J. Math. Biol. (1993) 31: 771–795

Journal of
Mathematical
Biology

An n-Level field theory of biological neural networks

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Received 16 December 1991; received in revised form 27 April 1992

Abstract. An n-level field theory, based on the concept of "*functional interaction*", is proposed for a description of the continuous dynamics of biological neural networks. A "*functional interaction*" describes the action from one substructure of a network to another at several levels of organization, molecular, synaptic, and neural. Because of the continuous representation of neurons and synapses, which constitute a hierarchical system, it is shown that the property of *non-locality* leads to a non-local field operator in the field equations. In a hierarchical continuous system, the *finite* velocity of the functional interaction at the lower level implies non-locality at the higher level. Two other properties of the functional interaction are introduced in the formulation: the *non-symmetry* between sources and sinks, and the *non-uniformity* of the medium. Thus, it is shown that: (i) The coupling between topology and geometry can be introduced *via* two functions, the *density* of neurons at the neuronal level of organization, and the *density-connectivity* of synapses between two points of the neural space at the synaptic level of organization. With densities chosen as Dirac functions at regularly spaced points, the dynamics of a discrete network becomes a particular case of the n-level field theory. (ii) The dynamics at each of the molecular and synaptic lower level are introduced, at the next upper level, both in the source and in the non-local interaction of the field to integrate the dynamics at the neural level. (iii) New *learning rules* are deduced from the structure of the field equations: Hebbian rules result from strictly local activation; non-Hebbian rules result from homosynaptic activation with strict heterosynaptic effects, i.e., when an activated synaptic pathway affects the efficacy of a non-activated one; non-Hebbian rules and/or non-linearities result from the structure of the interaction operator and/or the internal biochemical kinetics.

Key words: Field theory – Hierarchical neural networks – Non-local effects – Learning rules – Heterosynaptic effects

1 Introduction

A method is proposed to describe the dynamics of neural networks when three dependent biological constraints are considered: several levels of organization

(including the molecular level), geometrical densities of neurons and synapses, and non-local interactions between biological substructures.

The dynamics of formal neural networks are generally described with algebraic (Kohonen 1972, 1978) or statistical physics (Hopfield 1982, 1984) methods, and are based on two properties of their elements: the spatial summation according to a given topological connectivity, and learning rules which impose a modification of synaptic weights according to a correlation principle such as Hebb's postulate (Hebb 1949). Moreover, formal networks frequently are based on automata theory, due to the small number of assumed neuron states and the ease of implementating the learning algorithm on digital computers. For example, Hopfield networks, which are very similar to spin glass models in statistical physics (Toulouse et al. 1982, Peretto 1984), are based on a principle of optimization of an energy function with a sigmoid input-output relation. When this relation is steep, i.e., when the circuits are assumed to have a "high-gain" limit, network elements have one of two states, 0 or 1, and the energy function exhibits strong non-linearities. These conditions lead network states to attract minima in the space of configurations. Because such a process implies memorization, i.e., the possibility of convergence towards such attractors, it may be analogous to learning.

Biological neural networks present specific constraints (Crick 1989) which have to be considered in any study of the function of the network. Most of these constraints come from the organization of the system into several levels. From the molecular level, which is concerned with the dynamics of neurotransmitters, receptors and cytoplasmic neuromediators, to the level of groups of neurons, which generally constitute nuclei with a common physiological function, all the dynamics at each level are integrated to provide the function of the whole system.

Although physical models of memory have been proposed (e.g., Longuet-Higgins 1968), one important property in neural networks is the modifiability of their connections. Hebb (1949) hypothesized that stable long-term memory results from an increased synaptic efficacy, due to some structural change in biological neural networks. Many examples of such changes in synaptic function, e.g., associative learning at the behavioral level (Gingrich et al. 1987), associative long-term potentiation at hippocampal synapses (Levy et al. 1979, Kelso et al. 1986); learning and memory of movements in cerebellar cortex (Thompson 1986), have not been documented; and have been incorporated within models of perceptual learning and pavlovian conditioning (Grossberg 1967, 1982, 1990), of hippocampus (Traub et al. 1985), and of cerebellum (Albus 1971, Marr 1969, Fujita 1982). Interpretation of this postulate in terms of modern neurophysiology led to the concept of the Hebbian synapse, in which the strength is enhanced during nearly *simultaneous* pre- and postsynaptic activity. Brown et al. (1990) define an Hebbian synapse "as one that uses a time-dependent, highly local, and strongly interactive mechanism to increase synaptic efficacy as a function of the conjunction or correlation between pre- and postsynaptic activity". Such a principle of conjunction, and its extension toward a more general principle of covariance (Sejnowski 1977, Chauvet 1986), can be described as a macroscopic statistical principle, which does not *explicitly* include known cellular and molecular mechanisms. A first problem is to know how to deduce a quantitative formulation of the variation of synaptic efficacy from known cellular and molecular mechanisms (Gingrich et al. 1987), i.e., a formulation that integrates several levels of organization (Chauvet 1988b).

A second important property of biological neural networks is the large number of neurons and the large number of synapses per neuron. A continuous approach is therefore consistent with the continuous geometrical location of neurons and synapses, which is known from the experimental space densities of neurons and density-connectivities of synapses. Geometry with the space density function, and topology with the space connectivity function are then included in the formulation of the dynamics.

A consequence of this continuous approach with several levels of organization is the special formulation of physiological properties that depend, at each level of organization, on the geometrical location of the neurons and of the synapses for each neuron. It will be shown that these properties can be described with a non-local interaction operator as a consequence of the concept of non-locality in biological systems (Chauvet, 1993a). (i) Local interactions occur in an infinitesimally small space interval during an infinitesimal time interval, and the processes are described with local PDE (see, e.g., Murray 1990). They result from local phenomena at a given level as, e.g., the propagation of potentials (active propagation along the axonal membrane (Hodgkin et al. 1952); or the passive propagation with an attenuation factor along the dendritic membrane (Rall et al. 1973)), at the level of one neuron. (ii) Non-local interactions result from phenomena at a non-infinitesimal distance at different levels of organization. It will be shown that the existence of non-local effects appears logically through the formulation, and that some consequences can be suggested: (i) the influence on the dynamics of the delays of propagation between neurons (Chapeau-Blondeau et al. 1991), (ii) the distinction between intracellular and extracellular spaces which leads to considering propagation and diffusion in these spaces as non-local and local phenomena respectively. Recent experimental investigations on long-term potentiation (LTP), which is a persistent enhancement of synaptic efficacy following high-frequency stimulation (tetanization) of afferent fibers (Kelso et al. 1986) also provide some evidence for the existence of non-local effects. Specifically, LTP of inputs to the CA3 region of hippocampus, have confirmed that the induction of enhanced synaptic efficacy does not explicitly require activity in presynaptic fibers (Bradler et al. 1989, 1990). A mathematical expression of this physiological property of "true" heterosynaptic effect (Fig. 1) is that the dynamics of synaptic efficacy, for one subset of inputs, can depend on other subsets of synapses that are located further away on the same postsynaptic neuron, irrespective of the level of activity of the distant synapses.

To incorporate conditions appropriate for the existence of several levels of organization and continuous geometrical densities of neurons and synapses into models of neural networks, a field theory is proposed, which includes non-local interactions between biological substructures (Chauvet, 1993c). The dynamics of

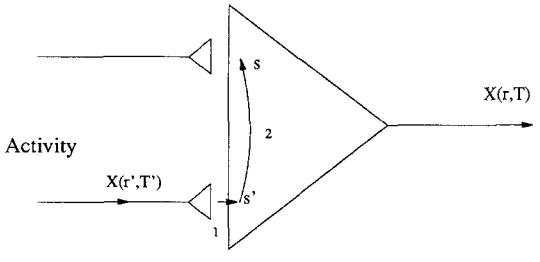

Fig. 1. Illustration of homo- (1) and hetero- (2) synaptic efficacies in the space of neurons ((r, T)-space) and in the space of synapses ((s, t)-space). Presynaptic activity is denoted $X \equiv Y(r', T')$, and postsynaptic activity is denoted $Y \equiv Y(r, T)$

a neural network are described when synaptic efficacy is modifiable and determined by both local and non-local sub-cellular mechanisms. The dynamics of a discrete formal neural network are again obtained, and conditions for classical Hebbian and non-Hebbian learning rules are deduced from the proposed field theory.

The field theory approach

Non-locality, non-symmetry and field theory

The concept of "*functional interaction*" is at the origin of the proposed theory. A functional interaction describes how a biological structure, called a "*source*", acts upon another biological structure, the "*sink*"; in the case of nervous system, the functional interaction is the expression of how one neuron acts upon another. For example, neural activity, considered as a space-time variable, is a functional interaction, as well as is synaptic efficacy. As will be shown later, there exist at least four fundamental properties for a functional interaction (Chauvet 1990): (i) *non-instantaneity*, i.e., its transport with a *finite velocity*, (ii) *non-locality* because of the possible long distance between sources and sinks considered at different levels of organization, (iii) *non-symmetry* between sources and sinks, and (iv) *non-homogeneity* of the medium. These four properties require a field theory.

The property of non-locality is a consequence of the size of the neuronal extent in the cartesian space: Two neuron-sources, can be infinitesimally close in the sense of the continuous density, but the corresponding neuron-sinks can be very far apart because of the extent of their axonal part in the cartesian space. The motoneurons, which give rise to the sciatic nerve, provide one such example. Another example can be deduced from the propagation of local synaptic or axonic potentials submitted to various intra and/or extracellular influences. Because the transport of the nervous influx occurs in the continuous space of one neuron, say

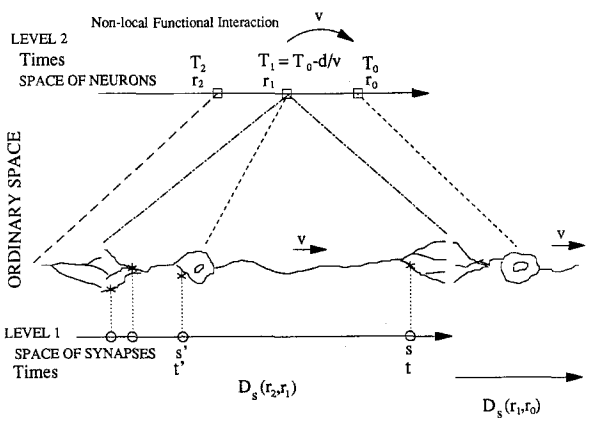

Fig. 2. Non-locality in the space of neurons where each point in the space of neurons in fact extended along a certain distance in the physical, cartesian space. The "point" considered is a volume, and includes the space of synapses (for this hierarchical system with two levels of organization). At time T_1 the value of the field variable is $\Psi(r_1, T_1)$ in point r_1 which includes the space $D_s(r_2, r_1)$ of synapses that connect neurons at r_2 with neurons at r_1, and the axonal space. It is shown that this is the source of non-locality.

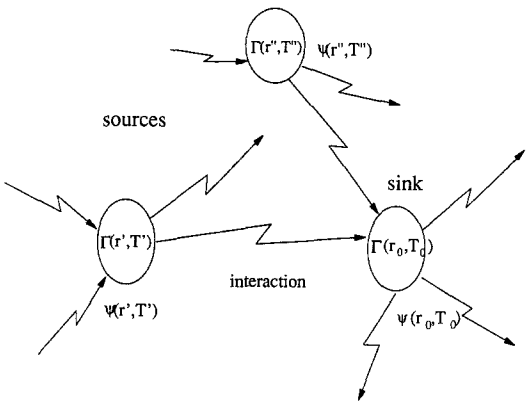

Fig. 3. Three interactive neurons represented as sources and sinks with an interaction operator from the source to the sink. Mathematically, this interaction is expressed by a field operator that operates on a field variable, and which carries it from point (r, T) or (r', T') to point (r_0, T_0). The source is represented by Γ.

with a velocity v_a, *and not in the continuous space of neurons*, what we see at time T_0 and at point r_0 in the space of neurons is what was emitted at time $T_1 = T_0 - d/v_a$ by neurons that are located at r_1 where $d = |r_1 - r_0|$ (Fig. 2). This non-local property, which expresses the coupling of biological substructures at a distance, is very general, and is the consequence of the structuring of the system into several levels of organization (Chauvet, 1993b). Therefore, for the same reason, the synaptic efficacy modifiability will be locally (in the sense of a density of synapses) the expression of non-local interactions with the velocity v_s at the molecular level.

As a consequence of the propagation of activity in the neural network, non-symmetry is a property which must be included in the formulation. For example, space-time equations like reaction-diffusion equations are symmetric and cannot represent this propagation phenomenon. That is the case of Hodgkin-Huxley equation which represents the two-way active propagation of action potential from any initial point of the axon.

A solution to these problems, a consequence of the existence of non-locality and non-symmetry, is to include in the local dynamic equation of one neuron, a non-local and non-symmetric term which describes the unidirectional action of this neuron at a distance. That can be done with the field theory (Chauvet 1988a, 1988b). In the framework of physical field theory, an interaction is propagated with a finite velocity from the source point to the sink point: A source in (r', T') creates a field ψ which acts in turn on a neighboring point (r_0, T_0) (Fig. 3). Because of the non-symmetry, sources and sinks do not have a similar role regarding their reciprocal influence: the source acts on the sink, but the sink does not act on the source. If, at point (r', T'), a source interacts with the field $\psi(r', T')$, then a new value $\psi(r_0, T_0)$ for the field variable is obtained at the neighboring point (r_0, T_0), and so on. Such a process could be described by the following general equation:

$$H(\psi)\psi(r_0, T_0) = \Gamma(r_0, T_0) \tag{1}$$

where ψ is the excitation field which is propagated by the non-linear field operator $H(\psi)$, (that can depend on ψ), from sources to sinks, and Γ is the source term for a source localized in (r_0, T_0). A similar formulation has been used for only one level of organization (Beurle 1956, Griffith 1963, Wilson et al. 1972, Fischer 1973, Kishimoto et al. 1979). The problem of the determination of the field operator H is not easy, because it mathematically requires it to take into account all orders of time and space derivatives, and integral operators. Most

often, that determination is imposed by the geometry of the space or by some symmetry considerations. For example, in some cases of good regularity, the field operator $H(\psi)$ can be considered as invariant with regards to rotations and translations, so that:

$$H(\psi) \equiv \frac{\partial}{\partial T} - D\nabla^2 - H_I(\psi) \tag{2}$$

where $D\nabla^2$ is a diffusion term that depends only on the properties of the medium, and H_I is an interacting non-local and non-symmetric operator. Therefore, with H_I depending on ψ, and with a potential kernel function V_ψ, Eq. (2) can be re-writen, at point (r_0, T_0) as:

$$\frac{\partial \psi}{\partial T} = D\nabla^2\psi + \int_{D_R(r_0)} V_\psi(r_0, T_0, r', T'; \Gamma(r', T'))\varrho(r')\, dr' + \Gamma \tag{3}$$

where $\varrho(r')$ is the density of units at r'. Thus, the role of V_ψ at (r', T') is to interact with the field ψ at (r_0, T_0), which is generated by the processes that are evolving in the source $\Gamma(r', T')$. In Eq. (3), the velocity v_a of transport along the extent of one neuron appears via the term $\psi(r', T') \equiv \psi(r', T_0 - d/v_a)$ where $d = |r' - r_0|$ (Fig. 2). As discussed above, the existence of the functional interaction with a finite velocity v at the lower level implies the non-locality. In the following, the notation will take into account the rank of the synapses: r_0 denotes the present point in the r-space; r_i denotes the points where presynaptic neurons are located. Subscript i is used to represent the rank of the synapse relatively to r_0. For example, neurons at r_1 are connected with neurons at r_0 via a monosynaptic pathway, neurons at r_2 are connected with neurons at r_0 via a disynaptic pathway, i.e., with neurons at r_1 via a monosynaptic pathway. Points r_1', r_1'' are points such as r_1 in r-space, r_2', r_2'' are points such as r_2, etc. *When subscript for afferent neuron points is not specified, then a monosynaptic pathway is assumed.*

Activity and synaptic efficacy as field variables

It is desirable to use as field variables observed quantities (observables) that have a physical meaning to improve the theoretical interpretation of the dynamics based on experimental data. At the neural level with a time scale $\{T\}$, the field variable is the *activity* which is defined as action potential frequency. At the synaptic level with the longer time scale $\{t\}$, the choice of field variables is more difficult (Fig. 4, see also Appendix A). The "*local postsynaptic membrane potential*" is denoted as $\Phi(s, t)$ at a point (s, t) in the space of one neuron at (r_0, T_0) and corresponds to a presynaptic neuron at (r, T). The "*local somatic depolarization*" is denoted as $\psi(r_0, T_0)$ at the axon hillock at (r_0, T_0) in the space of neurons. With these definitions, a general definition of *synaptic efficacy* between presynaptic neurons in r and postsynaptic neurons in r_0 at synapses in $s(r, r_0)$ could be:

$$\mu(s, t) = C[\Phi(s, t), \langle\psi(r, T)\rangle(t)] \qquad s \equiv s(r, r_0) \tag{4}$$

where $\langle\psi\rangle(t)$ is the average depolarization at time t, and the function C, which represents the synaptic efficacy, has to be determined. This expression for μ includes implicitly with $\langle\psi\rangle(t)$ the mechanisms for pre-synaptic efficacy, and with Φ the mechanism for post-synaptic efficacy. Their description will be given in Sec. 3. The "*instantaneous local somatic activity*" $X(r_0, T_0)$ in the time scale

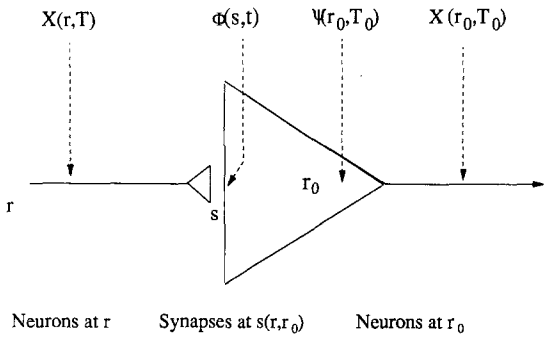

Fig. 4. Definition of field variables, input activity $X(r, T)$, postsynaptic potential $\Phi(s, t)$ in the space of synapses, soma membrane potential $\Psi(r_0, T_0)$, and output activity $X(r_0, T_0)$. Two of these, soma membrane potential and synaptic efficacy related to postsynaptic potential, have been chosen as independent variables

$\{T\}$ is deduced from ψ by a non-linear, generally sigmoïd input-output function F:

$$X(r_0, T_0) = X(\langle \psi(r_0, T_0) \rangle) = F(\langle \psi(r_0, T_0) \rangle (T_0)). \tag{5}$$

Therefore, the passage from one level of organization (synapse) to the higher (neuron) with ψ and μ as local somatic depolarization and local synaptic efficacy respectively, leads to our considering the ψ-field and the μ-field as evolving in two different spaces whose points are denoted by (r, T) and (s, t) respectively. In this conceptualization, a given space at one point within one level is a point for the next higher level. This property of "space inclusion" is characteristic of a hierarchical system with fields acting at multiple levels. In this paper, a formulation of the dynamics of the neural network described by both soma membrane potential and synaptic efficacy as field variables satisfying Eq. (4) (or similar ones of the same class of equations), is investigated as a consequence of the propagation of electrical potentials, whatever the mechanism. Although complex, this approach has the advantage of classical continuous mathematical analysis, and, further, partial integro-differential equations are solvable by numerical methods. Moreover, particular discrete cases can be deduced from the general continuous case when the densities are chosen as Dirac Delta functions.

Synaptic level of organization

Local synaptic efficacy. Influence of time scale

Presynaptic mechanisms at any point (s, t) in a neuron at (r_0, T_0) and corresponding to a presynaptic neuron at (r, T) are described by terms that can interpret the kinetic properties of neurotransmitter release (Magleby et al. 1982). A phenomenological expression of these kinetic properties as a function of time in the neuron space is obtained by fitting the observed curve with the product of the four functions that describe facilitation, potentiation, and depression (decreasing) rates:

$$\xi(s, t) = \xi^0(s, t) \prod_{i=1}^{4} (1 + d^{(i)}(s, t)). \tag{6}$$

This expression gives the probability of neurotransmitter release when the four functions $d^{(i)}s, t)$ are known. The basal level of presynaptic efficacy

$\xi^0((s, t); \langle X \rangle)$ is a function of activity $\langle X \rangle(t)$. A simplified expression including only two terms was used by Finkel et al. (1987) to describe presynaptic facilitation and synaptic depression. They interpret ξ as the *presynaptic efficacy*.

Postsynaptic mechanisms include transmitter-receptor binding, transitions between opened and closed channel states, conductance variation of voltage-dependent channels, the resulting variation of postsynaptic potentials (PSP), and the action of neuromodulators. Following Finkel et al. (1987), Changeux and Heidmann (1987), a simple two-state kinetic model can be chosen to describe the binding of transmitter to postsynaptic receptor sites. The instantaneous postsynaptic efficacy η is defined as a conductivity. In a simplified model, it is in direct relation with a sum of two conductance terms: (i) the product of the number $(1 - f_I(\Phi))$ of non-modified (but modifiable) channels with conductance g with the number of non-activated receptors $1 - R^*$; and (ii) the product of the number $(1 - f_I^*(\Phi))$ of modified channels with conductance g^* with the number of activated receptors R^*:

$$\eta = \eta^0[(1 - R^*)(1 - f_I(\Phi))g + R^*(1 - f_I^*(\Phi))g^*]. \tag{7}$$

The postsynaptic potential (PSP) is obtained using the Hodgkin-Huxley equation (Hodgkin et al. 1952):

$$\frac{\partial^2 \Phi(s, t)}{\varrho \, \partial s^2} = K\left[\left(C \frac{\partial \Phi}{\partial t}\right) + \sum_k g_k(\Phi - \Phi_k) - G(\Phi(s, t) - \Phi_\infty)\right] \tag{8}$$

where the g's and $G = Rg$ are the conductances that depend on the postsynaptic potential and on the synaptic and ionic currents; K is a coefficient, and C is the membrane capacity. This reaction-diffusion equation expresses the local conservation of the number of ions during their transport across the membrane. Because of the symmetrical space diffusion term, it cannot represent the transport of action potential from one neuron to another.

A variation of the postsynaptic potential requires two local mechanisms: (i) free diffusion of ions, depending on extrasynaptic channels and on the conditions of extracellular space, e.g., extracellular potentials. This variation is described by $D_0 \nabla_s^2 \Phi(s, t)$, where the diffusion coefficient D_0 includes the properties of matter in space, which generally are non-homogeneous. In case of non-homogeneity, diffusion is described by $\nabla_s(D_0 \nabla_s \Phi(s, t))$. (ii) spontaneous loss and gain of ions due to *homosynaptic* effects that are described by the term $-k_0 \Phi(s, t) \equiv \Gamma_0(\Phi(s, t))$. These two source terms are represented by J_k and J_{syn} for the ionic and synaptic currents respectively in the Hodgkin-Huxley Eq. (8), which can be re-written more generally as:

$$\frac{\partial \Phi(s, t)}{\partial t} = \nabla_s(D_0 \nabla_s \Phi(s, t)) + \Gamma_0(\Phi(s, t)). \tag{9}$$

Let us define the "*instantaneous local synaptic efficacy*" σ_0 as the function which describes *all* the phenomena that occur between presynaptic depolarization $\psi(r, T)$ and consequent postsynaptic potential $\Phi(s(r, r_0), t)$:

$$\psi(r, T) \xrightarrow{\sigma_0} \Phi(s, t). \tag{10}$$

Defined in such a way, instantaneous local synaptic efficacy σ_0 depends on various parameters that satisfy local dynamics, i.e., a system of differential equations for which the mathematical solution will determine the *long-term evolution* of instantaneous local synaptic efficacy. This fundamental hypothesis regarding the dynamics seems reasonable because: (i) the number of receptors is

dependent on internal chemical modifications, including those mediated by a neuromodulator; so, only the long-term average of instantaneous local synaptic efficacy will be observed, (ii) the concentration of neurotransmitter varies slowly, as its long-term average, and therefore depends on long-term activity, (iii) the concentration of neuromodulator results from intracytoplasmic kinetics and can evolve slowly as its long-term average. In other words, *the slow evolution of these dynamics implies variables which evolve in the same long time scale and which are the time averages of the observed concentrations of neuromodulators, neurotransmitters, and receptors. With these concentrations denoted respectively by* M, R, N, let $\langle M\rangle$, $\langle R\rangle$, $\langle N\rangle$, be the slow variables calculated on an interval ΔT. Then, the local synaptic efficacy μ_0 can be defined by:

$$\mu_0(s(r, t_0), t; \langle M\rangle, \langle R\rangle, \langle N\rangle = \langle \sigma_0(s(r, r_0), t; M, R, N)\rangle_{\Delta T}. \tag{11}$$

This equation introduces a new definition for local synaptic efficacy that takes into account the time scale of the phenomena. The next step in a formalization of neuronal interactions in terms of the field theory is to introduce in the mathematical formulation the non-local and non-symmetric effects. Note that in this continuous approach where a functional interaction operates from one point to another with a finite velocity, non-local effects are inherent, and appear in both levels of organization. One argument for the existence of such functional interactions at the synaptic level is the experimental evidence of true heterosynaptic effects (Bradler et al. 1990).

Non-local mechanisms for the variation of synaptic efficacy. Global synaptic efficacy defined from field effects

A more general form of the postsynaptic potential variations than that described by Eq. (8), and therefore by Eq. (9), is obtained by considering non-local potentials that depend on membrane receptors and neurotransmitters: the postsynaptic potential at s in the neuron localized at r_0, which is connected with neurons at r, results from other synapses localized at s' on the same dendritic tree, due to the activation of neurons localized in r' (Fig. 5). These heterosynaptic effects depend on two anatomical functions: (i) the *density-connectivity* $\pi(s', r'; r_0)$ which is defined in a space $D_s(r', r_0)$ and constituted by the synapses

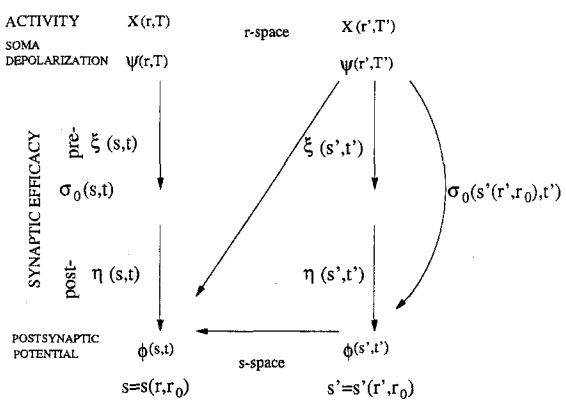

Fig. 5. Non-local heterosynaptic effects between two distant synapses at (s, t) and (s', t'), which originate in two distant points (r, T) and (r', T') in the space of neurons. Thus, presynaptic activities are considered at these two points: $X(r, T)$ and $X(r', T')$. For example, two effects converge in (s, t), one from the soma depolarization at (r, T), and the other from (r', T') via the synapse at (s', t')

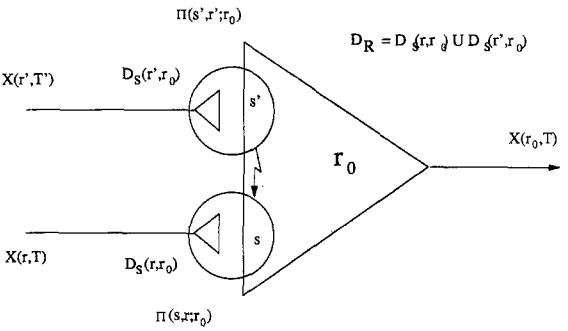

Fig. 6. The density-connectivity $\pi(s, r; r_0)$ represents the density of synapses at s, in the neuron at r_0, that are connected, with a certain probability, with neurons at r. Their spaces of definition are $D_s(r, r_0)$ and D_R that is the recombination of spaces D_s for all presynaptic neurons at r

localized at $s'(r', r_0)$ in the neurons at r_0, which are connected with the neurons at r'. (ii) the *density* of neurons $\varrho(r')$ at r' which is defined in space $D_R(r_0)$, the recombination of subspaces $D_s(r', r_0)$ when r' varies (Fig. 6). It is possible to describe this field effect as a distance effect with a mathematical *potential kernel function* $U_\Phi(s, t, s', t'; \xi, \eta)$ where variables ξ and η are considered as parameters that are defined at the lower level. The interaction operator is:

$$H_I^\Phi = \int_{D_R(r_0)} \varrho(r') \int_{D_s(r', r_0)} U_\Phi(s, t, s', t'; \xi, \eta)\pi(s', r'; r_0)\, ds'\, dr' \qquad (12)$$

where

$$s \equiv s(r, r_0)$$

$$D_R(r_0) = \bigcup_{r'} D(r', r_0).$$

In this way, U_Φ is a function which has to include the set of phenomena which occur at s' and act upon s (Figs. 5 and 6). For example, the passive conduction implies an attenuation of potential between these two points (Rall et al. 1973). The potential Φ is modified ($\delta\Phi$) due to a variation in the number of activated receptors, which occurs as a consequence of biochemical dynamics. Therefore, Eq. (9) for the postsynaptic potential can be written as:

$$\frac{\partial \Phi}{\partial t}(s, t) = \nabla_s(D_0 \nabla_s \Phi(s, t)) + \int_{D_R(r_0)} \varrho(r') \int_{D_s(r', r_0)} U_\Phi(s, t, s', t'; \xi, \eta)$$

$$\times \pi(s', r'; r_0)\, ds'\, dr' + \Gamma_0(\Phi(s, t)) \qquad (9')$$

with $t' = t - |s - s'|/v$, where v is the velocity of the interaction due to the propagation of the potential along the dendritic tree membrane. This is the postsynaptic potential non-local field equation.

Because this postsynaptic potential field equation is extended from Eq. (9), the instantaneous local synaptic efficacy $\sigma_0(s(r, r_0), t)$, as in (10) above which is deduced from Eq. (9) has also to be completed. Let $\sigma(s(r', r_0), t)$ be the new, non-local function that describes the effect of the presynaptic neurons on the postsynaptic one, and defined as:

$$\forall r' \quad \psi(r', T') \xrightarrow{\sigma} \Phi(s(r', r_0), t). \qquad (10')$$

Equation (9′) leads to the assumption of a similar, but crucially non-local, equation for σ:

$$\frac{\partial \sigma}{\partial t} = \nabla_s(D_1 \nabla_s \sigma) + H_I^{\sigma}(U_{\sigma}(s, t, s', t'; \Phi, \delta\Phi) + \Gamma_1(\sigma) \tag{13}$$

where H_I^{σ} is a field operator that depends on $\Phi(M, R, N)$ and $\delta\Phi(M, R, N)$, in accordance with the homo- and heterosynaptic mechanisms described above. Note that in Eq. (10′) for σ, a property is added to the property of σ_0 described in (10): due to diffusion of the postsynaptic potential, there exists a distance interaction of the instantaneous synaptic efficacy σ_0 which leads to a diffusion term.

As μ_0 was deduced from the σ_0-average by Eq. (11), the "global synaptic efficacy" μ will be *defined* from the σ-average by the same relationship:

$$\mu(s(r, r_0), t; \langle M\rangle, \langle R\rangle, \langle N\rangle) = \langle \sigma(s(r, r_0), t; M, R, N)\rangle_{\Delta T}. \tag{11′}$$

The most interesting implication of the non-locality property included in this formulation is the determination of the *non-local interaction operator* for μ because it allows us to deduce new learning rules, as will be shown below.

Structure of the neuronal field equation and interaction operator

Structure: Following the discussion about the time scale that leads to Eq. (11), the local synaptic efficacy μ_0 is defined as the time average of the instantaneous local synaptic efficacy σ_0, and consequently is dependent on the time average concentrations, e.g., $\langle M\rangle$, $\langle R\rangle$, $\langle N\rangle$, according to a *new specific equation.* Referring to the preceding discussion about the non-locality that leads to Eq. (13), the non-local synaptic efficacy σ is defined using the non-local equation (9′) by introducing a non-local interaction operator that represents electric or chemical interactions between two synapses via the membrane or the cytoplasm. Therefore, time scale and non-local interactions can be included in the global synaptic efficacy μ if we assume that it satisfies a non-local equation like (13) with an adapted interaction operator which depends on *long-term variables.* Formally, the equation for μ is deduced from Eq. (13) as:

$$\frac{\partial \mu}{\partial t} = \nabla_s(D_s \nabla_s \mu) + H_I^{\mu}(\mu) + \Gamma_{\mu}. \tag{15}$$

The first term corresponds to a local spatial variation and is directly deduced from Eq. (13). The second term describes the long-term spatio-temporal summation of all non-local effects, which lead from $\psi(r', T')$ to $\Phi(s(r', r_0), t)$. The third term corresponds to an instantaneous local reaction in the source.

Let U_{μ} be the *potential kernel function.* Although this function could be expressed in many different forms, only a few forms are consistent with experimental observations, and moreover, give rise to the Hebbian learning rules. Therefore, the following forms for U_{μ} are proposed:

$$H_I^{\mu} \equiv H_I^{\mu}[U_{\mu}(\langle \sigma\rangle, \delta\langle \sigma\rangle; \langle \xi(X)\rangle, \langle \eta(M, R, N)\rangle)] \tag{16.1}$$

$$U_{\mu}(\mu, \delta\mu; \langle \xi\rangle, \langle \eta\rangle) \equiv U_{\mu}(\mu, \delta\mu(\delta\langle R\rangle); \langle \eta(M, R, N)\rangle) \tag{16.2}$$

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Collectively, Eqs. (16.1) to (16.3) represent the dependence, of non-local interaction between two synapses on synaptic efficacy, μ itself, and/or its

variation $\delta\mu$, when some variables such as receptor, neuromodulator, neurotransmitter concentrations, $R, \ldots, M, \ldots, N$, are involved in the local dynamics. Long-term pre- and postsynaptic efficacy, $\langle\xi\rangle$ and $\langle\eta\rangle$, act as parameters. Such a formulation describes how distant synapses depend, either on the absolute value of global synaptic efficacy, or on its time variation, given the local pre- and postsynaptic efficacy, from eqs (6) and (7) considered as local conductivities.

The first two forms (16.2) and (16.3) suggested for U_μ lead to a potential kernel function determined from (12) such that:

$$U_\mu(s, t, s', t'; \langle\xi\rangle, \langle\eta\rangle) = A(s, s')\mu_0(s', t')\delta\mu(s', t') \qquad (16.2)'$$

$$U_\mu(s, t, s', t'; \langle\xi\rangle, \langle\eta\rangle) = A(s, s')\mu_0(s', t') \qquad (16.3)'$$

respectively, where μ_0 is the local synaptic efficacy, and written as points in the s-space. Thus with (16.3)', the μ-field equation is from (15):

$$\frac{\partial\mu(s, t)}{\partial t} = \nabla_s(D_s\nabla_s\mu(s, t)) + \int_{D_R(r_0)} \varrho(r') \int_{D_s(r', r_0)} \mu_0(s', t')\pi(s', r'; r_0)A(s, s')\, ds'\, dr' + \Gamma_\mu(s, t) \qquad (17)$$

$s \equiv s(r, r_0)$

$D_R(r_0) = \bigcup_{r'} D(r', r_0)$

where $t' = t - |s - s'|/v_\mu$, with v_μ being the velocity of the μ-interaction. $D_R(r_0)$ is the reconnection of subspaces $D_s(r', r_0)$ when r' varies. A is an attenuation function for the electric potential along the membrane between two points, s' and s per unit time.

Interaction mechanisms: The simplest way to deduce this interaction is to suppose that it results from the long-term evolution of chemical substances M and N. This long-term evolution determines the variation $\delta\langle R^*\rangle$ of activated receptors denoted by R^*, and consequently the variation $\delta\langle\sigma\rangle$ of the non-local synaptic efficacy at time t. A simplified case that nevertheless is consistent with the classical Hebbian rules, is obtained with the following assumptions:

(1) local pre- and postsynaptic linear dynamics are represented by:

$$\Gamma_\mu = m\mu(s, t) \qquad (18.1)$$

(2) the interaction between ξ and η that results in μ_0 (local synaptic efficacy, Fig. 5) in Eqs. (16.2)' and (16.3)', is multiplicative:

$$\mu_0(s, t) = \langle\xi(s, t)\eta(s, t)\rangle \qquad (18.2)$$

and a possible expression for $\delta\mu$ in (16.2)' is:

$$\delta\mu(s, t) = K\mu(s, t).$$

In (18.2), the functions ξ and η are given by Eqs. (6) and (7) as solutions of complex and non-linear local dynamics, or by the average of pre- and postsynaptic activities, $\langle X(r, T)\rangle$ and $\langle X(r_0, T_0)\rangle$, respectively, at two points r and r_0. The first class of solutions for ξ and η leads to a threshold-like behavior (5) for neuronal activity; this formulation includes non-linearities because of the existence of physiological mechanism at both the synaptic and the neuronal level of

organization. The second class of solutions for ξ and η presents a simplified description of the complex internal dynamics expressed by Eqs. (6) and (7), and leads to linear behavior. It imposes a further relation such as that described by Eq. (5), i.e., the expression of soma depolarization as a function of activity. In the latter case, we have:

$$\xi(s, t; \xi^0(\langle X\rangle)) = a(\xi^0(s); r)\langle X(r, T)\rangle(t) \tag{18.3}$$

$$\eta(s, t; \eta^0(\langle X\rangle)) = b(\eta^0(s_0, s); r)\langle X(r_0, T_0)\rangle(t). \tag{18.4}$$

Assumptions (18.2), (18.3) and (18.4) lead to the learning rules of the biological neural network, because they drive the variation of μ in time. These assumptions, and particularly these learning rules, can be included in Eq. (17) to give the particular μ-field equation:

$$\frac{\partial\mu(s, t)}{\partial t} = \nabla_s(D_s\nabla_s\mu(s, t)) + \int_{D_R(r_0)} \varrho(r') \int_{D_s(r', r_0)} a(s'; r')b(s'; r_0)A(s, s') \times \langle X(r', T')\rangle(t')\langle X(r_0, T_0)\rangle(t')\pi(s', r'; r_0)\, ds'\, dr' + m\mu(s, t). \tag{19}$$

It is clear from the nature of interaction mechanisms (18.2, 3, 4) that *strict heterosynaptic effects* can occur if, at a given point s', the synaptic efficacy μ_0 does not depend on neuronal activity. The contribution of one non-active synapse at point s' to the variation of μ at point s, is a direct consequence of the field equation (19). Note that the non-local nature of the interaction operator U_μ is important, because it includes the activity traces in the system, the time evolution of which is described by μ. So, events at $t' < t$, which occur at a distance, contribute to the space-time evolution of the μ-field.

Neural level of organization and neural field equations

Potential function for the neuron-neuron interaction

At the neural level of organization, the potential function describes the effect of divergent or convergent neurons from or to a (r_0, T_0)-point. Postsynaptic soma depolarization $\psi(r_0, T_0)$ at that point, results from the presynaptic depolarizations $\psi(r, T)$ that are modified according to synaptic efficacies $\mu(s(r, r_0), t)$. The neuronal space $\{s(r, r_0), t)\}$ is *in* the neural space $\{(r, T)\}$; the correspondence between these two spaces is expressed by the density-connectivity function $\pi(s(r, r_0), r; r_0)$. Such a property of inclusion of spaces with a connectivity function between them is a direct consequence of multiple levels of organization, as is the case with the nervous system.

The ψ-field equation has a particular form due to the mechanisms of firing:

$$\frac{\partial\psi(r_0, T_0)}{\partial T} = \nabla_r(D_r\nabla_r\psi(r_0, T_0)) + H_I^\psi(\mu) + \Gamma_\psi(r_0, T_0). \tag{20}$$

It is known that if the value for ψ exceeds a value defined as depending on the neuron internal state, then a steep variation of ψ occurs. This steep variation corresponds to firing. Thus, neural activity is a function of ψ (see Eq. (5)). An interpretation for this non-linear property can be given by a special expression for the source, Γ_ψ, by assuming that it depends on a strictly local transformation $\mathfrak{R}$ which leads to the "activation":

$$\Gamma_\psi(r_0, T_0) = \mathfrak{R}(\psi(r_0, T_0^-), \psi_{\text{refr}}) \tag{21}$$

where ψ_{refr} denotes the refractoriness of the neuron, and T^- refers to states of the neuron prior to activation.

Let $U_\psi(r, T, r_0, T_0; \mu)$ be the *potential kernel function* of the neuron-neuron non-local interaction. The simplest form for this function that is compatible with the accepted law of spatio-temporal summation is:

$$U_\psi(r_0, T_0, r, T; \mu) = B(r_0, T_0, r, T)\mu(s(r, r_0), T). \tag{22}$$

Then, the non-local interaction operator is:

$$H_I^\psi = \int_{D_R(r_0)} \varrho(r)\psi(r, T) \int_{D_s(r, r_0)} B(r_0, T_0, r, T)\pi(s, r; r_0)\mu(s, t)\, ds\, dr.$$

Combining Eqs. (20), (21), and (22) gives the ψ-field equation:

$$\frac{\partial\psi(r_0, T_0)}{\partial T} = \nabla_r(D_r\nabla_r\psi(r_0, T_0)) + \int_{D_R(r_0)} \varrho(r)\psi(r, T) \int_{D_s(r, r_0)} B(r_0, T_0, r, T) \times \pi(s, r; r_0)\mu(s, t)\, ds\, dr + \Re(\psi(r_0, T_0^-), \psi_{\text{refr}}) \tag{23}$$

where $\pi(s(r, r_0), r; r_0)$ is the synaptic density-connectivity space function between neurons at (r, T) and synapses per neuron at $(s(r, r_0), t)$, $\varrho(r)$ is the density of neurons in the r-space, and $B(r_0, T_0, r, T)$ is a space attenuation factor between the two points (r, T) and (r_0, T_0), which takes into account temporally preceding neuronal states for $T_0 - \tau \leqslant t \leqslant T_0$ where τ is a neuron time constant. In contrast to $A(s, s')$, the kernel function B includes the spacetime relation expressed by the velocity of the propagation of the membrane potential. This explains why A, and not B, is a coefficient whose dimension is unit per time.

Neural field equations and physiological interpretation

Equation (17) for the μ-field, deduced from (16.3)′ and (23) for the ψ-field can be combined to provide the neural field equations of the neural network in this continuous approach:

$$\frac{\partial\mu(s, t)}{\partial t} = \nabla_s(D_s\nabla_s\mu(s, t)) + \int_{D_R(r_0)} \varrho(r') \int_{D_s(r', r_0)} A(s, s')\pi(s', r'; r_0) \times \mu_0(s', t')\, ds'\, dr' + \Gamma_\mu(s, t) \tag{24.1}$$

$$\frac{\partial\psi(r_0, T_0)}{\partial t} = \nabla_r(D_r\nabla_r\psi(r_0, T_0)) + \int_{D_R(r_0)} \varrho(r)\psi(r, t) \int_{D_s(r, r_0)} B(r_0, T_0, r, T) \times \pi(s, r; r_0)\mu(s, t)\, ds\, dr + \Re(\psi(r_0, T_0^-), \psi_{\text{refr}}) \tag{24.2}$$

$$s \equiv s(r, r_0)$$

$$D_R(r_0) = \bigcup_{r'} D(r', r_0).$$

where $T = T_0 - d/v_a$, $d = |r - r_0|$, v_a is the transport velocity of the ψ-interaction, i.e., the neural activity, and $t' = t - |s - s'/v_\mu$, with v_μ being the velocity of the μ-interaction. Equations (24.1) to (24.2) are the neural 2-level field equations for the activity of neural tissue at two corresponding points $(s(r, r_0), t)$ and (r, T) in space-time level of organization. Their coupling is imposed by the synaptic efficacy μ, and the neural tissue is characterized by two geometric functions: the

density ϱ of neurons and the density-connectivity π of synapses. $D_s(r, r_0)$ is the space of synapses in neurons localized in r_0, which correspond to neurons in r. $D_R(r_0)$ is the neural integration space that is the recombination of subspaces $D_s(r, r_0)$. This formulation implies *non-linearities* which appear via the source terms. However, transformations F (Eq. (5)) and $\mathfrak{R}$ (Eq. (21)) are not deduced from the formulation, but could be determined on the basis of experimental investigations (Chauvet et al. 1990). Relation (4) for the global synaptic efficacy appears as the solution for the field equations (24.1) and (24.2) that include local and non-local effects from multiplicative interaction (18.2), where pre- and postsynaptic efficacies are determined by (6), and (7), and from the anatomical space functions π and A. Learning rules can either be imposed on the network according to Eqs. (18.3) and (18.4), or included in the dynamics from Eqs. (6) and (7).

The local effects, which are described by the *diffusion* term in field equations (24.1) and (24.2), correspond to transport across the "external" medium, i.e., the extracellular space for soma depolarization, and the cytoplasm for synaptic efficacy. They depend on the diffusion coefficients D_s and D_r, respectively. The non-local effects, due to the hierarchy of the system, and which are described by the interaction terms in field equations (24.1) and (24.2), correspond to the *propagation* "inside" the medium, i.e., neurons for the soma depolarization, and membrane and cytoplasm for the synaptic efficacy. What we see in the continuous space at each level of organization is the combination of these two types of transport, diffusion and propagation. Their contribution to the dynamics of the field variables is determined by the value of the diffusion coefficients in Eqs. (24.1) and (24.2). Thus, the proposed n-level field theory predicts a distance effect of synaptic activity due to propagation of that activity in the internal medium.

Hebbian and non-Hebbian rules

Dynamics for a discrete network

One important question is whether classical Hebbian or non-Hebbian learning rules can be deduced from this field theory, and also whether new properties can be obtained for the neural network with the new learning rules derived from the interaction operator. Although the formalism used with the field theory is deterministic and continuous at each level of organization, a discrete representation of neural network can be obtained by concentrating the values of field variables at any point of the space of neurons. That can be done with particular point density functions of neurons and synapses such as Dirac functions, $\delta(r - r_i)$, and can be used to study some discrete cases. They concentrate all of the field variable values, at any r, into n values at r_i, $\forall i = 1, n$. As a result, partial differential equations distributed in space are transformed into n differential equations, one for each point.

As an illustration, a neuron with only one input and one output is considered under the following assumptions: (i) a dendrite originating at r connects with a neuron at r_0, via a synapse at s (Fig. 6). Thus, the density function $\varrho(r') = \delta(r' - r)$ and the connectivity function $\pi(s', r'; r_0) = \delta(s' - s)$ which implies: $A(s, s') = 1$ and $B(r_0, T_0, r, T) = 1$; (ii) soma depolarization diffusion and refractoriness are negligible; the local firing dependence, $\mathfrak{R}$, is assumed to be piecewise linear (21): $\mathfrak{R} = p\psi$; (iii) the neuron membrane is homogeneous; (iv) the velocity of the dendritic transport of interaction is infinite: $v = \infty$. Learning

rules are given by Eqs. (18.3) and (18.4), with a and b constant. We start from the field equation (15) with the structure (16.2), then (16.2)′, and (18.2), i.e.:

$$\frac{\partial \mu(s,t)}{\partial t} = \nabla_s (D_s \nabla_s \mu(s,t)) \int_{D_R(r_0)} \varrho(r') \times \int_{D_s(r',r_0)} \mu_0(s',t') K \mu(s',t') \pi(s',r';r_0) A(s,s')\, ds'\, dr' + \Gamma_\mu(s,t). \quad (25)$$

Combining these assumptions with (25), (where the notation $\langle \psi \rangle(t)$ is now replaced by $\bar{\psi}(t)$), and (5), (18.3) and (18.4), transform the system of equations (24.2) and (25) into:

$$\frac{\partial \mu(s,t)}{\partial t} = abK \int_{D_R(r_0)} \int_{D_s(r',r_0)} X(\bar{\psi}(r',t)) X(\bar{\psi}(r_0,t)) \mu(s'(r',r_0),t') \times A(s,s') \delta(s'-s) \delta(r'-r)\, ds'\, dr' + m\mu(s,t)$$

$$\frac{\partial \psi(r_0,T_0)}{\partial T} = \int_{D_R(r_0)} \psi(r',T_0) \delta(r'-r) \int_{D_s(r',r_0)} \mu(s',t) \delta(s'-s)\, ds'\, dr' + p\psi(r_0,T_0).$$

It follows that:

$$\frac{\partial \mu(s,t)}{\partial t} = abKX\{\bar{\psi}(r_0,t)\} X\{\bar{\psi}(r,t)\} \mu(s_0,t) + m\mu(s,t)$$

$$\frac{\partial \psi(r_0,T_0)}{\partial T} = \mu(s,t)\psi(r,T) + p\psi(r_0,T_0). \quad (26)$$

With the following notation in terms of time only: $\mu(s,\cdot) = \mu(\cdot)$ for synaptic efficacy, and $\psi(r_0,\cdot) = \psi(\cdot)$, $\psi(r,\cdot) = \psi'(\cdot)$ for soma depolarization, Eqs. (26) become:

$$\frac{d\mu(t)}{dt} = \{abKX(\bar{\psi}'(t)) X(\bar{\psi}(t)) + m\} \mu(t) \quad (27.1)$$

$$\frac{d\psi(T)}{dT} = \mu(t)\psi'(T) + p\psi(T). \quad (27.2)$$

Each equation is written here in its own time scale, the short time scale $\{t\}$ for the synaptic level, and the rapid time scale $\{T\}$ for the neuronal level. Using the time scale of the synaptic level for the neuronal level, Eq. (27.2) changes to: $\bar{\psi}(t) \approx -(\mu(t)/p)\bar{\psi}'(t)$, that is a linear response, iff:

$$\bar{\psi}(t) \approx 0, \qquad \overline{\mu(t)\psi'(t)} = \mu(t)\bar{\psi}'(t). \quad (28)$$

Under those two conditions, soma depolarization proceeds in a linear fashion, i.e., the solution for $\bar{\psi}(t)$ is linear. The first condition expresses the slow short term variation of the soma depolarization average, i.e., a global variation around zero. The second condition follows from the fact that the synaptic efficacy varies on a time scale larger than the soma depolarization average (short time scale). With this interpretation, an important parameter is p, because it drives the system $\psi(\mu)$ and leads to non-linearities in (24.2), then in (25), together with the interaction operator that depends on μ. Therefore, the time average of the ψ-equation (27.2) leads to a similar relation between neural activities, due to the non-variability of μ in the short time scale.

Consequences of the μ-field existence: learning and variation of the level of learning

Various rules for the behavior of the previous discrete formal system can be obtained from (26) and (27) for a given field structure and from the μ-interaction operator hypotheses (16) and (18). To have a notation consistent with the classical ones used in formal neural networks, we put: $X(t) = F(\bar{\psi}'(t))$ for presynaptic activity, and $Y(t) = F(\bar{\psi}(t))$ for postsynaptic activity. Three kinds of rules are presented in Table 1. The distinction between them originates in the μ-operator and the source term. In classical Hebbian rules (see e.g., Kohonen 1978) and Easton-Gordon rules (1984), the μ-operator depends only on $\delta\mu$ and the source terms are zero and $m\mu = -\alpha\mu$ respectively. In the learning rules considered here, the μ-operator depends on μ and $\delta\mu$, and the source terms equals $m\mu$, $\forall m \neq -\alpha$. All other conditions are the same. These rules correspond to a linear system because of the multiplicative nature of the ψ-interaction operator (22).

It will now be shown that both the development of learning and the asymptotic level of learning depend on the μ-field. With X not dependent on time to simplify the presentation, the μ-field equation (Table 1, case 2), is written as:

$$\dot{\mu} = a_1\mu + a_2\mu^2 \tag{29}$$

where the coefficients are $a_1 = m$ and $a_2 = -\alpha X^2/p$, $\alpha = abK$, gives the Bernoulli equation: $z' + a_1 z = a_2$, with $z = -1/\mu$, $\mu \neq 0$. The solution is:

$$\mu(t) = \left[-\frac{a_2}{a_1} + \left(\frac{1}{\mu_0} + \frac{a_2}{a_1} \right) e^{-a_1 t} \right]^{-1}. \qquad \mu(0) = \mu_0 \neq 0. \tag{30}$$

A learning process is obtained if and only if the following conditions are fulfilled (Fig. 7 and Table 2):

$$a_1 > 0, \qquad a_2 > 0, \qquad \mu(0) < -a_1/a_2.$$

Because these coefficients have a phenomenological meaning at the molecular level, these conditions correspond to molecular mechanisms which could be evaluated from experiment. This is a way to choose the hypotheses which lead to the learning rules in Table 1.

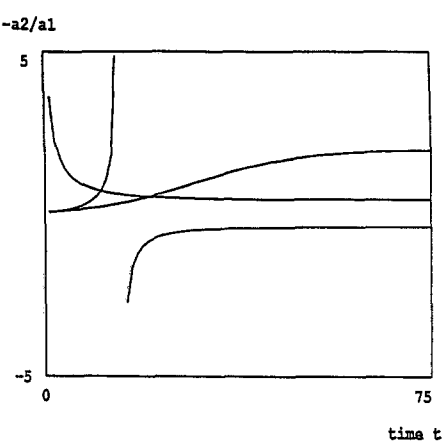

Fig. 7. Solutions of the Ψ-equation (22) (case 2, Table 2). Three outcomes are represented, and correspond to lines 2, 3, 4, in Table 3 ($a_1 > 0$ and $a_2 < 0$, with $\mu_0 < \mu(\infty)$ and $\mu_0 > \mu(\infty)$; $a_1 > 0$ and $a_2 > 0$, $\forall \mu_0$). Only the first case represents learning. Asymptotic values of $\mu(t)$ in each case are denoted $\mu_1(\infty)$, $\mu_2(\infty)$, and $\mu_3(\infty)$

Table 1. An example of Hebbian rules with their hypotheses regarding structure and interaction mechanisms (equations in parentheses). Three cases are considered: (1) Classical Hebbian rules where the variation of synaptic efficacy is in direct relation with the product between input and output, i.e., the source is null; (2) The rules discussed in this paper where the source is in direct relation with the synaptic efficacy; (3) The Easton-Gordon rules which are a particular case of (2). Various learning rules can be studied with particular structures of operators, sources, and non-local interactions

Hypotheses	(1) Classical Hebbian rules	(2) Present rules	(3) Easton-Gordon rules (1984)
	$\dot{\mu}(t) = \alpha X(t) Y(t)$ $Y(t) = \mu X(t)$	$\dot{\mu}(t) = (\alpha X(t) Y(t) + m)\mu$ $Y(t) = -\frac{\mu}{p} X(t)$	$\dot{\mu}(t) = \alpha(X(t) Y(t) - \mu)$ $Y(t) = \mu X(t)$
Structure:			
μ-operator			
$H_I(\mu) \equiv H_I^{\mu}(U_{\mu})$	$U_{\mu}(\delta\mu(\delta\langle R\rangle); \langle\eta(M, R, N)\rangle)$ (16.3)	$U_{\mu}(\mu, \delta\mu(\delta\langle R\rangle); \eta(M, R, N)\rangle)$ (16.2)	$U_{\mu}(\delta\mu(\delta\langle R\rangle); \eta(M, R, N)\rangle)$ (16.3)
Ψ-operator			
$U_{\Psi}(\mu(s', t'), \Psi(r'', T''))$	(22)	(22)	(22)
Source			
Γ_{μ}	0	$m\mu(s(r, r'), t)$	$-\alpha\mu(s(r, r'), t)$
$\Gamma_{\Psi} = p\Psi$	(21)	(21)	(21)
Interaction mechanisms			
Multiplicative			
$\mu_0(s, t) = \langle\xi(s, t)\eta(s, t)\rangle$	(18.2)	(18.2)	(18.2)
$\xi = a\langle X\rangle(t)$	(18.3)	(18.3)	(18.3)
$\eta = b\langle Y\rangle(t)$	(18.4)	(18.4)	(18.4)
Time scale conditions			
$\bar{\Psi}(t) \approx 0$ $\mu(t)\Psi'(t) = \mu(t)\bar{\Psi}'(t)$	(28)	(28)	(28)

Table 2. Example of the study of learning, i.e., the μ-field equation, in case (2) represented in Table 1: properties of the solution $\mu(t)$ of Eq. (30) for various values of parameters a_1 and a_2. We see that only one case, represented in the second line, corresponds to a learning curve, i.e., an asymptotic monotonic variation of $\mu(t)$ (these variations are also shown in Fig. 7, curve 1)

a_1	a_2	μ_0	Existence of t_c	$\mu(\infty)$	Notes
<0	<0			0	$\mu(t)$ *decreases* $\rightarrow -0$
>0	<0	$<-a_1/a_2$	NO	$-a_1/a_2$	Learning curve (Curve 1, Fig. 7)
>0	<0	$>-a_1/a_2$		$-a_1/a_2$	$\mu(t)$ *decreases* $\rightarrow -a_1/a_2$ (Curve 2, Fig. 7)
>0	>0		YES	$-a_1/a_2$	Critical positive time $t_c = \frac{1}{a_1}\ln\left(1+\frac{a_1}{a_2\mu_0}\right)$ (Curve 3, Fig. 7)
<0	>0	$>-a_1/a_2$	YES	0	Critical positive time $t_c = \frac{1}{a_1}\ln\left(1+\frac{a_1}{a_2\mu_0}\right)$
<0	>0	$<-a_1/a_2$		0	$\mu(t)$ *decreases* $\rightarrow -0$

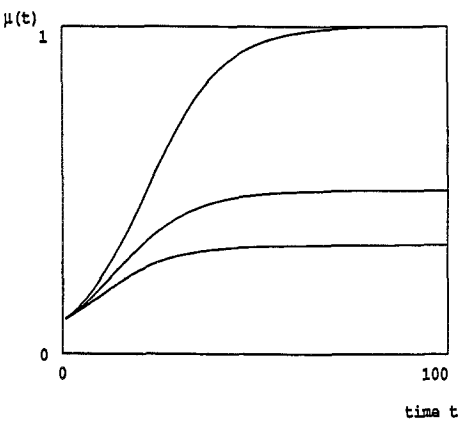

Fig. 8. Learning curves (curve 1, Fig. 7) obtained for various values of the coefficients a_1 and a_2 from the solution of the Ψ-equation (22) which corresponds to case 2 in Table 2. Asymptotic values of $\mu(t)$ in each case are denoted $\mu_1(\infty)$, $\mu_2(\infty)$, and $\mu_3(\infty)$

For a two-input system, with a reinforcement condition (Uttley 1979, Chauvet 1986), a typical two-way learning curve is obtained (Fig. 8). So, it is possible to deduce from the μ-field equation (24.1) the hypothesis assumed by Finkel et al. (1987, p. 744, Eq. (18)) on the relation between pre- and postsynaptic efficacies as a statistical relation between heterosynaptic inputs. With the learning rules here, another result is obtained: as shown in the expression for μ in the above Eq. (30), the level of learning depends on synaptic efficacy parameters like α (Fig. 7).

Some definitions of learning rules: Hebbian and non-Hebbian rules

Within the proposed formalism, the pre-to- postsynaptic neuronal transformation is (i) either imposed sigmoïd-like from Eq. (5):

$$X(r, t) = (1 + E(r, t))^{-1} \tag{31}$$

where $E(r, t) = \exp[\langle \psi(r, T; \mu) \rangle(t)] = \exp[\bar{\psi}(t)]$ and $\psi(r, T; \mu)$ is the solution of Eqs. (24.2) with hypotheses (18.3) and (18.4) which include pre- and post activities; (ii) or calculated from the field equations (24) with the interaction mechanism (18.2), where local pre- and postsynaptic efficacies ξ and η are known from Eqs. (6) and (7) by introducing into them detailed molecular mechanisms.

Hebbian rules for activities, defined by Brown et al. (1990) as "time-dependent, highly local, and strongly interactive mechanism", result from the particular structure given by equations (16), the interaction operator (18), time scales conditions (28), and above all, the assumption of infinite velocity of the field variable. The latter means that non-local effects cannot occur, because, in this case, the interaction operator becomes local in time, then in space. So, the problem is the explanation of these equations at the metabolic level by introducing biophysical mechanisms such as the ones modeled by Kelso et al. (1986) and Zador et al. (1990) in Eqs. (6) and (7). A coarse interpretation could be the long-term internal biochemical modifications of the neuron state when it is activated. In this case, coefficients a and b in the expression of pre- and postsynaptic efficacies (18.3, 4) are important due to their dependence on basal levels of pre- and post synaptic activity. Some experiments could give an answer, for example by modifying the internal neuron state.

From this analysis, let us propose mathematical definitions for learning rules: (i) *Strict or true heterosynaptic effects* result from at least one non-local synaptic contribution to a local one with interaction mechanisms (18.3, 4), which describe the influence of afferent activities, non verified; they imply *non-Hebbian* rules because mechanisms are not "highly local"; (ii) *Active heterosynaptic effects* result from local pre- and postsynaptic mechanisms described by (18.3, 4) as a direct relation with activity: they imply *Hebbian* rules (see, e.g., Lynch et al. 1977); (iii) *Non-linear* Hebbian or non-Hebbian rules result from the structure of operator $U_\mu(s, t, s', t'; \langle \xi \rangle, \langle \eta \rangle)$ in Eq. (16.1), and $U_\mu(r_0, T_0, r, T; \mu)$ in Eq. (22), with different forms of interaction potential function, and from pre- and postsynaptic efficacies (6) and (7) placed in the expression (17) of local synaptic efficacy $\mu_0(s, t)$. Here too, experiments have to determine the physiological implications for this potential function.

Discussion and conclusion

In the field of computational models, two approaches have been considered: the cellular automaton model (represented by the Hopfield model, 1982) and the cellular neural network model (Chua et al. 1988). The first one is made of a massive aggregate of regularly spaced identical elements called cells, which communicate with each other directly only through its nearest neighbors. Each cell has the properties of an automata, i.e., it processes signals in discrete time. The second one has the same fundamental properties as the cellular automaton model, except that it is a large-scale non-linear analog circuit which processes signals in real time. The cellular neural network includes a source term that creates non-linearities, and therefore is similar to the diffusion equation (9). Although cells are not directly connected together, they may affect each other indirectly because of the propagation effects of the continuous-time dynamics in the network. However, two major differences exist between them and the present model: (1) space properties are included via the connectivity and not via the densities, i.e., the topology rather than the geometry is considered; (2) only one

level of organization is taken into account, i.e., the non-linear source term is imposed and not included in the formulation. The consequence of these two differences is that the non-locality of interactions is not considered.

The approach proposed in this paper is deterministic and continuous, but, above all, physiological. Physical approaches in formal neural networks (e.g. Amit 1989), stochastic approaches in biological neural networks (Hervé et al. 1990), in visual cortex (Bienenstock et al. 1982), even with a mean-field approximation (Cooper et al. 1988), constitute another way of representing the evolution of a neural network. For example, it has been shown that the behavior of deterministic or stochastic neural networks can be described by reaction-diffusion equations with mathematical arguments (Cottet 1990). The discretization in space and time lets us use a finite difference method that could be considered equivalent to generating a sequence of spatially ordered compartments. This is the usual technique to represent the spatial distribution of membrane potential conduction with compartmental analysis (Jacquez 1988). For example, this technique was largely used by Traub (1985) for large scale simulations in Hippocampus, and Ambros-Ingerson et al. (1990) for the olfactory cortex. Besides the fact that this transformation from partial differential equations (PDE) into differential equations must satisfy a space-time condition between the discretization steps, the meaning of the parameters in the PDE with several levels of organization would be lost in the transformation, or at least difficult to express. Above all, the non-local effects, which are basic in this approach, could not be interpreted *explicitly* without hierarchical organization. However, here, the problem is mainly the physio-mathematical continuous description of elementary mechanisms, and their integration, rather than the techniques to use for the numerical convergence conditions that have to be satisfied in solving the equations. Comparisons of the four preceding models are summarized in Table 3.

Table 3. Comparison of the neural networks discussed in the text, established between biological and computational models, and according to the representation in time and space, the relation between topology and geometry, the type of state value, real or integer, the number of levels of organization, and the linear character of the dynamics. The present study is distinct from the cited models

	Biological models		Computational models	
Model	Partial differential equations: Field	Ordinary differential equations: Compartment	Cellular neural network	cellular automata
time space	continuous continuous	continuous discrete	continuous discrete	continuous discrete
topology/ geometry	connectivity/ density	connectivity by space discretizing	connectivity	connectivity
state value	real	real	real	binary number
levels of organization	n	1	1	1
dynamics	non-linear (source included in the next lower level)	linear	non-linear (source imposed)	non-linear (network function imposed)

A correlated limiting factor is the geometry of the dendritic and neuronal spaces, including the density of elements. The continuous approach imposes these anatomical data as geometric functions that can explicitly be introduced in the equations, and thus constitutes a fundamental part of the formulation. Indeed, because they result from the study of anatomical data, they can be complicated space functions.

With the present analysis, field theory lets us represent quantitative relationships between phenomena that evolve in a neural network, e.g., densities of active synapses and densities of active neurons. The ultimate goal of this approach will be to deduce an explicit formulation of such relationships to incorporate their interpretation within general laws of neural network dynamics. An important consequence of the explicit formulation is that a new representation with several levels of organization is needed, and thus, new concepts, such as functional interaction, non-symmetry, and non-locality are defined.

Our choice follows from the fact that two explicit levels of organization are imposed by: (i) a description of the physiological mechanisms that occur *in the sources*, and (ii) a description of the *non-local interactions* between sources and sinks. The description of the biological neural network, which has to take into account the physiological mechanisms, has to include these mechanisms. The physiological description of the neural network leads to equations with a physiological meaning for the model parameters. The macroscopic approach deduced from the principle of conjunction is well adapted to the study of discrete neural networks such as connectionist models (Rumelhart et al. 1986) used in the field of connectionism. But non-linearities and learning rules are generally imposed on the system. With our continuous approach, which includes anatomy, geometry, and elementary physiological mechanisms on several levels of organization: (i) the dynamics of biological neural networks can be described with implicit learning rules (Chauvet et al., in progress); (ii) an explicit formulation of a monosynaptic connection between pre- and postsynaptic neurons leads to an interpretation of experimental input/output curves that describe the extracellular field potential vs. stimulating intensity (Chauvet et al. 1990); (iii) non-linearities emerge from the formulation with the molecular mechanisms included in the source terms of the fields; and (iv) collective behaviour of cooperatively coupled cell assemblies (Singer 1988) at each level of organization, and selection of groups of neurons (Edelman 1981, Edelman et al. 1984) can be directly deduced with the field theory proposed here (Chauvet 1990).

Appendix A: Nomenclature

$a_1 = m$, $a_2 = -\alpha X^2/p$ coefficients of the learning Bernoulli equation
m source term coefficient for the μ-equation
p source term coefficient for the ψ-equation
v_a transport velocity of the ψ-interaction
v_μ transport velocity of the μ-interaction
$A(s, s')$ space attenuation factor for the synaptic efficacy, in units per time
$B(r_0, T_0, rT)$ space attenuation factor for passive membrane voltage propagation
D diffusion coefficient with subscripts:
  0 for local
  1 for non-local
$D_R(r_0)$ recombination of subspaces $D_s(r', r_0)$ when r' varies

$D_s(r', r_0)$ space constituted by the synapses localized at $s(r', r_0)$ in the r'-neurons connected with the r_0-neuron
$F(\langle \Psi(r, T) \rangle(T))$ input-output network function
H^{Ψ} field operator
H_I non-local interaction operator
H_I^{Ψ} non-local interaction operator in neurons space
U_{μ} potential kernel function of the synapse-synapse interaction operator
U_{Ψ} potential kernel function of the neuron-neuron interaction operator
$X(r, T)$ instantaneous local somatic activity
$\mathfrak{R}$ local transformations in neuron source
$\alpha = abK$ learning coefficient
δ Dirac function
η instantaneous postsynaptic efficacy
η^0 basal postsynaptic efficacy
ξ instantaneous presynaptic efficacy
ξ^0 basal presynaptic efficacy
$\mu(s, t)$ (global) synaptic efficacy in the s-space (time scale $\{t\}$)
$\Psi(r, T)$ local somatic depolarization in the r-space (membrane potential)
$\pi(s, r; r_0)$ density-connectivity function
σ instantaneous non-local synaptic efficacy (time scale $\{T\}$)
σ_0 instantaneous synaptic efficacy
$\Gamma(r, T)$ source
Γ_{Ψ} source term in the ψ-equation
ψ_{μ} source term in the μ-equation
$\Phi(s, t)$ postsynaptic local membrane potential
∇ gradient operator
$\langle \rangle(t) = (t)$ time average at time t

Acknowledgements. I wish to thank Drs T. W. Berger and E. F. Thiels for helpful discussions. This research was supported by grants from DRET (88/1194) and INSERM (88/9002) (France), and the Conseil General de Maine et Loire.

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