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Review Article

Connectivity and Dynamics of Neural Information Processing

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Abstract

In this article, we systematically review the current literature on neural connectivity and dynamics, or equivalently, structure and function. In particular, we discuss how changes in the connectivity of a neural network affect the spatiotemporal network dynamics qualitatively. The three major criteria of comparison are, first, the local dynamics at the network nodes which includes fixed point dynamics, oscillatory and chaotic dynamics; second, the presence of time delays via propagation along connecting pathways; and third, the properties of the connectivity matrix such as its statistics, symmetry, and translational invariance. Since the connection topology changes when anatomical scales are traversed, so will the corresponding network dynamics change. As a consequence different types of networks are encountered on different levels of neural organization.

Au: Provide 4–6 Keywords per original instructions.

Introduction

Information in the brain is processed on a variety of scales, from the synaptic level in the form of long-term potentiation (Markram et al., 1997), through spike timing of single neurons (Abeles, 1991), to the synchronization of neural networks in different areas (Singer and Gray, 1995; Bressler and Kelso, 2001). Several mechanisms for neural information processing have been postulated based on the evolution of neuroelectric activations. For instance, the emergence of a pattern of synchronized neural activity has been proposed as a mechanism for the grouping of spatially distributed processes (Singer and Gray, 1995). Specific network patterns of cortical areas (e.g., insula, inferior parietal lobule) and subcortical areas (superior and inferior colliculi) have been identified as being activated during the integration of multisensory information (*see* Calvert, 2001 for a review). On the other hand, there are situations in which neural activity evolves spatiotemporally, but it remains unclear if information is actually being processed. For instance, Paul Nunez (1995) refers to the notion of brain states observed in the electroencephalogram (EEG) in association with cognitive or mental states, e.g., such as the individual sleep stages and the REM sleep. These states are related to the dynamic properties of the system, e.g., REM sleep is characterized by the occurrence of sleep spindles which are sequences of amplitude modulated 8–12 Hz oscillations in the EEG. Similarly, during partial epileptic seizures, the propagation of seizure activity from the pathological source interferes or suppresses normal information processing in other cortical regions. These examples show that neural information processing and neural activity progression are not synonymous, but related (*see also* Jirsa, 2004 for further discussion).

The temporal dynamics and spatial distribution of neural activity will be a function of the underlying cortical network connectivity. Not only has the connectivity a major impact on the direction of information processing, but also uses the brain changes of connectivity as a mechanism for learning. In this review, we wish to focus on how the qualitative spatiotemporal network dynamics change as the connectivity changes as a function of its topology and its synaptic weights. The connection topology addresses the existence of a set of connections, whereas the synaptic weights introduce a quantitative weighting of these connections, as well as the distinction between excitation and inhibition. A notion of physical space in neural network models is desirable, since the effective geometry in which a dynamic occurs is determined by the connection topology and its boundary conditions. For instance, an activation pulse in a network of neurons may be appropriately described as a pulse traveling on a closed line for one type of connectivity, and as pulse traveling on a sphere for a different type of connectivity. Using simple examples, we will discuss how such effective geometry of a system arises from its connectivity and boundary conditions. In real networks, the transmission along pathways will require time, since the transmission speeds will not be infinite. These transmission delays increase the complexity of the system by raising the number of degrees of freedom to infinity, because the progression of the system's dynamics depends on a continuous interval of past activations (hence infinitely many thereof). We will derive a general network equation for the dynamics in neural systems and discuss how the dynamics change under varying connection topology and transmission speeds. Statistical properties of network connectivity will be discussed in the context of the statistics of its related network dynamics. There are three major aspects under which we will compare the network models: The nature and dynamics of the network nodes, the connection topology, and the presence of time delays via transmission. The stability of networks with no delay, or equivalently with infinite propagation speeds along axons, has been studied for various types of network nodes that include fixed point, limit cycle, and chaotic attractors. If all oscillator nodes are identical, then general criteria for the stability of the synchronized network state have become available for arbitrary connectivity (*see* "Infinite Propagation Speed, Arbitrary Connectivity"). If the network oscillators are non-identical, then general criteria for stability of the synchronized state still exist, but strong constraints must be imposed on the connectivity (such as global coupling). If delays via propagation are introduced, then most research has focused on fixed-point attractors as network components rather than limit cycle or chaotic attractors. Here, general criteria for the stability of the synchronized fixed point state of the network are available for arbitrary connectivity with discrete and distributed delay.

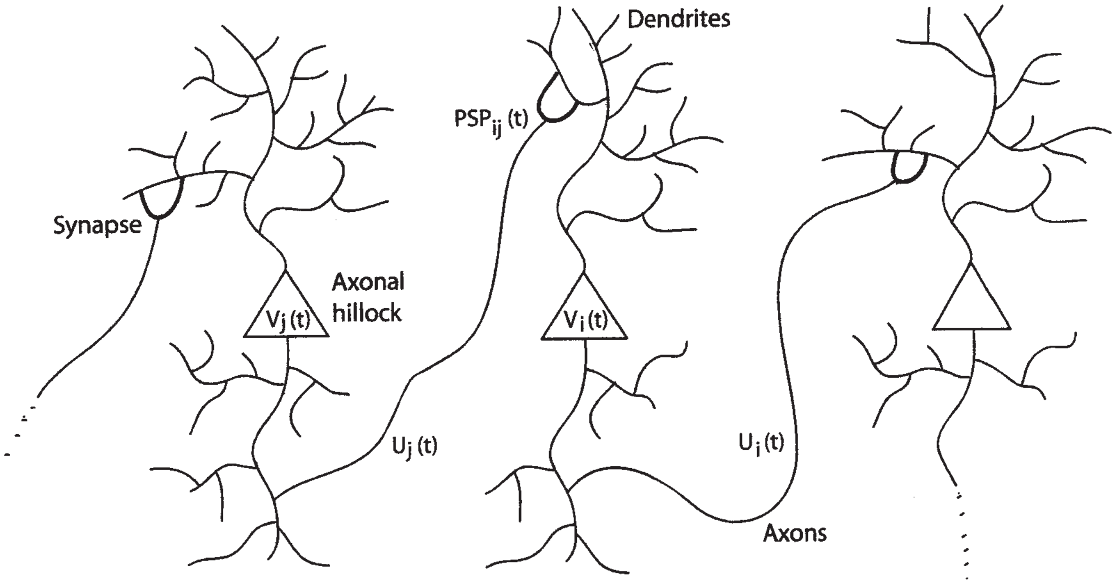

Fig. 1. Network constituents: firing rate $u_i(t)$ at neuron i, membrane potential $v_i(t)$, postsynaptic potential $PSP_{ij}(t)$.

General Neural Network Dynamics

What Constitutes a Network?

For our purposes we consider the following network constituents: $v_i(t)$ is the membrane potential of neuron i at time t, which means we consider single compartment models of neurons only, i.e., a neuron is a zero-dimensional entity with no spatial expansion, implying that these effects are not relevant for a network dynamics (but *see* comments in "General Network Dynamics"). $u_i(t)$ is the firing rate of neuron i at time t. Its precise form will depend strongly on the underlying neuron model that has been chosen. However, it is commonly agreed that the firing rate is a function S_i of the membrane potential $v_i(t)$. The firing rate will be small (but not necessarily continuous) for small membrane potentials, increases for increasing values of $v_i(t)$ and finally saturates for large $v_i(t)$. Since our current neuron model does not have any spatial extension, we can readily write $u_i(t)=S_i(v_i(t))$. $PSP_{ij}(t)$ is the postsynaptic potential which has been generated on the postsynaptic side of neuron i as a consequence of the arrival of a presynaptic action potential spike (*see* Fig. 1). The latter was generated by neuron j and traveled along the axon until it arrived at the synapse on the dendritic tree of neuron i. The total postsynaptic potential $\phi_{ij}(t)$ of this particular synapse connecting neurons i and j is a function of the $PSP_{ij}(t)$. We assume the shape of the $PSP_{ij}(t)$ to be invariant and that the incoming firing rate u_j is slower than the synaptic resolution which is a few milliseconds. Then we may write $\phi_{ij}=\Sigma_k PSP_{ij}(t-t_k)$ where t_k denotes the arrival time of the k-th action potential spike.

Fig. 1

Most neural network models are composed of the basic elements illustrated in Fig. 1. In the subheading "General Network Dynamics," we will derive equations that capture most of their dynamic characteristics. However, there are multiple scales of organization traversing from the single neuron to the scalp topographic scale of the EEG and magnetoencephalogram (MEG). As these scales change, the connection topologies change from probabilistic to structured and hierarchical, as expressed by the connectivity matrix. We will refer to a connectivity

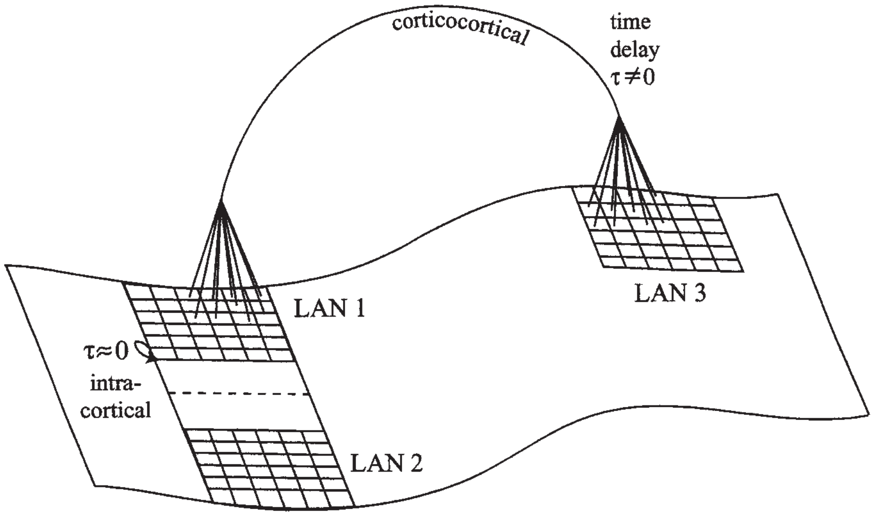

Fig. 2. Local area networks (LAN) are coupled locally (intracortical) with no time delays and globally (cortico-cortical) with time delays.

matrix as homogeneous if it is translationally invariant in physical space. If this invariance is broken, it is called heterogeneous. Generally speaking, to the extent that nonhomogeneous properties are considered in spatiotemporal pattern formation in physics or chemistry, they are typically introduced as spatially varying parameters or inputs to a homogeneous system (*see* e.g., Hendrey et al., 1999 for a recent example). In biology, translational variance is more common: Valentino Braitenberg and Almut Schüz (1991) showed that the short-range intracortical fiber system of the mouse cortex actually shows a distribution with a connection probability which decays exponentially over the distance. The long-range corticocortical fiber system displays the same exponential decay of the connection probability over distance, but not its homogeneity and its distribution rather appears to patchy (Braitenberg and Schüz, 1991). This connection topology implies that neural activity progression, which is synonymously used to information processing in the context of this review, occurs not only along neighborhood-based patterns of connectivity through the cortical gray matter, but also along projecting pathways across long distances through the white matter (*see* Fig. 2). Rolf Kötter and Friedrich Sommer (2000) performed a nice study to test this hypothesis. They compared the activations generated by network models with different connection topologies to activations obtained from electrophysiological measurements of the global spread of epileptic activations following intracortical stimulation. As a general result, it turns out that a network with large-scale connectivity derived from experimental data reproduces cortical propagation of activity better than networks with purely neighborhood-based or random connectivity. These results certainly emphasize the importance of large-scale connectivity, however, they do not exclude the importance of neighborhood-based connectivity owing to large experimental errors and a very specific example data set (epileptic activity). Tagamets and Horwitz (1998) developed a model approach based on globally coupled networks which characterize the sequence of information processing in the occipitotemporal pathway associated with object vision. This pathway is viewed to consist of five major areas

Fig. 2

(LGN, V1/V2, V4, TEO/IT, and PF) that are represented by local networks composed of Wilson-Cowan-like units (*see* "Finite Propagation Speed, Symmetric and Translationally Invariant Connectivity"). Here transmission delays between the units have not been accounted for, but rather have been absorbed into the intrinsic dynamics of the individual networks such as increased rise or decay times of activations. Such absorption is possible as long as the complexity of the dynamics is not increased any further. Under these conditions, a variety of large-scale phenomena observed in positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) were successfully reproduced (Tagamets and Horwitz, 1998; Horwitz et al., 2000; *see also* Jirsa et al., 2002 for a discussion on large-scale brain imaging and network dynamics).

In terms of neural information processing, there seems to be the following generally agreed upon picture among the community to arise (Freeman, 1975; Mallot and Brittinger, 1989; Mallot and Giannakopoulos, 1996; Freeman, 1992; Tagamets and Horwitz, 1998; Bressler and Kelso, 2001; Bressler, 2002, 2003): Functional-specific areas, such as primary sensory areas or multisensory convergence zones, are represented by local area networks (LAN, *see* Bressler, 2002 and Freeman's KI sets [Freeman, 1992]) which are characterized by a connection topology with local excitation and lateral inhibition. The extent of the LAN is small enough to ignore time delays via propagation. The synaptic weights are often symmetric and sometimes translationally invariant. The translational invariance may be broken to allow for the spatial and sometimes topographic encoding of information while still being subject to change via learning. The LANs are coupled either near to each other through neighborhood-based connectivity, or via the white matter across large distances. In the latter case, long propagation delays of up to 200 ms have to be considered. The connection topology is very specialized, hence asymmetric and translationally variant. Figure 2 illustrates these connectivity and dynamic features. In this picture, we do not commit to a particular mechanism for information processing in the brain such as binding (Singer and Gray, 1995) or the existence of information convergence zones (Calvert, 2001), but rather define the framework and the conditions in which such mechanisms may be embedded.

General Network Dynamics

In the following development of a general network equation which involves all of the constituting elements discussed in the previous subheading, we will follow the lines of thought in Ermentrout, 1998 and extend some of these. Our presentation does not include all potential mechanisms that qualitatively affect the spatiotemporal network dynamics, but we hope the major elements are there. Most recently, there has been some discussion on the dispersive properties of the dendritic integration process and its potential for the generation of a variety of new spatiotemporal phenomena (Bressloff, 1996). We do not include these processes into our considerations of general network dynamics. Our underlying neuron model is point-like and our discussion of the interplay of structure and dynamics is based on this assumption. The inclusion of spatially distributed dendritic trees and finite speeds along them will probably not change our qualitative results in this review, but it will certainly make the underlying equations more complex and change their interpretation. Here we choose not do so, but wish to raise the reader's awareness of this issue.

Our basic assumptions are as follows: During the time dt the number of action potentials arriving at the synapse, which connects neurons i and j, is $u_j(t)dt$. The firing rate $u_j(t)$ is a function of the membrane potential, $u_j(t)=S_j(v_j(t))$, as defined in the previous subheading. Given the above assumption of independently arriving action potentials, the process of summing the action potentials is identical to integration over time

$$\begin{aligned}\phi_{ij} &= \sum_k PSP_{ij}\left(t-t_k\right)\\ &= \int_{t_0}^{t} PSP_{ij}(t-s)\,u_j\left(s-\delta_{ij}\right)ds\end{aligned} \tag{1}$$

where t_0 is some initial time and δ_{ij} is the time during which the action potential traveled from the axonal hillock of neuron j to its presynaptic terminal of neuron i. Because all internal neuronal structure has been ignored (such as the dendritic tree and the finite propagation times along dendrites), the membrane potential reads $v_i(t) = \Sigma_j\, \phi_{ij}(t)$ and its dynamics are defined with $u_i(t) = S_i(v_i(t))$ and Equation 1.

$$v_i(t) = \sum_j \int_{t_0}^{t} PSP_{ij}(t-s) S_j\left(v_j\left(s-\delta_{ij}\right)\right)ds \tag{2}$$

Here the membrane potential $v_i(t)$ is considered the variable of interest. Equation 2 defines our general network equation. Equivalently, the firing rate formulation of equation 2 may be obtained as

$$\begin{aligned}u_i(t) &= S_i\left(v_i(t)\right)\\ &= S_i\left(\sum_j \int_{t_0}^{t} PSP_{ij}(t-s)\,u_j\left(s-\delta_{ij}\right)ds\right)\end{aligned} \tag{3}$$

This variable change has been referred to as S-Σ-exchange by Steven Grossberg (1998). Excitatory synapses have a positive postsynaptic potential, $PSP_{ij} > 0$, inhibitory synapses a negative postsynaptic potential, $PSP_{ij} < 0$.

Under additional constraints upon the network constituents, our general network equation may be simplified. Most of the time course of the postsynaptic potential $PSP_{ij}(t)$ depends only on the postsynaptic cell i which is captured by $PSP_{ij}(t) = w_{ij} G_i(t)$ where w_{ij} is a constant scaling factor, the synaptic weight. $G_i(t)$ is identified with the time course of the diffusion process of the transmitters in the synaptic cleft after release at the presynaptic terminal and their binding process to the receptors on the postsynaptic terminal. Then the dynamics of $v_i(t)$ is given by

$$v_i(t) = \int_{t_0}^{t} \underbrace{G_i(t-s)}_{\hat{K}} \sum_j w_{ij} S_j\left(v_j\left(s-\delta_{ij}\right)\right) ds \tag{4}$$

where the inverse $\hat{K}^{-1}$ of the integral operator $\hat{K}$ will be a sum of differentials with constant coefficients c_n

$$\begin{aligned}\hat{K}^{-1} v_i(t) &= \sum_{n=0}^{\infty} c_n \frac{\partial^n}{\partial t^n} v_i(t)\\ &= \sum_j w_{ij} S_j\left(v_j\left(t-\delta_{ij}\right)\right)\end{aligned} \tag{5}$$

if $G_i(t-s)$ is an exponential or polynomial. For example, a simple, but often used, integral kernel is $G_i(t-s) = e^{-(t-s)/\tau}$ which leads to the following differential equation for sufficiently small time delay, $\delta_{ij} \approx 0$ with $c_0 = 1$, $c_1 = \tau$ and $c_n = 0$; $n \geq 2$:

$$\tau \dot{v}_i = -v_i + \sum_j w_{ij} S_j\left(v_j(t)\right) \tag{6}$$

Here τ defines the intrinsic time scale on which $v_i(t)$ evolves. Equations of this form will be discussed in more detail in "Infinite Propagation Speed, Symmetric and Translationally Variant Connectivity," but it is immediately evident that the intrinsic dynamics of equation 6 is a fixed point attractor, $v_i = 0$, which may or may not be destabilized by the interactions with the other neighbors. A more general situation, with an active rather than just passive intrinsic dynamics, could be accomplished by the following extension:

$$\tau \dot{v}_i = f(v_i) + \sum_j w_{ij} S_j\left(v_j(t)\right) \tag{7}$$

where $v_i \in R^m$ is the time-dependent, but now m-dimensional neural activity at a location denoted by the index i. The intrinsic dynamics of the neural activity v_i is characterized by

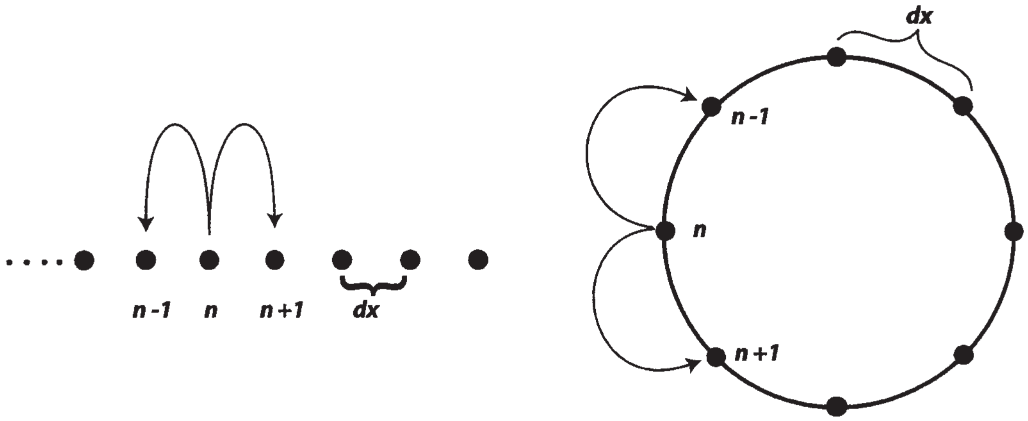

Fig. 3. One-dimensional connectivity with open boundary conditions (left) and periodic boundary conditions (right).

the function $f(v_i)$ which defines the time course of v_i if all other connections were severed.

Connectivity and Dynamics: Foundations

The effective geometry in which the dynamics of a system evolves is determined by its connectivity matrix w_{ij} as used in equation 4, together with the boundary conditions of the system. The following three aspects play a critical role in the characterization of the resulting dynamic system:

1. Finite propagation times along axons. Most modeling approaches ignore the time delays caused by propagation times. This assumption is valid if the time delay, (τ = traveled distance/propagation speed) is small compared to the time scale on which the system is studied. Large area networks that are used to describe inter-areal information processing, generally do not satisfy this condition.
2. Symmetry of connectivity, and
3. Translational invariance of connectivity determining the mathematical methods required to describe the system.

A symmetric connectivity matrix is given when its elements satisfy the condition $w_{i+a,j+a} = w_{i-a,j-a}$, where *a* is a positive integer. The stronger constraint of translational invariance requires that the values of the matrix elements are a function of the distance $|i-j|$ only rather than a function of the absolute value of the indices *i, j*. This formulation is mathematically precise. A slightly weaker formulation for near-neighbor connections is given in the following.

An effective one-dimensional geometry will be achieved, if the neural sites *i* with the activity $v_i(t)$ may be indexed such that elements w_{ij} in the connectivity matrix may be ordered "around" its diagonal within a width which remains approximately the same along the diagonal (*see also* Fig. 6 for an illustration of the connectivity matrix). A meaningful choice of an index scheme will identify the neighborhood as shown in Fig. 3. As long as most of the elements of the connectivity matrix follow this distribution scheme, the effective physical geometry will be one-dimensional. In the following subheadings, different types of deviations from this ideal case shall be discussed. The boundary conditions will determine if the one-dimensional space, the line, will be closed for periodic boundaries or open otherwise. Open boundaries are either zero amplitude ($v_1 = v_N = 0$) or zero flux boundaries ($v_1 = v_2$ and $v_N = v_{N-1}$).

Fig. 3

In the particular example of Fig. 3, the connectivity has been chosen to resemble a diffusion

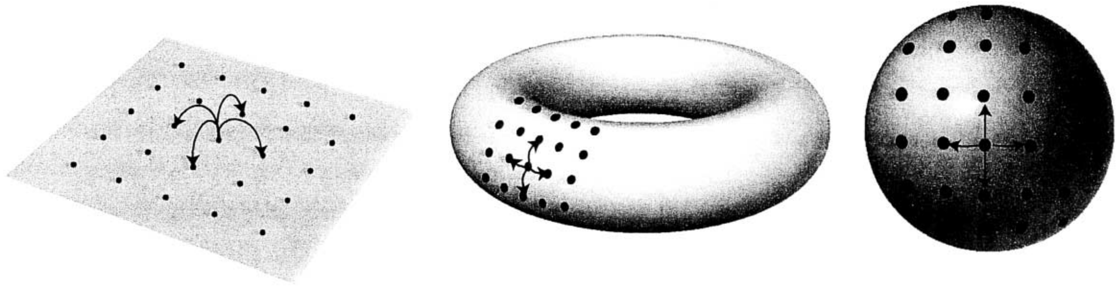

Fig. 4. Two-dimensional connectivity with open boundaries (left), double periodic boundaries (torus, middle) and spherical boundaries (right).

process in equation 6: the diagonal elements are $w_{ii} / -2D/dx^2$, its nearest neighbors are $w_{i,i+1} = w_{i,i-1} = D/dx^2$ and all other elements are zero. Here D is the diffusion constant. An intrinsic spatial length scale has been introduced by scaling the physical space x between two neighboring neural sites which assumes a uniform spacing dx of the nodes. If uniformity is not given, then the spatial scale becomes a function of the indices itself, $dx = dx(i, j)$. In the continuous limit $dx \rightarrow 0$ and $v_i(t) \rightarrow v(x, t); x \in R$ the dynamics of equation 6 may be written as:

$$\tau\dot{v}(x,t) = -v(x,t) + D\Delta S(v(x,t)) \qquad (8)$$

where $\Delta = \partial^2/\partial x^2$ is the one-dimensional Laplace operator. Systems with the dynamics of equation 8 are known as reaction-diffusion systems and are less common in neuroscience, but frequently used in the general pattern formation literature (*see* Haken, 1975; Cross and Hohenberg, 1993 for excellent reviews).

An effective two-dimensional geometry will be obtained if an indexing scheme is adopted in which the neighborhood relations of the connectivity matrix may be expressed as shown in Fig. 4. Again, as long as most of the elements of the connectivity matrix are ordered "around" its diagonal within an approximately constant width for each space dimension, then the effective geometry will be a two-dimensional surface. If the boundary conditions are periodic, then closed surfaces are obtained which z torus or a sphere or ellipsoid. In principle, it is possible to construct many two-dimensional closed surfaces with arbitrary topologies, but these three (torus, sphere, and ellipsoid) are the most common in applications. Open two-dimensional surfaces typically obey zero amplitude or zero flux boundary conditions.

Fig. 4

Connectivity, Statistics, and Dynamics

A rapidly evolving topic of research is the study of complex networks that focus on the statistical mechanics of network topology and the generation of such network topologies. Here is a strong word of caution: In this literature, the processes to generate the network structures (such as algorithms) are referred to as "dynamics" (Watts and Strogatz, 1998; Albert and Barabási, 2002). This dynamics obviously does not refer to the evolution of activity patterns generated by the interplay of the network's intrinsic dynamics and interactions. The relationship of the latter—network structure and the dynamics of network activity—is the focus of this review. Hence, whenever we refer to network dynamics in this review, we mean network activity evolution. For completeness, we provide a brief summary of some of the main ideas in the field of the statistical mechanics of complex networks in the following subheading. Still, there is a limited number of statistical approaches which in fact attempt to connect the statistics of network structure to dynamics. These are discussed in "Statistically Inspired Approaches to Network Dynamics." In the subheadings thereafter we will discuss how different aspects of connectivity, primarily symmetry, translational invariance, and transmission speeds, influence the global dynamics of a network system.

Statistical Mechanics of Complex Networks

Network topologies are characterized from the perspective of statistical mechanics by the number of nodes N and by the number k of connections (or "edges") to other nodes. Since not all nodes in the network have the same number of connections (referred to as the node degree), the spread in the node degrees is characterized by a distribution function $P(k)$, which gives the probability that a randomly selected node has k connections. Besides the degree distribution, two other measures characterize network topologies: the average path length l of a network is defined as the number of edges in the shortest path between two nodes, averaged over all pairs of nodes (Watts and Strogatz, 1998, Albert and Barabási, 2002). The clustering coefficient C_i of node i that has k_i edges which connect it to k_i other nodes, is defined as

$$C_i = \frac{2E_i}{k_i(k_i - 1)} \tag{9}$$

where E_i is the number of edges that actually exist between the k_i nodes. The total number of all possible edges is $k_i(k_i–1)/2$. The clustering coefficient of the whole network is the average of all the C_i from every node i. Albert and Barabási wrote a nice review on the statistical description of network topologies (Albert and Barabási, 2002). Here we summarize the basic properties of the three main network types.

Random networks have been studied by Erdös and Rényi using random-graph theory (Erdös and Rényi, 1959). A graph is a pair of sets *{P,E}* where P is a set of N nodes and E is a set of edges connecting two elements of P. The graph is typically illustrated by dots corresponding to nodes and by lines corresponding to edges. Every pair of nodes is connected with equal probability p and the majority of nodes have approximately the same degree, close to the average degree $\langle \mathrm{k} \rangle$ of the network. The degree distribution of the random network is a Poisson distribution with a peak at $P\langle \mathrm{k} \rangle$. Random-graph theory studies the properties of the connection probability associated with graphs with N nodes as $N \rightarrow \infty$.

Scale-free networks are characterized by a hierarchy of connections that is self-similar across different scales or levels of structure and thus obeys a power law degree distribution of the form $P(k) = Ak^{-a}$ (Barabási and Albert, 1999). In a scale-free network, structures extend over a wide range of scales. Such network topologies develop when new connections are added preferentially to nodes that already have many connections (Huberman, 1998; Huberman and Adamic, 1999; Albert and Barabási, 2002).

Small world networks are characterized by a short characteristic path length and a high clustering coefficient (Albert and Barabási, 2002). Given the same number of nodes N and the average number of connections $\langle \mathrm{k} \rangle$ both a random network and a small world network have a similarly small average path length l. Under these same conditions a small world network has a higher clustering coefficient than a random network.

Statistically Inspired Approaches to Network Dynamics

So far, none of the primarily statistically oriented research discussed in the previous subheading has made any attempt to connect the structural properties of connectivity to dynamics. Here, Olaf Sporns and colleagues (Sporns et al., 2000; Sporns and Tononi, 2002; Sporns, 2004) have made some relevant contributions.

Their strategy is the following: Choose a simple, though generic network which is well-suited to be solved computationally for large neuron numbers such as

$$u_i(t+dt) = S\left(\sum_j w_{ij} u_j(t) + n(t)\right) \tag{10}$$

where $n(t)$ is Gaussian noise. Equation 10 is similar to the system in equation 3 for the case of no time delays. The transfer function S is identical for all neurons and is a unimodal saturating nonlinearity such as a hyperbolic tangent. Sporns drives the system with noise for various connection topologies and calculates the covariance matrix, which is interpreted as synonymous to a *functional connectivity*. The connection topology is referred to as *structural connectivity*. Connection matrices with different topologies are used such as a random synaptic weight distribution, as well as distributions optimized for global statistical measures such as entropy or complexity. The corresponding network dynamics and hence functional connectivity is solved computationally and shows characteristic differences as a function of the underlying structural connectivity. In particular, connectivity structures optimized for the complexity measure employed by Sporns and colleagues (Sporns and Tononi, 2002; Sporns, 2004) show the greatest amount of clustering in the functional connectivity. This implies that such networks have the largest capability to be simultaneously functionally segregated and functionally integrated. It is notable that in Sporns' data, both functional and structural connectivity have most of their entries around the diagonal and display strong symmetry after reordering of the indices. As discussed in "Connectivity and Dynamics: Foundations," this ordering scheme and its connectivity matrix suggest an effective one-dimensional geometry of the underlying system. It is an open question to how far the symmetry constraint and the general results are actually a consequence of the particular choice of the model in equation 10 and how these results would change if a richer dynamics were allowed. For instance, Lago-Fernández et al. (2000) performed a similar computational study of coupled Hodgkin-Huxley neurons (Hodgkin and Huxley, 1952) that addressed at least the beginnings of this question. They also used the three connection topologies (random, regular, and small world) and found that the small world topologies allow the network to respond with fast rise times (a feature known from random networks), but still show coherent oscillations (as known from regularly connected networks). Similar approaches are being taken to tackle related problems in structure and dynamics of different fields such as ecological and social networks (Barabási and Albert, 1999) or in networks of gene regulation (Chen and Aihara, 2002; Shehadeh et al., 2004).

Connectivity and Dynamics

Infinite Propagation Speed, Arbitrary Connectivity

The current research in the field of synchronization of dynamic network systems primarily focuses on the two extremes of a range: from networks of identical nodes with complex intrinsic dynamics and arbitrary connectivity to networks of non-identical nodes with complex intrinsic dynamics, but constrained connectivity. Here we will discuss some results on the former case. The latter case is nicely discussed in Kaneko, 1997 for globally coupled multiattractor systems and in Ott et al., 2002 for globally coupled chaotic and periodic oscillators. De Monte et al. (2003) developed a systematic approach to derive mean field equations for globally coupled non-identical dynamic systems with no delay. Their approach is based on a variation of the dispersed parameters and provides a low-dimensional set of equations valid for the coherent regime of network activity. Gerstner, van Hemmen, and colleagues (Gerstner, 2000; Eggert and van Hemmen, 2001;

Gerstner and Kistler, 2002) provided a series of papers in which they discuss a general approach based on Volterra integral equations which is able to capture the population dynamics of most of the common neuron models in the literature. Similarly, Peter Érdi (Arbib and Érdi, 2000; Kiss and Érdi, 2002) and his group have established a population approach, the so-called POP model, which uses stochastic methods, in particular the Fokker-Planck equation, to describe the evolution of neural ensemble activity. In the following, we discuss some general results that address the stability and synchronization properties for all models of this type, that is, identical nodes with arbitrary connectivity but without delay.

Pecora and Carroll (1998) developed an approach which determines the stability of the synchronous state of an oscillator network of the form in equation 7. The coupled oscillators are all identical and may include fixed point, limit cycle, and chaotic attractors. The same coupling function is used to couple all oscillators and is assumed to be approximated linearly, $S_j(v_j) = S(v_i) \approx v_i$. The nodes are coupled in an arbitrary a fashion with $w_{ij} \in R$ where W is the matrix of coupling coefficients $\{w_{ij}\}$. The $N-1$ constraints $v = v_1 = v_2 = \ldots = v_N$ define the synchronization manifold which is invariant if $\Sigma_j w_{ij} = 0$. To determine the stability of the synchronized state, Pecora and Carroll consider the variations ξ_k which all are transverse to the synchronization manifold. The dynamics of the ξ_k is given by the variational differential equation:

$$\dot{\xi}_k = \left[Df + \gamma_k DS\right]\xi_k \tag{11}$$

where Df and DS are the Jacobian matrices of f and S, and γ_k is the k-th eigenvalue of the connectivity matrix W. For $k = 0$, the variational equation for the synchronization manifold is given with $\gamma_0 = 0$. The Jacobian functions Df and DS are the same for each k, since they are evaluated on the synchronized state obtained from the intrinsic dynamics of equation 7. Hence each degree of freedom, that is the synchronized and transverse components, may be evaluated separately. Now, the following is the crucial conceptual step: The master stability equation

$$\dot{u} = \left[Df + (\alpha + i\beta)DS\right]u \tag{12}$$

resembles the structure of equation 11 with the same functions Df and DS. The maximum Lyapunov exponent (or any other convenient measure of stability) of the variations in equation 12 is identical to the maximum transverse Lyapunov exponent. It is calculated as a function of the constants α and β and plotted as a surface over the complex plane spanned by α and β. This surface is referred to as the master stability function. Figure 5 shows an example that we derived for the dynamics of coupled chaotic Hindmarsh-Rose neurons (Hindmarsh and Rose, 1984). Negative Lyapunov exponents indicate stability; positive Lyapunov exponents indicate instability. If every eigenmode of the dynamic system is stable, then the synchronized state is stable, or else it is not. This means that the stability of every dynamic system may be determined by numerically calculating its synchronized state and then determining its master stability function. Given a connectivity matrix W, its N eigenvalues γ_k may be calculated and then their location identified in the master stability function. If all eigenvalues are in the stable regimes, then the synchronized state is stable. This approach provides a beautiful computational tool to identify the stability of dynamic systems with arbitrary connectivity.

Fig. 5

Chen et al. (2003) present a related approach which provides explicit analytical constraints on the coupling strengths by combining the master stability function and Gershgörin's disk theory. Gershgörin's disk theory states that all eigenvalues of an $N \times N$ matrix $W = P\{w_{ij}\}$ are located in the union of N disks (called

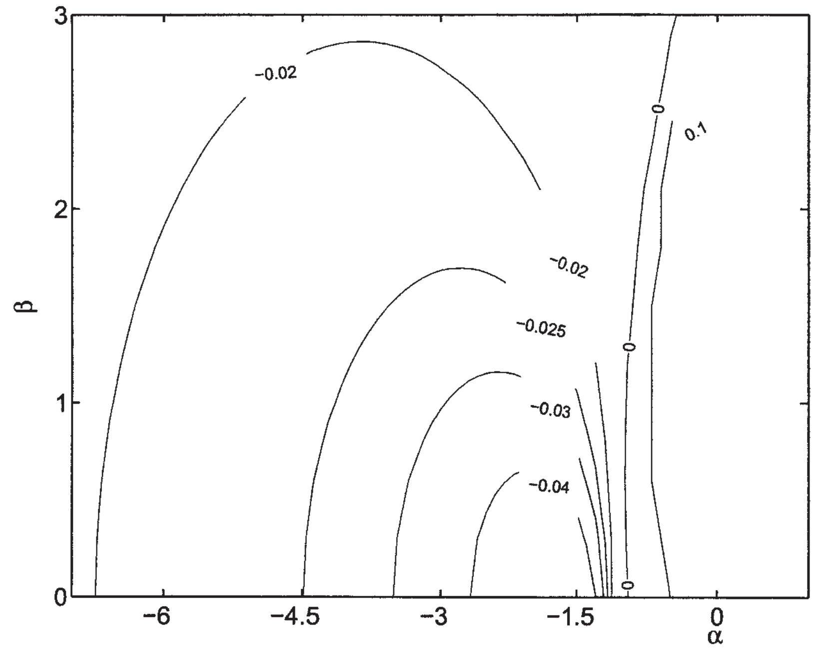

Fig. 5. The master stability function for the Hindmarsh-Rose system is plotted.

Gershgörin disks), where each disk is given by

$$\left\{ z \in C: \left| z - w_{ii} \right| < \sum_{j \neq i} \left| w_{ji} \right| \right\}, i = 1, \cdots, N \qquad (13)$$

Applying Gershgörin's disk theorem to the variational equation (11), the following sufficient stability constraints are obtained:

1. The center of every Gershgörin disk of $D^k = \{d^k_{ij}\}$ lies in the stability zone Ω where $d^k_{ij} = w_{ij} - w_{kj}$. That is, $(w_{ii} - w_{ki}, 0) \in \Omega$.
2. The radius of every Gershgörin disk of D^k satisfies the inequality

$$\sum_{j=1, j \neq i}^{N} \left| w_{ji} w_{ki} \right| < \delta\left(w_{ii} - w_{ki} \right), i = 1, \ldots, N; i \neq k \qquad (14)$$

Here $\delta(x)$ is the distance from point x on the real axis to the boundary of the stability zone Ω. As k varies from 1 to N, N sets of stability conditions are obtained which constrain the coupling strengths.

Finite Propagation Speed, Arbitrary Connectivity

Finite propagation speeds in transmission lines cause delays via propagation. These may be negligible over short distances in LANs, but certainly not over larger distances where the delays may become substantially larger than the time scale of the intrinsic dynamics of single neurons or LANs. The introduction of a discrete delay into even a one-dimensional dynamic system makes the dynamics infinite-dimensional. As a consequence, even the simplest network structures, such as local fixed-point elements coupled via symmetric nearest neighbor connections, may display a rich variety of oscillatory behavior. A nice introduction to time-delayed dynamic systems is given by MacDonald (1989). Bélair (1992, 1993) and Van den Driessche and Zou (1998) studied fixed-point elements with arbitrary real couplings which included a single, discrete time delay. They provided general criteria for the stability of the network's equilibrium states. An earlier, but more restricted result on stability has been given by Brauer (1987). Similarly, Chen and Aihara applied related criteria to genetic regulatory networks with time delay (2002) and Earl and Strogatz derived an upper bound for stability of the synchronous state of identical phase oscillators with delayed coupling (2003). In all cases, the challenge is the discussion of a transcendental characteristic equation as we will subsequently show in detail. Generalizations of the stability discussion of such networks may involve distributed delays (Atay, 2003; Zhao, 2003) or multiple delays (Shayer and Campbell, 2000). If the local dynamics of the network nodes are to include oscillatory behavior, analytical results are rare, but some computational studies report delay-induced wave and clustered oscillatory behavior (Campbell and Wang, 1998) as well as amplitude death (Reddy et al., 1999). In the following, we present an extension of these results on delayed networks and briefly discuss the stability of the equilibrium point of (4) under the condition of a local fixed point dynamics and identical, but distributed delays $\delta_{ij} = \tau$. A complete discussion can be found in Jirsa and Ding, 2004.

Consider an N-dimensional state vector $\mathrm{v} = (\ldots v_i \ldots)$ with $v_i \in R; i = 1; \ldots ;N$ with the dynamics

$$\dot{v}_i = -v_i + \sum_{j=1}^{N} a_{ij} v_j (t - \tau) \tag{15}$$

where $a_{ij} \in R$, $\tau \in R_0^+$. First, we will consider discrete delays only. The stability of the functional differential equation (15) is determined by the characteristic polynomial

$$H(z) = z + 1 - \lambda e^{-z\tau} = 0 \tag{16}$$

if all the roots satisfy $Re(z) < 0$ where $u = e^z$, $z \in C$ is an eigenmode of equation 15. The characteristic polynomial defines a critical surface in a three-dimensional parameter space spanned by a, b and τ where a and b are the real and imaginary parts of the eigenvalues of the connectivity matrix a_{ij}. Below the critical surface, all the solutions are stable, that is $Re(z) < 0$; above the critical surface, all the solutions are unstable, that is $Re(z) > 0$. For generally distributed delays, it turns out that the critical surface of the discrete delay case defines a lower bound for the critical surfaces of the distributed delay case (Jirsa and Ding, 2004).

Infinite Propagation Speed, Symmetric, and Translationally Invariant Connectivity

The classic paper on networks with no delay and symmetric and translationally invariant connection topologies is Amari's study of neural fields (1977). Amari discussed spatially and temporally continuous fields with local fixed-point dynamics as intrinsic dynamics. Hence the field dynamics is identical to the dynamics of equation 6 once the transition to the spatial continuum is made: $w_{ij} \to w(x, y)dx$, $v_i(t) \to v(x, t)$ and $\Sigma_j \to \int_\Gamma$ where Γ represents the length of the onedimensional medium with space variable x. The spatial symmetry and translational invariance implies $w(x, y) = w(|\mathrm{x}-\mathrm{y}|)$. Then the field equations may be written as

$$\tau \frac{\partial v(x,t)}{\partial t} = -u(x,t) + \int_\Gamma w(x-y) \\ S\left[v(y,t)\right] dy + h + s(x,t) \tag{17}$$

where S is strongly nonlinear, typically the Heaviside function, and $w(|x-y|)$ is excitatory for proximate connections and inhibitory for greater distances. Such types of connectivity—center-on and surround-off connectivity—have been shown to be omnipresent in the cortex and its subsystems. $s(x,t)$ denotes external input and h a constant resting potential and background activity. In this type of networks, oscillations are not possible, but locally excited regimes may exist and self-sustain with no input $s(x, t) = 0$. If input is provided, then the locally excited regions travel in the direction of increasing field value v until they get pinned at the stimulus location. If several stimuli are provided, then the details of stimulus location and the presence of already excited local regions will determine the typically multi-stable final network dynamics. In all cases, the final stationary network state will be a fixed-point attractor. It was these properties which attracted the attention of neural modelers who applied these fields to a variety of phenomena ranging from rhythm recognition (Large, 2001) to motor movement preparation (Erlhagen and Schöner, 2002). If two or more layers are coupled (Amari, 1977), then a more complex dynamics following equation 7 will be introduced which includes oscillations and traveling wave phenomena.

Infinite Propagation Speed, Symmetric and Translationally Variant Connectivity

Networks that are small enough allow for the delays via propagation to be neglected. Such local network structures typically represent functionally defined areas, or sections thereof, and hence perform local processing and encoding by their synaptic weight distribution. In particular, the latter property requires translational variance of the connection topology within small networks for better flexibility and to provide sufficient storage space. Such networks, with no time delay and symmetric but translationally variant connection topology, have found wide use in the field of pattern recognition. Probably the most famous network is the Hopfield model, which comes in a discrete (Hopfield, 1982) and a time-continuous version (Hopfield, 1984). The time-continuous form of the Hopfield model is identical to the system in equation 6. Equation 15 is sometimes referred to as the delayed Hopfield model. Both the discrete and time-continuous undelayed Hopfield model, obey a fixed-point dynamics, because there is a Liapunov function L for equation 6 if the weights are symmetric, $w_{ij} = w_{ji}$. The existence of a Liapunov function guarantees that the stationary state is unique, hence there is no multi-stability.

A generalization of the dynamics of systems discussed in this subheading is given by the Cohen- Grossberg Theorem (Cohen and Grossberg, 1983) which states the following:

A network $x_i(t) \in R$, $i = 1, \ldots, N$ has the following dynamics

$$\dot{x}_i = a_i(x_i)\left[b_i(x_i) - \sum_{j=1}^{N} c_{ij} d_j(x_j)\right] \tag{18}$$

and the following conditions are valid

$c_{ij} = c_{ij}$ symmetry
$a_i(x_i) \geq 0$ positivity
$d_i'(x_i) \geq 0$ monotonicity

where the prime denotes the partial derivative with respect to x_i. Then there is a Liapunov function $\mathcal{L}$ which reads

$$\begin{aligned} \mathcal{L} = &-\sum_{i=1}^{N} \int_{x_0}^{x_i} b_i(\xi) d_i'(\xi) d\xi \\ &+ \frac{1}{2} \sum_{j,k=1}^{N} c_{jk} d_j(x_j) d_k(x_k) \end{aligned} \tag{19}$$

where x_0 is an arbitrary constant. The Liapunov function has the properties $\partial\mathcal{L}/\partial t \leq 0 \;\; \forall x_i$ and $\mathcal{L}(0) = 0$. Thus the existence of a globally asymptotically stable fixed point is guaranteed.

Finite Propagation Speed, Symmetric, and Translationally Invariant Connectivity

In contrast to the previous subheading, for large-scale area networks often the assumption is made that neighboring neurons act similarly in the average, and hence their average dynamics may be captured by a mean field or neural ensemble dynamics. As a consequence, the connection topology becomes translationally invariant as long as the large-scale corticocortical fiber system is not considered. On the other hand, delays via propagation have to be accounted for large-scale as opposed to local area networks. The model system in (4) captures these structural features nicely, where the transfer function $G_i(t-s)$ is identified with a mean synaptic transfer process. Since the synaptic processes act typically on a short time scale τ compared to the dynamics of $v_i(t)$, most neural field approaches neglect or truncate them at first order of τ Following the lines of (Jirsa and Haken, 1996, 1997), the same transition from (4) to the neural ensemble average as presented in section 4.3.3 leads to the following neural field dynamics

$$\begin{aligned}\hat{K}^{-1}v(x,t) &= \ldots + \tau\dot{v}(x,t) + v(x,t)\\ &= \int_{\Gamma}\omega(|x-y|).S\left[v\left(y,t-\frac{|x-y|}{c}\right)\right]dy,\end{aligned} \tag{20}$$

where c is the propagation velocity along axonal fibers and $\hat{K}^{-1}$ is the inverse of the integral operator. The neural field equation (20) is a nonlinear retarded differential-integral equation with a spatially invariant integral kernel. To gain a first intuition of the neural field's dynamics, the following simplifications may be made: We set $\tau = 0$ and assume the cortical surface ? Γ to be one-dimensional with its connectivity $w(\,|\,x-y\,|\,)$ as

$$w(|x-y|) = \frac{1}{2\sigma}e^{-|x-y|/\sigma} \tag{21}$$

Then the method of Green's functions (Nunez, 1995; Jirsa and Haken, 1996, 1997; Robinson et al., 1997) may be applied which transforms (20) into the Fourier-space of physical space-time, reshuffles the terms in the equation and performs a back-transformation into physical space-time. The resulting equation is a local nonlinear partial differential equation of the form

$$\begin{aligned}&\ddot{v} + \left(\omega_0^2 - c^2\Delta\right)v + 2\omega_0\dot{v}\\ &\quad = \left(\omega_0^2 - \omega_0\frac{\partial}{\partial t}\right)\cdot S[v(x,t)]\end{aligned} \tag{22}$$

where ω_0=c / σ>/4>0 and the one-dimensional Laplacian $\Delta = \frac{\partial^2}{\partial x^2}$. Without any input, the left-hand-side is a damped wave equation and has oscillatory properties. The spatially uniform pattern is generally stable, if the slope of the sigmoid function S[.] on the right-hand-side of (22) is sufficiently small, though transient wave propagation may occur following small discrete perturbations (Robinson et al., 1997). If the slope increases beyond a threshold, then the spatially uniform state is destabilized and wave propagation may occur. Coombes et al. (2003) discuss the effects of connectivity strengths which do not decrease with increasing distance, but rather remain constant within a finite regime. In this case, it is not sufficient to describe the spatiotemporal dynamics by a local partial differential equation as in (22), but non-local delayed terms arise.

To consider rhythms and other oscillatory dynamics within a population, higher order intrinsic dynamics have to be taken into account as characterized by (7). Most neural field approaches (Wilson and Cowan, 1972, 1973; Nunez, 1974; Freeman, 1992; Jirsa and Haken, 1996; Wright and Liley, 1996; Jirsa and Haken, 1997; Robinson et al., 1997) consider two sets of locally coupled populations of inhibitory and excitatory neurons. Paul Nunez (1974) was among the first to derive a neural field dynamics of the kind described by (20). In his early work, his focus was on identifying the dispersion relations of the linearized wave dynamics of (20) given specific distributions of intracortical and corticocortical fiber systems. At about the same time, Hugh Wilson and Jack Cowan derived equivalent descriptions of field activity (1972, 1973), but in terms of firing rates rather than membrane potential. The stationary values of these equations may readily be obtained from the Nunez system by an S-Σ-exchange (compare [3] and see Jirsa and Haken, 1997 for a detailed derivation). The equation for the dynamics of the firing rates read for one population

$$\begin{aligned}u(x,t+T) &= \ldots + T\ddot{u}(x,t) + u(x,t)\\ &= S\left[\int_{\Gamma}\omega(|x-y|)\cdot u\left(y,t-\frac{|x-y|}{c}\right)dy\right],\end{aligned} \tag{23}$$

where a small T has been introduced to capture the effects of averaging processes within the neural ensemble (Wilson and Cowan, 1972). Two coupled populations, one excitatory and one inhibitory, provide the following partial differential-integral equations:

$$\begin{aligned} T\dot{u}_e(x,t) = {} & -u_e(x,t) \\ & +S\Big[\int_\Gamma w_{ee}(|x-y|)\cdot u_e(y,t-\Delta t) \\ & -w_{ei}(|x-y|)\cdot u_i(y,t-\Delta t)dy\Big] \end{aligned} \tag{24}$$

$$\begin{aligned} T\dot{u}_i(x,t) = {} & -u_i(x,t) \\ & +S\Big[\int_\Gamma w_{ie}(|x-y|)\cdot u_e(y,t-\Delta t) \\ & -w_{ii}(|x-y|)\cdot u_i(y,t-\Delta t)dy\Big] \end{aligned} \tag{25}$$

where $\Delta t = |x-y| / c$. The indices e and i distinguish between excitatory and inhibitory population activity. The connectivity matrices wab denote the direction of connections from population b to a, where $a, b = i, e$. As a function of the connectivity functions which were considered to vary only in the range of interactions, Wilson and Cowan discussed the possible wave phenomena which may occur in the system (24, 25). For instance, to guarantee spatial localization of activations, the constraint must be imposed that excitatory to inhibitory interactions are of longer range than excitatory to excitatory interactions. Such is a fundamental mechanism to constrain the spatial dispersion of activations. Other possible dynamic phenomena include steady states, standing and traveling waves.

With the improvement of imaging methods in the 90s, such as high-resolution EEG and the development of MEG and fMRI, several authors used this space and time continuous approach to address a variety of phenomena observed in the large scale dynamics of scalp topographies. Jirsa and colleagues (Jirsa and Haken, 1996, 1997) derived the wave equation (22) and addressed phenomena in motor coordination (Fuchs et al., 1992; Jirsa et al., 1995, 1998; Fuchs et al., 2000). Wright and colleagues introduced much physiological detail and were able to address issues of rhythm generation (Wright and Liley, 1996), as well as clinical aspects such as hysteresis phenomena in anesthesia (Steyn-Ross et al., 1999). Robinson and colleagues provided an alternative derivation of the wave equation (22), introduced expressions for the corticothalamic loop, and implemented detailed physiologically realistic parameter ranges (Robinson et al., 1997, 2001). Frank and colleagues developed a Fokker-Planck approach to the wave equation (22), which captures the time evolution of the stochastic properties of the neural fields (Frank et al., 1999, 2000).

The approaches discussed in this subheading are targeted towards the understanding of large-scale phenomena. All of the model studies mentioned previously make the assumption of a homogeneous, that is symmetric and translationally invariant, connection topology, even though it is well-known from Braitenberg-Schüz's work (1991) that the corticocortical fiber system is strongly heterogeneous. As a consequence, we must conclude that the homogeneous assumption is not appropriate for large-scale networks when considered from the perspective of functionally relevant activity propagation. For very special cases, the homogeneous connectivity assumption may provide a first insight into a large-scale dynamics, e.g., when dealing with global characteristics of activity (such as dispersive properties of the cortex [Nunez, 1995] or global EEG power spectra [Robinson et al., 2001]) or when dealing with highly symmetric situations (activations localized in one area [Jirsa and Haken, 1997; Fuchs et al., 2000] or activations highly symmetric with respect to left-right [Jirsa et al., 1998]).

Finite Propagation Speed, Asymmetric and Translationally Variant Connectivity

The interareal connection topology of the corticocortical fiber system is patchy as shown by Braitenberg and Schüz (1991). Such a connectivity leads naturally to a dynamics with finite time delays $\Delta t = |x-y|/c$ between two areas located at x and y. Following the same derivation as for (20), but with arbitrary connectivity, $w(x;y)\ 6 = w(|x-y|)$, the resulting equation reads

$$\begin{aligned}\hat{K}^{-1}v(x,t) &= \ldots + \tau\dot{v}(x,t) + v(x,t)\\ &= \int_\Gamma \omega(x,y)\cdot S\left[v\left(y,t-\frac{|x-y|}{c}\right)\right]dy \qquad (26)\end{aligned}$$

To extend the local intrinsic dynamics of (26) beyond fixed point dynamics, we generalize the field $v(x,t)$: $R\times R\rightarrow R$ to n-dimensional vector fields $v(x,t)$: $R\times R\rightarrow R^n$ and obtain

$$\begin{aligned}\tau\dot{v}(x,t) &= f(v(x,t)) +\\ &\int_\Gamma \omega(x,y)\cdot S\left[v\left(y,t-\frac{|x-y|}{c}\right)\right]dy \qquad (27)\end{aligned}$$

where f is a n-dimensional vector function (*see* [Jirsa, 2004] for the example of event related potentials). If the corticocortically connected areas are far from each other, then the intracortical homogeneous connectivity may be neglected and we obtain a set of coupled delay-differential equations as discussed in detail in "Finite Propagation Speed, Arbitrary Connectivity" for a fixed-point dynamics. Under this condition, the corresponding variables refer to the mean field dynamics of an area. Mallot and colleagues (Mallot and Brittinger, 1989; Mallot and Giannakopoulos, 1996) discussed in a series of papers, a conceptual framework in which, rather than just mean fields, local networks communicate across distances. These local networks have an intrinsic fixed-point dynamics, but exchange information via time-delayed pathways. Mallot and colleagues applied this approach to examples of the thalamocortical loop (Mallot and Giannakopoulos, 1996) and for the geniculate-striate pathway of the visual system (Mallot and Brittinger, 1989). Freeman's KIII sets, which consist of coupled KII sets with delay, present another example of discretely coupled local networks (Freeman, 1975, 1992).

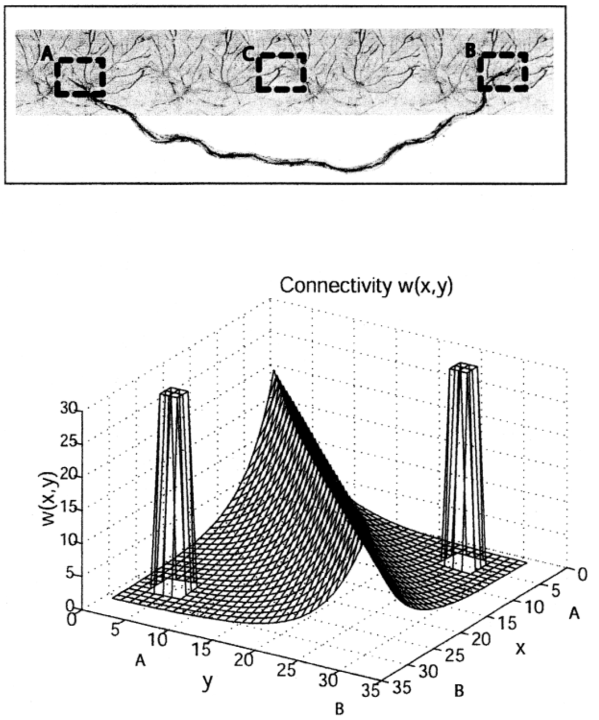

Fig. 6. Two-point connection. The homogeneous connection topology is illustrated within a one-dimensional continuous medium. A bilateral projection from *A* to *B* introduces a heterogeneity into the connectivity. The site C is only homogeneously connected.

The dynamics of local and global networks can be captured by systems (26) for a fixed point dynamics and (27) for a more complex intrinsic dynamics. In the following we wish to discuss one fundamental example that nicely illustrates the interplay between connectivity and spatiotemporal dynamics. Figure 6 shows a continuous neural sheet with a homogeneous intracortical connectivity and two select sites, *A* and *B*, which are connected via an additional direct pathway. The resulting connectivity function reads

$$\begin{aligned}w(x,y) = w(|x-y|)&\\ +w_{ab}\delta(x-a)\delta(y-b)&\\ +f_{ab}\delta(x-b)\delta(y-a)&\end{aligned} \qquad (28)$$

where $w(|x-y|)$ is the homogeneous part if the connectivity and w_{ab}, w_{ba} are the constant

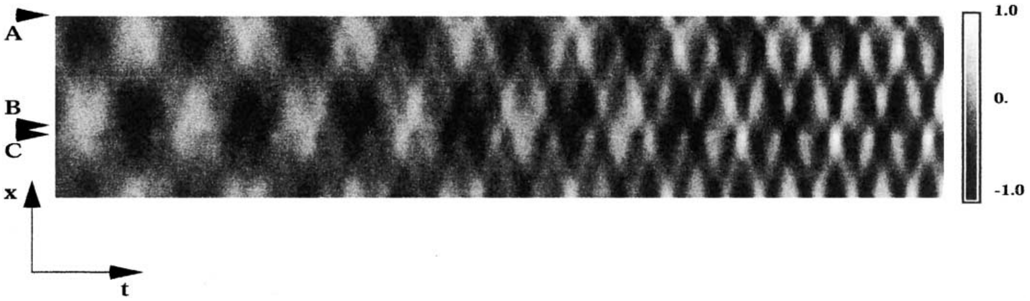

Fig. 7. Spatiotemporal phase transition. Space x is vertical and time t is horizontal. The originally stable spatiotemporal pattern is destabilized by making a small change in one terminal of the heterogeneous projection from $x = B$ to $x = C$. The transient spatiotemporal dynamics (evolving after the change of the distance d occurred at time $t = 0$) is shown.

connection strengths of the heterogeneous pathway. The connection matrix and its topology is illustrated in Fig. 6. The locations of A, B are given by $x = a, b$. If $w_{ab} = w_{ba}$, then the connectivity is symmetric. First, we consider how local changes in connectivity guide the system through a series of bifurcations, second, we show how heterogeneous components in the connectivity affect the variability of spontaneous network activity.

Fig. 6

1. Spatiotemporal bifurcations:

Jirsa and Kelso (2000) studied the dynamics defined by (26) as a function of the distance $d = |a-b|$ between the sites A and B, and derived a characteristic transcendental equation, similar to (16), for the linear stability of each spatial network pattern. In particular, stability criteria may be formulated as a function of connectivity. As the control parameter d is scaled up continuously, the system is guided through a series of phase transitions. For illustration, an example of a phase transition is shown in Fig. 7. The change in the connection topology destabilizes the initial stationary dynamics and the system undergoes a transition to a new stationary state via a Hopf bifurcation. Detailed bifurcation diagrams are given in (Jirsa and Kelso, 2000).

Fig. 7

2. Reduced variability of spontaneous network dynamics:

Even if no destabilization of the global dynamics occurs, the signature of a heterogeneous connection topology is present through changes of the variability in spike timings. Jirsa (2002) studied a network with a local FitzHugh-Nagumo dynamics (FitzHugh, 1961; Nagumo et al., 1962) and asked the following question: Given three sites A, B, and C in a neural sheet with a dynamics described by (27), how does the inclusion of an additional two-point connection from A to B render the variability of the occurrence of spike timings for a given noise level? Here, spike timing refers to the time difference between two spikes occurring at two sites. A reduced timing variability between A and B means that if a spike occurs at a site A, then there is an increased probability for a spike to occur at site B. An illustration of the spontaneous spatiotemporal activity is given in Fig. 8. A clustering of spikes occurs at the heterogeneously connected sites over time. Spikes occur more frequently with a fixed time lag between A and B, rather than between A and C. Close to bifurcation thresholds at which the system performs phase transitions, it turns out that there is a parameter space for which the introduction of a unilateral pathway, from one site to the other, does not significantly alter the timing precision of events at the connected sites. However, the bilateral connection causes a significant reduction of the timing variability.

Fig. 8

Concluding Remarks

In this review, we systematically discussed the interplay between connectivity and dynamics, or equivalently between structure and function.

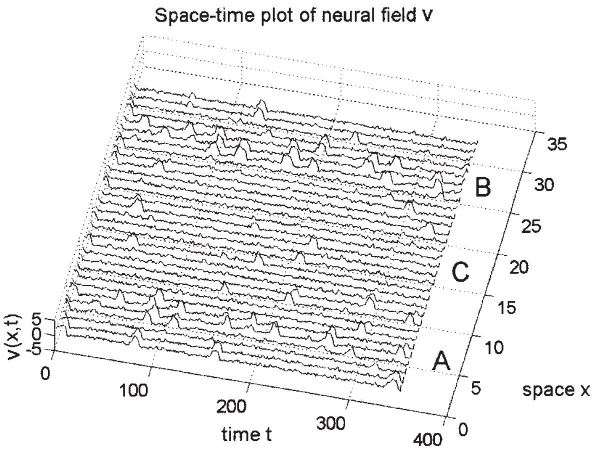

Fig. 8. Spontaneous spatiotemporal field activity with a bilateral pathway between *A* and *B*. The variability in the timing of the spikes occurring at *A* and *B* is reduced compared to the spike timings occurring at *A* and *C*.

Our main focus has been the discussion of how changes in connectivity affect the spatiotemporal network dynamics qualitatively. The three major criteria of comparison have been the local dynamics at the network nodes, the presence of time delays and properties of the connectivity matrix. For each type of these network models, we sketched the existing current knowledge, or more importantly, the limits thereof.

Why is this classification important? Neural information processing is not localized in one small area of the brain, neither can it be described by one class of network model. In fact, as we tried to convince the reader, there is an anatomical hierarchy of connection topologies which require different network models on different levels of organization. For this reason, the study of neural information processing leads us naturally to the study of networks of networks, rather than to the study of a single network.

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