †Present address: Nestor, Inc., 1 Richmond Square, Providence, RI 02906.

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where M_{is}^l and M_{is}^r are the s th LGN "synapses" from the left and right eye to the i th cortical cell.

$$d = (d^l, d^r)^T \text{ and } d^{l,r} = (d_1^{l(r)} \dots d_n^{l(r)})^T \quad [2c]$$

are the time-averaged inputs from the left and right eye as described in ref. 1,

$$L = (L_{ij}) \quad [2d]$$

is the matrix of cortico-cortical synapses, and L_{ij} is the synapse from the j th cell to the i th cell. (Notice that italicized symbols always contain left and right eye components.)

In the monotonically increasing region above threshold and below saturation, in a linear approximation,

$$c = Md + Lc. \quad [3]$$

We consider a region of cortex for which the neural mapping of the input from the visual field is constant (all of the cells, in effect, look at a given region of the visual field). Under these conditions, for an input, d , constant in time, the equilibrium state of the network would be

$$c = (1 - L)^{-1}Md. \quad [4]$$

Equilibrium as well as nonequilibrium information may be critical to the evolution of the network as well as of primary importance in information processing.[‡]

Mean-Field Approximation

For a given LGN-cortical vector of synapses, m_i (the i th row of M), and for a given input from both eyes, d , Eq. 3 for the firing rate of the i th cortical cell becomes

$$c_i = m_i d + \sum_j L_{ij} c_j, \quad [5]$$

where the first term is due to the input from LGN and the second due to input from other cortical cells. We define \bar{c} as the spatially averaged firing rate of all of the cortical cells in the region defined above:

$$\bar{c} = (1/N) \sum_i c_i. \quad [6]$$

The mean-field approximation is obtained by replacing c_j in the sum in Eq. 5 by its average value so that c_i becomes

$$c_i = m_i d + \bar{c} \sum_j L_{ij}. \quad [7]$$

Here, in a manner similar to that in the theory of magnetism, we have replaced the effect of individual cortical cells by their average effect (as though all other cortical cells can be replaced by an "effective" cell). It follows that

$$\bar{c} = \bar{m}d + \bar{c}L_0 = (1 - L_0)^{-1}\bar{m}d, \quad [7a]$$

[‡]If we expand $(1 - L)^{-1}$, we obtain $(1 - L)^{-1} = 1 + L + L^2 + \dots$, an expansion in mono-, di-, tri-, . . . synaptic events. How many synaptic events one includes depends on the time interval of importance. For synaptic modification we assume that time intervals of the order of a half second are appropriate. Thus c represents the average over about $\frac{1}{2}$ sec of the number of spikes that are the result of an external presentation (5). The poststimulus time histogram can be broken into much smaller time intervals, thus separating mono-, di-, trisynaptic events and excitatory and inhibitory synapses.

where

$$\bar{m} = (1/N) \sum_i m_i \quad [8]$$

and

$$L_0 = (1/N) \sum_{ij} L_{ij},$$

so that

$$c_i = \left(m_i + \left(\sum_j L_{ij} \right) (1 - L_0)^{-1} \bar{m} \right) d.$$

If we assume that the lateral connection strengths are a function only of $i-j$ (not dependent on the absolute position of a cell in the network, therefore dependent only on the distance of two cells from one another), L_{ij} becomes a circular matrix so that

$$\sum_i L_{ij} = \sum_j L_{ij} = L_0 = \text{constant} \quad [9]$$

and

$$c_i = (m_i + L_0(1 - L_0)^{-1}\bar{m})d. \quad [10]$$

In the mean-field approximation we can therefore write

$$c_i(\alpha) = (m_i - \alpha)d = (m_i^l - \alpha^l) \cdot d^l + (m_i^r - \alpha^r) \cdot d^r, \quad [11]$$

where the mean field

$$\alpha = (\alpha^l, \alpha^r) = -a(\bar{m}^l, \bar{m}^r) \quad [12]$$

with

$$a = |L_0|(1 + |L_0|)^{-1}, \quad [12a]$$

and we assume that $L_0 < 0$ (the network is, on average, inhibitory).[§]

The Cortical Network

The behavior of visual cortical cells in animals reared in various conditions suggests that some cells respond more rapidly to environmental changes than others. In MD, for example, some cells remain responsive to the closed eye in spite of the very large shift of most cells to the open eye. Singer (6) found, using intracellular recording, that geniculo-cortical synapses on inhibitory interneurons are more resistant to MD than are synapses on pyramidal cell dendrites. In dark rearing some cells become nonresponsive to visual stimuli, while most cells retain some responsiveness (3, 4). Recent work suggests that the density of inhibitory GABAergic synapses (GABA, γ -aminobutyric acid) in kitten striate cortex is also unaffected by MD during the critical period (7, 8).

These results suggest that some LGN-cortical synapses modify rapidly, while others modify relatively slowly, with slow modification of some cortico-cortical synapses. Excitatory LGN-cortical synapses onto excitatory cells may be those that modify primarily. (Since these synapses are formed exclusively on dendritic spines, this raises the possi-

[§]The average magnitude of cortico-cortical inhibition, L_0 , must be smaller than 1. Otherwise \bar{c} would be smaller than 0 (Eq. 7a). There would be so much inhibition that on average no cells would fire.

bility that the mechanisms underlying synaptic modification exist primarily in axo-spinous synapses.) To embody these facts we introduce two types of LGN-cortical synapses: those (m_i) that modify (according to the modification rule discussed in ref. 1) and those (z_k) that remain relatively constant. In a simple limit we have

$$\dot{m}_i = \phi(c_i, \bar{c}_i)d$$

and [13]

$$\dot{z}_k = 0.$$

(In what follows \bar{c} denotes the spatial average over cortical cells, while \bar{c}_i denotes the time-averaged activity of the i th cortical cell.) The function ϕ is discussed in ref. 1. We assume for simplicity, and consistent with the above physiological interpretation, that these two types of synapses are confined to two different classes of cells and that both left and right eye have similar synapses (both m_i or both z_k) on a given cell. We therefore can write

$$c_i = m_i d + \sum_j L_{ij} c_j$$

and [14]

$$c_k = z_k d + \sum_j L_{kj} c_j.$$

Further, in what follows, we assume for maximum simplicity that there is no modification of cortico-cortical synapses, although what experimental results there are suggest only that modification of inhibitory cortico-cortical synapses is slow (7, 8). The consequences of a theory including cortico-cortical synapse modification for the full network were briefly discussed in ref. 1 and will be discussed more fully in the mean-field approximation elsewhere.

In a cortical network with modifiable and nonmodifiable LGN-cortical synapses and nonmodifiable cortico-cortical synapses, the synaptic evolution equations become

$$\dot{m}_i = \phi(c_i, \bar{c}_i)d,$$

$$\dot{z}_k = 0,$$

and [15]

$$\dot{L}_{ij} = 0.$$

This leads to a very complex set of coupled nonlinear stochastic evolution equations that have been simulated and partially analyzed elsewhere (2). The mean-field approximation permits dramatic simplification of these equations, leading to analytic results and a fairly transparent understanding of their consequences in various conditions. In this approximation Eqs. 14 become

$$c_i = m_i d + L_0 \bar{c}$$

and [16]

$$c_k = z_k d + L_0 \bar{c},$$

so that we can now write

$$c_i(\alpha) = (m_i - \alpha)d = (m_i^l - \alpha^l) \cdot d^l + (m_i^r - \alpha^r) \cdot d^r$$

and [17]

$$c_k(\alpha) = (z_k - \alpha)d = (z_k^l - \alpha^l) \cdot d^l + (z_k^r - \alpha^r) \cdot d^r,$$

where now $\alpha^{l(r)}$ contains terms from modifiable and nonmodifiable synapses:

$$\alpha^{l(r)} = a(\bar{m}^{l(r)} + \bar{z}^{l(r)}),$$

$$\bar{m}^{l(r)} = N^{-1} \sum_{i=1}^{N_m} m_i^{l(r)}, \quad [18]$$

$$\bar{z}^{l(r)} = N^{-1} \sum_{k=1}^{N_{nm}} z_k^{l(r)}$$

and a is defined in Eq. 12a. ($N = N_m + N_{nm}$, where N_m is the number of cells with modifiable synapses and N_{nm} is the number of cells with nonmodifiable synapses.) Since it is assumed that neither L nor z changes as the network evolves, only $\bar{m}^{l(r)}$ is time dependent.

Position and Stability of Fixed Points of LGN-Cortical Synapses in the Mean-Field Network

We now generalize the arguments given in refs. 1 and 9 for the position and stability of the fixed points of the stochastic nonlinear synaptic modification equations. In the mean-field network

$$\dot{m}_i(\alpha) = \phi(c_i(\alpha), \bar{c}_i(\alpha))d = \phi[m_i(\alpha) - \alpha]d, \quad [19]$$

where $c_i(\alpha)$ is defined by Eq. 17 and $\bar{c}_i(\alpha)$ is an average of the form

$$\bar{c}_i(\alpha) = \tau \int_{-\infty}^t \exp[(t' - t)\tau^{-1}] c_i(\alpha, t') dt'. \quad [20]$$

The mean field, $\alpha^{l(r)}$ as given by Eq. 12, has a time-dependent component $\bar{m}^{l(r)}$. This varies as the average over all of the network modifiable synapses and, in most environmental situations, should change slowly compared to the change of the modifiable synapses to a single cell:

$$|\dot{\bar{m}}^{l(r)}| \ll |\dot{m}_i^{l(r)}|. \quad [21]$$

We, therefore, define an adiabatic approximation in which we assume that α is slowly varying and determine the trajectory of m_i for fixed α . (We imagine that m_i reaches its fixed point before α varies. The nonadiabatic situation is analyzed in the Appendix. It is shown there that, in any case, the position and stability of the fixed points are unaltered.) In the adiabatic approximation we can write

$$(m_i(\alpha) - \alpha) = \phi[m_i(\alpha) - \alpha]d. \quad [22]$$

We see that there is a mapping

$$m_i^l \leftrightarrow m_i(\alpha) - \alpha \quad [23]$$

such that for every $m_i(\alpha)$ there exists a corresponding (mapped) point m_i^l that satisfies

$$\dot{m}_i^l = \phi[m_i^l]d, \quad [24]$$

the original equation for the mean-field zero theory. Therefore, if we start from the corresponding initial point

$$m_i^l(t_0) = m_i(\alpha, t_0) - \alpha, \quad [25]$$

the m_i' trajectory viewed from the m_i coordinate system is the trajectory of $m_i(\alpha = 0)$. Thus, we can compute $m_i(\alpha)$ from the $\alpha = 0$ trajectory, using

$$m_i(\alpha) = m_i' + \alpha = m_i(\alpha = 0) + \alpha. \quad [26]$$

The transformation

$$m_i'' = m_i + \alpha \quad [27]$$

gives a coordinate system whose origin is displaced from the mean-field zero coordinates by α . The trajectory of a solution of the $\alpha = 0$ theory measured from this coordinate system gives a solution of the $\alpha \neq 0$ theory for the corresponding point:

$$m_i''(\alpha) = m_i'(\alpha) + \alpha = m_i(0) + \alpha = m_i(\alpha). \quad [28]$$

It follows that at corresponding points

$$c_i(\alpha) = c_i(0)$$

and [29]

$$\bar{c}_i(\alpha) = c_i(0),$$

so that the modification threshold, θ_M , is unaltered in this mapping.[†] When this argument is applied to the fixed points, we conclude that for every fixed point of $m_i(\alpha = 0)$ there exists a corresponding fixed point for $m_i(\alpha)$ with the same selectivity and stability properties. Therefore, just as for the $\alpha = 0$ theory, for arbitrary α only selective fixed points are stable. Further, at corresponding fixed points we obtain the same cell output.

From this we see that if the background inhibition is changed (e.g., by long-term application of bicuculine or a GABA agonist) and the LGN-cortical synapses are allowed to evolve to the new fixed points in the same visual environment, the outputs of the cortical cells will evolve to what they were before the background inhibition was altered. (It is presumed that a cortical cell does not jump from one stable fixed point to another in this process.)

The above is limited as follows:

(i) The LGN-cortical synapses are restricted to be positive (excitatory). Therefore, if α is too small (insufficient background inhibition), $m_i(\alpha)$ will not be able to reach its fixed points with only positive components.

(ii) The LGN-cortical synapses cannot increase beyond some physiological and/or molecular limit. Therefore, if α is too large, the cell will never fire, thus restricting the evolution of $m_i(\alpha)$.

Evolution of the Mean-Field Network Under Various Rearing Conditions

We are now in a position to calculate the evolution of cortical cells under various rearing conditions. In what follows we give as one example the evolution of cortical cells in the mean-field network under MD conditions. The argument is similar to that given in ref. 1. A more detailed analysis including comparisons with experiment will be presented elsewhere (E. E. Clothiaux, M. F. Bear, and L.N.C., unpublished).

[†]For simplicity we often compute \bar{c}_i as an average over the environment $\{d^1 \dots d^K\}$. Thus, for example, for the monocular case, at a selective fixed point $(m_i - \alpha) \cdot d^1 = \theta_M$ (preferred input), $(m_i - \alpha) \cdot d^j = 0$ ($j > 1$, nonpreferred inputs), so that $\bar{c}_i = (1/K) \sum_j (m_i - \alpha) \cdot d^j = \theta_M/K$. With the definition $\theta_M = (\bar{c}_i)^2$ we obtain $\bar{c}_i = K$ so that $\theta_M = K^2$ independent of the mean field, α .

Under MD conditions, the animal is reared with one eye closed. For the sake of analysis assume that the right eye is closed and that only noiselike signals arrive at cortex from the right eye. Then the environment of the cortical cells is

$$d = (d^j, n)^T. \quad [30]$$

Further, assume that the left eye synapses have reached their selective fixed point, selective to pattern d^1 . Then $(m_i^l, m_i^r) = (m_i^{l*}, x_i)$ with $|x_i| \ll |m_i^{l*}|$. For the preferred open eye pattern (d^1, n) we have $c_i(\alpha) = \theta_M + (x_i - \alpha^r) \cdot n$, while for the nonpreferred open eye patterns (d^j, n) , $j > 1$, $c_i(\alpha) = (x_i - \alpha^r) \cdot n$. Following the argument of ref. 1, a time average over the full pattern environment gives

$$\dot{x}_i = -\kappa(x_i - \alpha^r) \text{ with } \kappa \text{ a positive number.} \quad [31]$$

For a constant or slowly varying mean field this leads to an asymptotic solution for the fixed point:

$$x_i = \alpha^r = a(\bar{x} + \bar{z}^r). \quad [32]$$

We see that

$$x_i^*(\alpha^r) = x_i^*(0) + \alpha^r = \alpha^r, \quad [33]$$

as expected from the above general argument. If we now include the self-consistency condition that is a consequence of the variation of the mean field and use

$$\bar{x} = (1/N) \sum_{i=1}^{N_m} x_i, \quad [34]$$

we obtain

$$\bar{x} = \lambda a(1 - \lambda a)^{-1} \bar{z}^r, \quad [35]$$

where $\lambda = N_m/N$ is the ratio of the number of modifiable cells to the total number of cells in the network. This yields

$$x_i^* = a(1 - \lambda a)^{-1} \bar{z}^r. \quad [36]$$

That is, the asymptotic state of the closed eye synapses is a scaled function of the mean field due to nonmodifiable cortical cells. The scale of this state is set not only by the proportion of nonmodifiable cells but in addition by the averaged intracortical synaptic strength L .

Thus, contrasted with the mean-field zero theory, the deprived eye LGN-cortical synapses do not go to zero. Rather they approach the constant value dependent on the average inhibition produced by the nonmodifiable cells in such a way that the asymptotic output of the cortical cell is zero (it cannot be driven by the deprived eye). However, lessening the effect of inhibitory synapses (e.g., by application of bicuculine) reduces the magnitude of α so that one could once more obtain a response from the deprived eye.

Discussion

Having defined a mean-field approximation that greatly simplifies the equations for the response and evolution of cortical cells, we have obtained a fundamental result: the stability and position of the fixed points in this network are related to the fixed points in the absence of mean field ($\alpha^{(r)} = 0$) by

$$m_i^*(\alpha) = m_i^*(0) + \alpha, \quad [37]$$

where $m_i^*(\alpha)$ is a fixed point of Eq. 22 in the mean field α ,

while $m_i^*(0)$ is a fixed point of this equation of zero mean field.

Thus if $m_i^*(\alpha)$ is restricted to the first quadrant (positive values for all of its components due to the excitatory nature of LGN-cortical synapses), as long as α is large enough and nonspecific (there is sufficient inhibition for all pattern inputs), $m_i(\alpha)$ can still reach all of the fixed points that would have been reached by $m_i(0)$ (not restricted to the first quadrant). This means that if network inhibition is sufficient, the selective stable fixed points can be reached even though LGN-cortical synapses are excitatory. Once reached, the fixed points, $m_i^*(\alpha)$, have the same stability characteristics as the corresponding $m_i^*(0)$.

We find, consistent with previous theory and with experiment, that most learning can occur in the LGN-cortical synapses; inhibitory (cortico-cortical) synapses need not modify. Some nonmodifiable LGN-cortical synapses are required. It becomes interesting to ask whether these could be associated with some anatomical feature (e.g., might these be synapses into shafts rather than spines).

As in the zero mean-field theory, zero cell output is an unstable fixed point. Thus in BD the cell output could be on average above or below spontaneous activity (depending on the level of inhibition). Some "nonvisual" cells would reappear if excitation were enhanced or inhibition diminished.

In MD the closed eye response goes to

$$c = (x - \alpha) \cdot d \rightarrow 0. \quad [38]$$

Therefore, LGN-cortical synapses do not go to 0. Rather

$$x \rightarrow \alpha. \quad [39]$$

Thus if inhibition is suppressed one would expect some response from the closed eye. This is in agreement with experiment.

Various models for memory storage and retrieval have been suggested. These differ in several ways. One of the most important from the point of view of computational complexity as well as for realization in silicon is the degree of connectivity of each unit. What is suggested here is that much that is significant in at least one layer of visual cortex can be obtained in a primarily feed-forward network of very simplified lateral connectivity. The original connectivity in which each of the N neurons in this layer of cortex is connected to every other [N^2 connectivity] can be replaced by a mean-field network in which a neuron receives n LGN inputs and a single (mean-field) input [$(n + 1)$ connectivity].

Appendix: Asymptotic Behavior of Mean-Field Equations with Time-Dependent Mean Field

From Eqs. 19 and 23, the trajectory of the corresponding

point is

$$\dot{m}_i' = \phi[m_i']d - \dot{\alpha}. \quad [A1]$$

Using Eq. 18, we have

$$\dot{\alpha} = a \frac{\dot{m}}{\bar{m}(\alpha)} = a(1 - a)^{-1} \dot{\bar{m}}', \quad [A2]$$

so that

$$\dot{m}_i' = \phi[m_i']d - a(1 - a)^{-1} \dot{\bar{m}}'. \quad [A3]$$

At the fixed points $\dot{m}_i' = 0$. When all of the cells of the network have reached their respective fixed points $\dot{m}_i' = 0$ for each cell. Therefore, $\dot{\bar{m}}' = 0$. It follows that when the network has stabilized at a global fixed point

$$\phi[m_i']d = 0 \quad [A4]$$

for all inputs. This is the same condition as the $\dot{\alpha} = 0$ (adiabatic) case. Thus the position and stability of the fixed points are the same as those in the adiabatic theory. However, since $0 < (1 - a) < 1$, the absolute value of average movement of the entire network towards the fixed points

$$|\dot{\bar{m}}'| = (1 - a) \overline{\phi[m_i']d} \quad [A5]$$

is slower than in the adiabatic theory.

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