ELSEVIER

BioSystems 48 (1998) 215–222

BioSystems

On the role of self-excitation in the development of topographic order in the visual system of the frog

Zoltán Somogyvári [a,b,*], Attila Andai [a,b], György Székely [c], Péter Érdi [a]

[a] *Department of Biophysics, KFKI Research Institute for Particle and Nuclear Physics, Hungarian Academy of Sciences, P.O.B. 49, 1525 Budapest, Hungary*

[b] *Eötvös Loránd University, Múzeum krt. 4–8, 1088 Budapest, Hungary*

[c] *Department of Anatomy, University Medical School, P.O.B. 12, 4012 Debrecen, Hungary*

Abstract

A two-level model is presented for explaining the development of the topographically ordered retinotectal connections of the visual system of the frog. The columnar structure of the tectum were taken into account explicitly. Simulations suggest that the self-excitatory intracolumnar connections play an important role in the formation of the topography. In the case of self-excitation the system is able to recognize the topology of the visual field by the continuity of the object's movement. © 1998 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Topographic order; Neural development; Self-excitation

---

1. Introduction

The frog's visual system is a prototype of organized neural structures. It is a multilayered structure composed of retina, lateral geniculate complex, pretectal nuclear complex, tectum opticum and nucleus isthmi. The mappings between the layers are topographic, i.e. preserve neighborhood (at least in large scale).

There is a long-lasting debate on the origin of the ordered connections related to the concept of neural specificity (Székely, 1990). According to Sperry's classic idea (Sperry, 1943), reinforced by Attardi and Sperry (1963), there is a specific point-to-point projection directed by gradients of chemical marker molecules between the retina and the tectum and the neural order derives from the interaction between the pre- and postsynaptic cells. The regeneration and plasticity experiments offered a new insight into the formation of retinotectal connections and suggested that the principle of the survival of the topographic mapping between the layers are stronger than that which prescribes the individual connections between a pre- and postsynaptic cell.

We have been trying to model the development and plasticity of the whole visual system of the

---

[*] Corresponding author.

PII S0303-2647(98)00068-9

frog. In spite of many efforts, there are still a series of problems to be solved, such as the generation of the anisotropy (Somogyvári et al., 1998), the mechanism of the conversion of temporal order to spatial order, the formation of the topography between all subsequent layers, etc.

Here we restrict ourselves to the problem of the formation of topography in the case of complex postsynaptic target. More precisely, in case of the retinotectal connections, it is generally assumed that the target structure is an array of single tectal cells. However, it is more realistic to take into account explicitly the internal network structure of the tectum. Functionally, in the tectum both recurrent excitatory and inhibitory connections exist. The specific question to be investigated here is the role of the recurrent (self-) excitation in the development of the topographic order. Is there any optimal value of self-excitation, which promotes the formation of topographic retinotectal connections?

2. Neurobiological background

In frogs, the main visual center is the tectum opticum. Retinotectal connections are the prototype of the topographically ordered maps which preserve information between subsequent layers of cells. The regeneration and plasticity experiments—such as expansion and compression, when a whole retina projects onto a surgically formed half retina or when a half retina's inervation expands across a whole tectum (Jacobson, 1978)—show that these mappings implement more the continuous mappings in a mathematical sense, than the more strict, point-to-point addressing. Based on a large number of experiments, the development of the highly ordered afferent pathway of the tectum opticum seems to be activity-dependent.

Activity-dependent mechanisms mainly express their effects via the modification of synaptic strengths, due to the temporal correlation between the pre- and postsynaptic sides. In addition, the spatial correlation of activities within the input layer is indispensable to form ordered (and not diffuse) projections. (Kalil and Dubin, 1988)

The spatiotemporal correlation in the input layer originates from two sources. First, the spatial correlation of the natural images is reflected on the retina. The natural objects are generally continuous: the closer the two points in the space the larger the similarity between the light intensities. Second, the movement of objects is always continuous and this yields higher temporal correlation between activities of the neighboring cells than that of the farther ones. Even without incoming stimulus, spontaneously generated activity waves travel across the retina and provide correlation. Consequently, the activity of axons arriving to it's target area carries information about it's neighborhood. This feature can provide a possibility to establish topographically ordered projections, if the postsynaptic structure as receiving system can recognize and make use of it.

Is the spatial correlation enough for the generalization of refinement of topographical projection? Or, can a local neural circuit transform the temporal order to spatial order, thus improving the precision of the projection?

2.1. The receiver system: the tectum opticum

The tectum itself is a multilayered structure and functionally has a columnar organization. The main cellular elements of a tectal column are primarily the large pyramidal cells as the only output point of the system, two types of the large pear-shaped neurons 'a' and 'b', the small pear-shaped neurons and stellates as representatives of the inhibitory cells. The 'a' type large pear-shaped neurons receive optical afferent input and by inervating each other by recurrent connections, a reverberating circuit is formed. The ascending axons of the 'a' type large pear-shaped cells excites the small pear-shaped neurons, which feedback the impulses to the basal dendrites of the large pear-shaped neurons. Thus a second reverberating circuit is formed. The 'b' type cells receive non-optical afferent fibers on their basal dendrites and send dendritic collaterals to the synaptic sites between dendritic appendages and optic terminals, providing inter-columnar connections. Finally the large pyramidal cells sum up the activation and inhibition (Székely and Lázár, 1976).

3. Short summary of previous models

Numerous mathematical models have been introduced to describe the mechanism of the formation of such ordered neural mappings (e.g. Willshaw and van der Malsburg, 1979; Amari, 1980; Gierer, 1981; Whitelaw and Cowan, 1981; Amari, 1982; Kohonen, 1982; Overton and Arbib, 1982; Érdi and Barna, 1984; Bienenstock, 1985; Cottrell and Fort, 1986; Ritter and Schulten, 1986; Obermayer et al., 1995). Some models are based on marker theories, while others implement activity-dependent mechanisms [reviewed by Linsker (1988)]. In the system-matching models [arrow model, Hope et al. (1976); branch arrow model, Overton and Arbib (1982); extended branch arrow model, Overton and Arbib (1982)] the information available for the individual retina fibers is less specific than in other genetically determined models.

In the marker theories, growing axons are directed to the appropriate point by the aid of some guiding substance. The activity-dependent mechanisms can be formulated in terms of modifiable synapses in consequence of correlated electrical patterns. In all the previous models, only the spatial correlation of the input was taken into consideration. The Whitelaw and Cowan (1981) model was an attempt to integrate marker- and activity-dependent algorithms, by combining a gradient of adhesive specificity with a synaptic updating rule used by Willshaw and van der Malsburg (1979). The model describes plasticity, mismatch, rotation, and compound eye experiments. For a brief review of these models, in the context of self-organization (Érdi and Szentágothai, 1985) and development of the nervous systems, see Arbib et al. (1997), Section 4.4.

4. The model

4.1. Representation

The retina has been represented with a one dimensional chain of cells. The tectum opticum has been considered as another one dimensional chain of lumped tectal column. The state of a tectal column is represented with the output activity of the column as its normalized instantaneous firing frequency—a real number between zero and one. The stimulus spreads from the retina to the tectum through the potentially entire connection weight matrix between these two layers. The strength of a connection is represented with an other real number between zero and one. In the tectal layer, neighboring columns are connected to each other via collateral excitation and inhibition, according to the ‘Mexican hat’ function.

4.2. Dynamics

A two-level phenomenological dynamics is adopted, where only the effect of various cellular elements of the column to the output activity are taken into consideration and the internal variables are neglected. A discrete time continuous state space model has been defined.

4.2.1. Activity dynamics

The activity of a retinal cell is induced by visual stimulus. A piecewise linear model has been used. A tectal column sums up the presynaptic excitation given by retinal cells multiplied by the synaptic weight. This value, mapped by the response function of the primary cell, results in the preliminary activity of this column. The role of self-excitation—performed by large pear-shaped cells created reverberating loops—is taken into consideration by the first term of the following expression. This yields that the effect of a stimulus does not disappear at the moment of the end of the stimulus, but decays exponentially.

$$b_j^*(t+1) = c_1 \cdot b_j(t) + c_2 \cdot \alpha\left[\sum_{i=1}^{N} W_{ij}(t) \cdot a_i(t) - f_j(t)\right]$$

where: c_1 controls the strength of the recurrent excitation; $c_2 = 0.9$; a_i is the activity state of a retinal cell; $b_j(t)$ is the former output activity state of the tectal column; b_j^* is the intrinsic activity state of the tectal column; f_j is the threshold of the primary cell; W_{ij} is the connection weight matrix;

$N = 30$ is the number of retinal cells; $\alpha(x)$ is the response function of the primary cell. This function has a relatively sharp threshold and saturation as the upper limit of the firing frequency,

$$\alpha(x) = c_3 \cdot \mathrm{th}(x) + c_3 \quad \text{if } x < 0$$

$$\alpha(x) = c_4 \cdot \mathrm{th}(x) + c_3 \quad \text{if } x \geq 0$$

Where $c_3 = 0.1$, $c_4 = 1$.

The role of the recurrent inhibition—made by the stellate cells—was considered as changes of the response function's threshold. This negative feedback results in the exponential convergence of the threshold to the time-weighted average of received activity of the tectal column. Time-weighting is defined in such a way that the more recent values are more important. This mechanism ensures that a column always works in its most sensitive range. Where $c_5 = 0.9$,

$$f_j(t+1) = c_5 \cdot f_j(t) + (1 - c_5) \cdot \sum_{i=1}^{N} W_{ij}(t) \cdot a_i(t)$$

Finally, two other effects modify the activity of a column. The collateral excitation from the neighboring columns increase and the inhibition from the more distant ones decrease the activity of the column, according to a 'Mexican Hat' function. These procedures yield the final output activity $b(t+1)$ of the column, in the next time step.

$$b_k(t+1) = \frac{\sum_{l=1}^{M} c_7 \cdot H_{kl} \cdot b_l^*(t+1)}{\sqrt{\sum_{k,l=0}^{M} H_{kl}^2}} + c_6 \cdot b_k^*(t+1)$$

$$H_{kl} = \cos\,[c_8 \cdot \mathrm{abs}(k - l)] \cdot \mathrm{e}^{-c_9 \cdot \mathrm{abs}(k - l)}$$

$M = 30$ is the number of tectal columns, $c_8 = N/4$, $c_9 = 2N$, $c_7 = 1$, $c_6 = 10$, and H_{kl} is the 'Mexican Hat' function.

4.2.2. Connection dynamics

The synaptic weights change according to an extended Hebbian-like learning rule which consists of three terms:

1. The original Hebb term (Hebb, 1949) , which expresses the correlation between the activity states of the pre- and postsynaptic units.
2. A non-linear selective decreasing term.
3. A correlation term between the changes of the two activity states in time. The synaptic weights of the collateral connections are assumed to be not modifiable.

The first term simulates the effect of the LTP performed by NMDA receptors, the second term expresses the effect of the LTD and the third term comes from the general property, that the neural system is sensitive for changes.

$$\mathrm{d}W_{ij}^*(t) = \varepsilon_1 \cdot a_i(t) \cdot b_j(t) - \varepsilon_2 \cdot [a_i(t) - b_j(t)]^2 + \varepsilon_3 \cdot [a_i(t) - a_i(t-1)] \cdot [b_j(t) - b_j(t-1)]$$

$$W_{ij}(t+1) = W_{ij}(t) + c_{10} \cdot W_{ij}(t) \cdot \mathrm{d}W_{ij}^*(t) \quad \text{if } \mathrm{d}W_{ij}^*(t) \leq 0$$

$$W_{ij}(t+1) = W_{ij}(t) + c_{10} \cdot [1 - W_{ij}(t)] \cdot \mathrm{d}W_{ij}^*(t) \quad \text{if } \mathrm{d}W_{ij}^*(t) \geq 0$$

Where $\varepsilon_1 = 1$, $\varepsilon_2 = 0.1$, $\varepsilon_3 = 0.1$, $c_{10} = 0.1$.

4.3. Simulation results

A topology measure was defined as follows:

$$G = \sum_{i,j=1}^{N-1} \frac{\sum_{k=1}^{M} W_{ij} \cdot W_{i+1,k} \cdot H_{jk}}{\sum_{l=1}^{M} W_{i+1,l} \cdot \sum_{e=1}^{N-1} W_{ie}}$$

The highest possible value is $G = 1$.

It is known that the freshly arrived axons generate non-specific dense and large arborizations. Therefore the experiments start with randomly chosen values of connection weights, from the [0.1, 0.3] interval, with uniform distribution.

When non-structured, random visual stimuli were presented to the retina, topographical projections failed to develop, the measure remained at a low value. These simulation results are in accordance with those physiological experiments, which show that in tadpoles growing up in a stroboscopic light, the projections developed don't show topographic order. Travelling activity

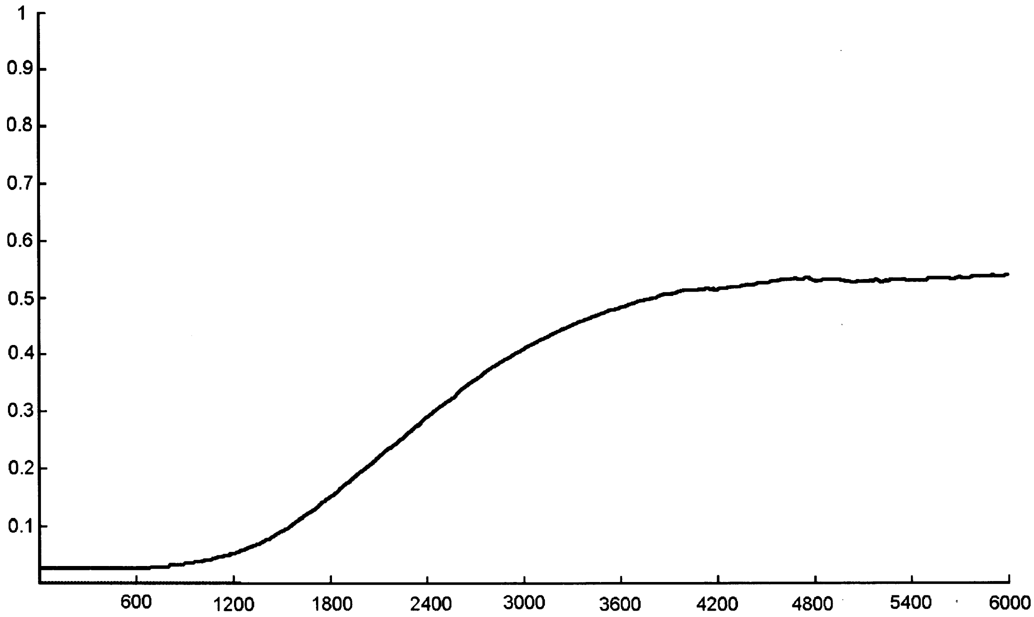


Fig. 1. The development of retinotopy during 6000 iterations. $c_1 = 0.5$.

waves, however, imply the formation of topographically ordered connections.

The effect of self-excitation is most clearly visible, when the spatial correlation of the input is cleaned off. Thus a simple visual stimulus was used: one retinal cell was active, while the others were in silence. This active point ran through the retina at a speed of one cell/timestep. The direction of the movement was randomly chosen. The temporal evolution of the measure is shown (Fig. 1).

Some stages of the development may be discriminated. During the first stage of the simulation the measure of topology increases quadratically. This stage can be identified, as the period of the extinction of those axons, which makes synapses on 'wrong' places. This simulated behavior corresponds to observed developmental patterns. The originally diffuse projection sharpens parallel with a considerable extinction of axonal branches. In the stage of the linear increase, the survived connections become stronger. During the last stage, the degree of topographic order is converging to it's limit. Finally it reaches it's limit value—in this case 0.5—after a few thousands of iterations. A typical synaptic matrix of the normally developed retinotectal connections is shown (Fig. 2).

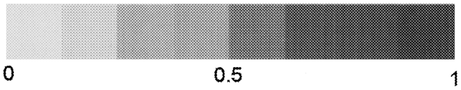
Both the velocity of the development and the maximal value of the measuring number are influenced by the numerical values of about ten parameters. Specifically, here the effect of the measure of the self-excitation is examined, while the other parameter values were fixed at some reasonable values.

Fig. 5 summarizes the effect of the self-excitation. The results show that, at low values of self-excitation, the topography also remains at low value. The projection fields of retinal cells became sharp enough, but they were randomly dispersed all across the tectum (Fig. 3). While the self-excitation and the characteristic length of the 'trace' of moving stimuli increases, the projection fields starts to agglutinate (Fig. 4). The measure of topography, which expresses the continuity and the sharpness of the projection became higher. At $c_1 = 0.5$, it reaches its highest value. After this peak, the recurrent excitation hinders the response of the system for the new stimulus, so the topography falls steeply.

5. Conclusions

A two-level network model was given to describe the normal development of retinotectal connections. The main deviation of the present model from the previous ones is that it explicitly takes into account both the inter- and intra-columnar effects at the tectal level. The simulations showed that self-excitation has an important role in the formation of retinotopy and the system gains the capacity to use the continuity feature of movement of visible objects. There is an optimal value for the development of topographic order.

The recurrent inhibitory mechanism interacts with the self-excitation. The cooperation and competition between these mechanisms is the subject of further studies. A special learning rule was constructed to describe synaptic modifiability. The effect of the parameters of the learning rule for the topographic order will be discussed elsewhere.



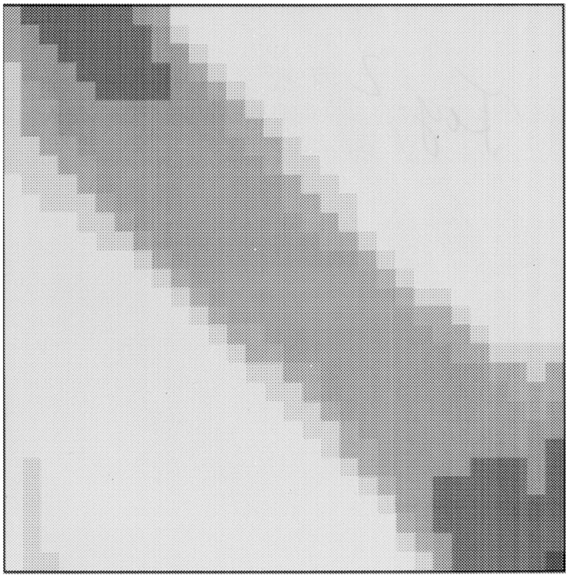


Fig. 2. A well developed connection matrix, $c_1 = 0.5$. The darker tone of gray stands for the higher the synaptic weight in figure. The diagonal shape of strong connections mean that the neighboring cells project into neighboring cells, so the topography is preserved.

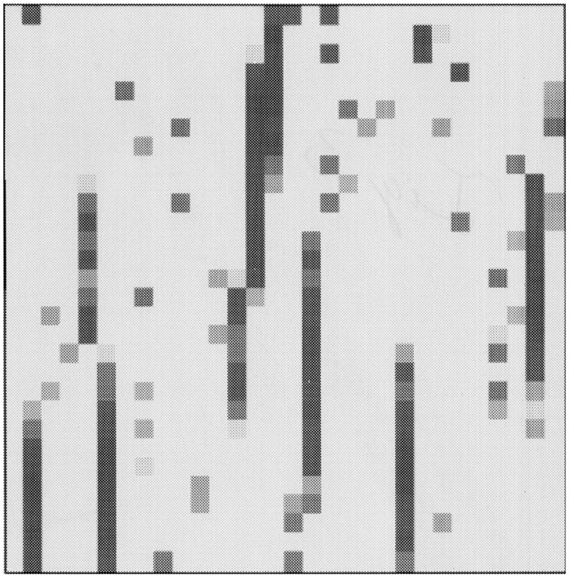


Fig. 3. The topography fails to develop, $c_1 = 0.025$.

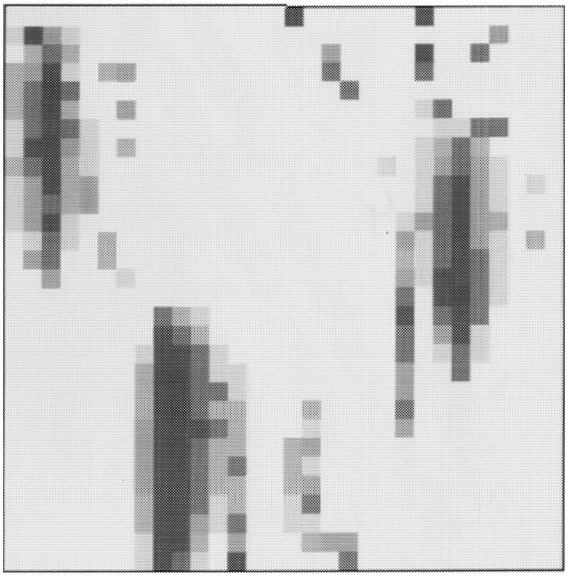


Fig. 4. The projection patches start to aggregate, $c_1 = 0.25$.

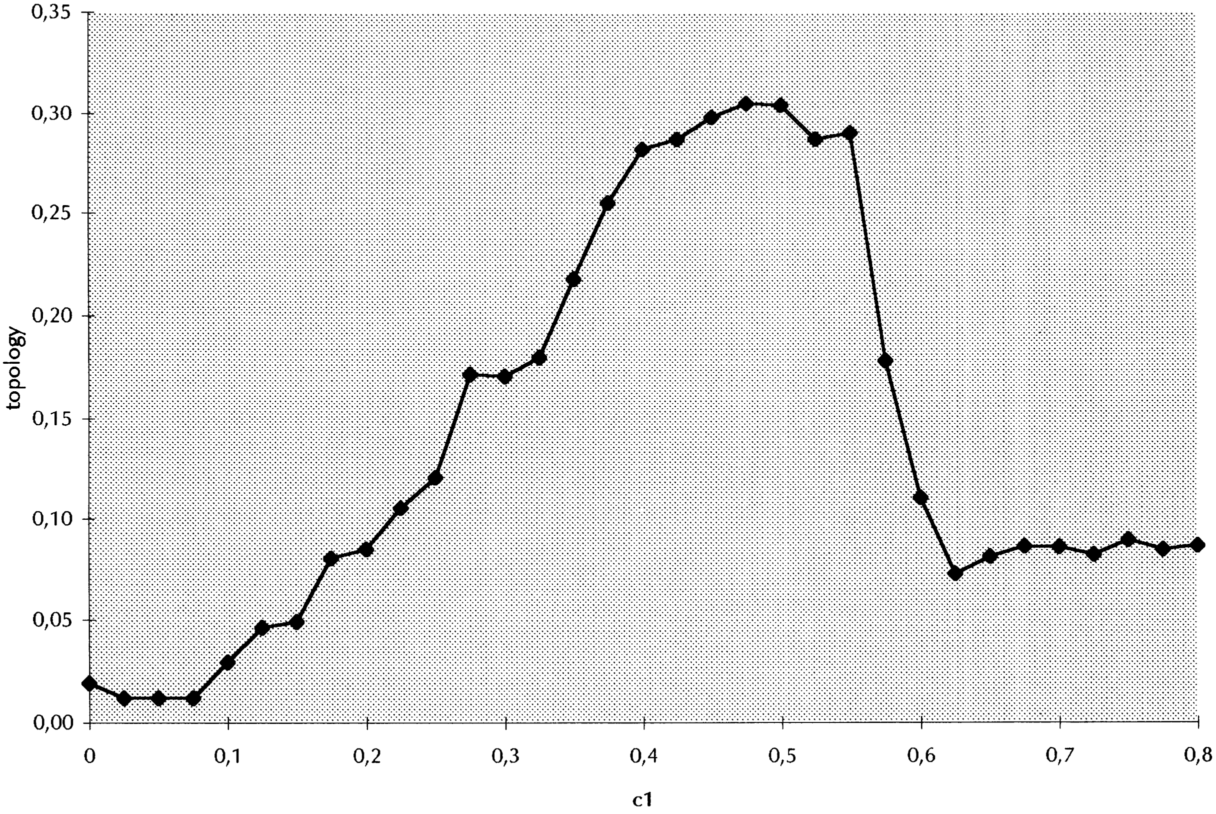


Fig. 5. The limit value of the measure of topographic order exponentially extrapolated from 6000 iteration. Each point shows the average of five of these limit values in accordance with varying strength of self-excitation. Self-excitation enables the system to be built up on the continuity of object movement in the visual field, improves the topography for a while, but in the case of too large values it suppresses the sensitivity of the receiving system.

Acknowledgements

The researches were partially supported by the Hungarian Scientific Research Fund, under grant no. T 017784, and by the Fogarty International Research Collaboration Award (FIRCA), HHS grant no. 1 R03 TW00485-01.

References

Amari, S., 1980. Topographic organization of nerve fields. Bull. Math. Biol. 42, 339–364.

Amari, S., 1982, Competitive and cooperative aspects in dynamics of neural excitation and self-organization, in: Competition and Cooperation in Neural Nets: Lecture Notes in Biomathematics, vol. 45, S. Amari and M.A. Arbib (eds.) (Springer, Berlin, Heidelberg, New York) pp. 202–225.

Arbib, M.A., Érdi, P. and Szentágothai, J., 1997, Neural Organization: Structure, Function and Dynamics, A Bradford Book (MIT Press, Cambridge MA).

Attardi, D.G., Sperry, R.W., 1963. Preferencial selection of central pathways by regenerating optic fibers. Exp. Neurol. 7, 46.

Bienenstock, E., 1985, Dynamics of the central nervous system, in: Dynamics of Macrosystems: Lecture Notes in Economics and Mathematical Systems, J.P. Aubin, D. Saari and K. Sigmund (eds.) (Springer, Berlin) pp. 3–20.

Cottrell, M., Fort, J.C., 1986. A stochastic model of retinotopy: a self organizing process. Biol. Cybern. 53, 405–411.

Érdi, P., Barna, G., 1984. Self-organizing mechanism for the formation of ordered neural mappings. Biol. Cybern. 51, 93–101.

Érdi, P., Szentágothai, J., 1985, Neural connectivities: between determinism and randomness, in: Dynamics of Macrosystems, J.P. Aubin, D. Saari and K. Sigmund (eds.) (Springer, Berlin, Heidelberg, New York) pp. 21–29.

Gierer, A., 1981). Development of projections between areas of the nervous system. Biol. Cybern. 42, 69–78.

Hebb, D.O., 1949, The First Stage of Perception: Growth of the Assembly. The Organization of Behaviour: A Neuropsychological Theory (Wiley, New York).

Hope, R.A., Hammond, B.J., Gaze, F.R.S., 1976. The arrow model: retinotectal specificity and map formation in the goldfish visual system. Proc. R. Soc. Lond. B 194, 447–466.

Jacobson, M., 1978, Developmental Neurobiology (Plenum Press, New York).

Kohonen, T., 1982. Self-organized formation of topologically correct feature maps. Biol. Cybern. 43, 59–69.

Linsker, R., 1988. Self-organization in a perceptual network. Comput. Mag. 21, 105–117.

Obermayer, K., Sejnowski, T., Blasdel, G.G., 1995. Neural pattern formation via a competitive Hebbian mechanism. Behav. Brain. Res. 66, 151–160.

Overton, K.J. and Arbib, M.A., 1982, System-matching and topographic maps: the branch-arrow model (BAM), in: Competition and Cooperation in Neural Nets: Lecture Notes in Biomathematics, vol. 45, S. Amari, M.A. Arbib (eds.) (Springer, Berlin, Heidelberg, New York) pp. 202–225.

Ritter, H., Schulten, K., 1986. On the stationary state of Kohonen's self-organizing sensory mapping. Biol. Cybern. 54, 99–106.

Kalil, R.E. and Dubin, M.W., 1988, The role of action potentials in the morphological development of retinogeniculate connections in the cat, In: Bentiquolo, M., Spreafico, R. (Eds), Cellular Thalamic Mechanisms. Elsevier Science.

Somogyvári, Z., Andai, A., Székely, Gy. and Érdi, P., 1998, A self-organising model of the ontogeny of frog's visual system: the anisotropical development, in: Cybernetics and Systems '98, R. Trappl (ed.), Austrian Society for Cybernetic Studies, Wien, pp. 317–322.

Sperry, R.W., 1943. Effect of 180° rotation of the retinal field of visuomotor coordination. J. Exp. Zool. 92, 263–279.

Székely, Gy. and Lázár, Gy., 1976, Cellular and synaptic architecture of the optic tectum, in: Frog Neurobiology, R. Llias and W. Precht (eds.) (Springer Verlag, New York) pp. 407–434.

Székely, Gy., 1990. Problems of the neuronal specificity concept in the development of neural organization. Concept Neurosci. 1 (2), 165–197.

Willshaw, D.J., van der Malsburg, C., 1979. A marker induction mechanism for the establishment of ordered neural mappings: its application to the retinotectal problem. Phil. Trans. R. Soc. Lond. B 287, 203–243.

Whitelaw, V.A., Cowan, F.D., 1981. Specificity and plasticity of retinotectal connections: a computational model. J. Neurosci. 1, 1369–1387.