

The Cause of Complexity in Nature: An Analytical and Computational Approach

Klaus Mainzer

Abstract This work is going to present the cause of complexity in nature from an analytical and computational point of view. The cause of complex pattern formation is explained by the local activity of cells in complex systems which are analytically modeled by nonlinear reaction-diffusion equations in physics, chemistry, biology, and brain research. There are not only rigorous analytical criteria of local activity and the edge of chaos, but also constructive procedures to visualize them by computer simulations. In technology, the question arises whether these criteria and procedures can be used to construct artificial life and artificial minds.

Keywords Complexity • Computation • Nonlinearity • reaction-diffusion equations • Nature • Edge of chaos

1 Introduction

According to several prominent authors, a main part of twenty-first century science will be on complexity research. The intuitive idea is that global patterns and structures emerge from locally interacting elements like atoms in laser beams, molecules in chemical reactions, proteins in cells, cells in organs, neurons in brains, agents in markets etc. by self-organization [1]. But what is the cause of self-organization? Complexity phenomena have been reported from many disciplines (e.g., biology, chemistry, ecology, physics, sociology, economy etc.) and analyzed from various perspectives such as Schrödinger's order from disorder [2], Prigogine's dissipative structure [3], Haken's synergetics [4], Langton's edge of

K. Mainzer (✉)

Chair of Complexity Research/Philosophy of Science, Munich Center for Technology in Society, Technische Universität München, Munich, Germany
e-mail: mainzer@tum.de

chaos [5] etc. Steven Wolfram declared computer experiments with pattern formation of cellular automata as “new kind of science” [6]. But concepts of complexity are often based on experiments, examples or metaphors only. We argue for a mathematically precise and rigorous definition and analytical theory of local activity as the cause of self-organizing complexity which can be tested in an explicit and constructive manner [7, 8].

Boltzmann’s struggle in understanding the physical principles distinguishing between living and non-living matter, Schrödinger’s negative entropy in metabolisms, Turing’s basis of morphogenesis [9], Prigogine’s intuition of the instability of the inhomogeneous, and Haken’s synergetics are in fact all direct manifestations of a fundamental principle of locality. It can be considered the complement of the second law of thermodynamics explaining the emergence of order from disorder instead of disorder from order, in a quantitative way, at least for reaction diffusion systems.

The principle of local activity is precisely the missing concept to explain the emergence of complex patterns in a homogeneous medium. Cellular automata are an illustrative model of local active cells with pattern formation. They can be characterized as complex dynamical systems in the strictly mathematical sense with corresponding equations and proofs. In short, there are analytical models of cellular automata, in order to find precise answers and predictions of pattern formation [10].

The local principle can be generalized and proven for the class of nonlinear reaction-diffusion systems in physics, chemistry, biology, and brain research. The principle of local activity is the cause of symmetry breaking in homogeneous media. The Brusselator model was one of the first systems of equations used to explain self-organizing chemical reactions of the reaction-diffusion type. Based on this model, a theory of dissipative structures operating far from thermodynamic equilibrium was developed [11]. Under stability theory techniques, Prigogine and his group derived a critical bifurcation boundary for the uncoupled cell. They studied stationary and dynamic patterns emerging in the neighborhood of this boundary. But, except for the stability boundaries, the far from thermodynamic-equilibrium theory is too coarse to predict sharper and more precise domain of emergent behavior. Especially, they ignored the relatively small subset of the edge of chaos where the emergence of complexity is most likely.

The local activity theory is applied, again, to the Gierer-Meinhardt-equations to illustrate the emergence of complexity. On the basis of autocatalysis and lateral inhibition, Gierer and Meinhardt proposed a mathematical model [12] to explain pattern formation (morphogenesis) in living systems. Using numerical integration, they were able to produce a number of patterns relevant to the formation of biological structures. An analytical treatment of the Gierer-Meinhardt model from the synergetics perspective was presented in Ref. [13] using the order parameter concept combined with linear stability analysis. While the later approach offers significant contributions in understanding the dynamics of the Gierer-Meinhardt model, it is still too coarse to predict the precise domain in the cell parameter space where emergent behavior may occur. The local activity theory offers a rigorous

and effective tool for sharpening existing results in the sense that it can identify more precisely those regions in the cell parameter space which are capable of emergent behaviors, and also in fine tuning such regions into a relatively small subset called the edge of chaos where the emergence of complex phenomena is most likely [14].

The long-lasting action and pace-maker potentials of the Purkinje fiber of the heart were first described by the Hodgkin-Huxley equations of the cardiac Purkinje fiber model of morphogenesis in [11]. The bifurcation diagrams of the corresponding computer simulations supply a possible explanation for why a heart with a normal heart-rate may stop beating suddenly: The cell parameter of a normal heart is located in a locally active unstable domain and just nearby an edge of chaos. The membrane potential along a fiber is simulated in a Hodgkin-Huxley model by a computer [15]. Computer simulations show that oscillatory patterns, chaotic patterns, or divergent patterns may emerge if the selected cell parameters are located in locally active domains but nearby the edge of chaos. This research demonstrates once again the effectiveness of the local activity theory in choosing the parameters for the emergence of complex (static and dynamic) patterns in a homogeneous lattice formed by coupled locally active cells.

One of the marvels of the HH (Hodgkin-Huxley) equations is its ability to generate an action potential (spikes) in response to an external current excitation emulating the net synaptic current excitation. Although the spikes were consistently generated numerically, no one knew the physical and mathematical origin of the action potential (spikes). Local activity can be demonstrated to be the origin of spikes. In particular, neurons can be shown to be poised near a tiny subset of the local activity domain, which we call the edge of chaos. The domain of the edge of chaos is determined by deriving an explicit scalar complexity function of a small-signal Hodgkin-Huxley circuit model [16].

A completely new application of local activity is the so-called edge of chaos where most complex phenomena emerge. In this particular case, a hidden excitability allows a unit to be destabilized when interacting with dissipative environments. Although a diffusion process has a tendency to equalize differences, an originally dead or inactive cell becomes alive or active upon coupling with other cells by diffusion [17]. This phenomenon seems to be counterintuitive, but can mathematically rigorously be proven and confirmed with different applications in reality.

In the parameter spaces of reaction diffusion systems, the domains of local activity can be visualized in computer simulations. The edges of chaos are very small regions with the ability of creation. They seem to be hidden in the domains of activity like pearls in shells on the sea ground. Actually, in research, the “edge of chaos” was only used as metaphor, but not as mathematically precise concept. Its discovery in the parameter spaces of dynamical systems is, to our best knowledge, completely new in complexity research. The local activity principle and its pearl, the edge of chaos, are couched in rigorous mathematics. Above all, they are characterized by constructive procedures to compute and visualize their complexity. Therefore, researchers from other disciplines that can describe their

dynamical systems via differential equations, such as reaction-diffusion equations, can actually calculate easily with a computer the parameter values where complexity and creativity can occur.

We argue that the principle of local activity and edge of chaos are really fundamental in science. Twenty-first-century engineering sciences are more and more inspired by the life sciences. In systems biology, cells, organs, and organisms are considered complex systems which can be modeled by complex networks with great similarity to electronic circuits. In the tradition of engineering sciences, synthetic biology uses schemes of systems biology to construct new artificial organisms for practical applications (e.g., new bacteria for cleaning polluted water). Robots become more and more autonomous and self-organizing systems. The question arises whether the principle of local activity can be applied in these technical systems.

2 Mathematical Definition of Local Activity and Edge of Chaos

The principle of local activity had originated from electronic circuits, but can easily be translated into other non-electrical homogeneous media [8]. The transistor is an example of a locally-active device, whereby a “small” (low-power) input signal can be converted into a “large” (high power) output signal at the expense of an energy supply (namely a battery). No radios, televisions, and computers can function without using locally-active devices such as transistors. For the formation of complex biological and chemical patterns, Schrödinger and Prigogine demanded nonlinear dynamics and an energy source as necessary conditions. But, for the exhibition of patterns in an electronic circuit (i.e., non-uniform voltage distributions), the demand for nonlinearity and energy source is too crude. In fact, no patterns can emerge from circuits with cells made of only batteries and nonlinear circuit elements which are not locally active.

In general, a spatially continuous or discrete medium made of identical cells interacting with all cells located within a neighborhood is said to manifest complexity if the homogeneous medium can exhibit a non-homogeneous static or spatio-temporal pattern under homogeneous initial and boundary conditions. The principle of local activity can be formulated mathematically in an axiomatic way without mentioning any circuit models. Moreover, any proposed unified theory on complexity should not be based on observations from a particular collection of examples and explained in terms that make sense only for a particular discipline, say chemistry. Rather it must be couched in discipline-free concepts, which means mathematics, being the only universal scientific language.

However, in order to keep physical intuition and motivation behind this concept, we start with a special class of spatially-extended dynamical systems, namely the reaction-diffusion equations which are familiar in physics and chemistry.

Our first definition of local activity refers to a discretized spatial model which can easily be illustrated by cellular nonlinear networks. All results apply to the associated systems of continuous reaction-diffusion partial differential equations which will be later analyzed e.g., in fluid dynamics of physics and chemistry. Let us consider a spatial lattice of identical cells located at grid points and changing their states by local reaction-diffusion. In general, the change of a local cellular state depends on all the other cellular states in the spatial lattice and (at least in some cases) on the local diffusion of the cell.

In mathematical terms, the dynamics of the whole system is defined by a system of discrete reaction-diffusion equations describing the changes of the local cellular states in the spatial lattice [7, 18] or corresponding continuous reaction-diffusion partial differential equations. In the discretized as well as continuous case, the state variables pertain to reaction cells lumped at lattice points $r \triangleq (j, k, l)$. This dynamical system implements kinetic equations in chemistry and circuit models in network theories. The equations can be represented in the following compact vector form with

$$\mathbf{V}_a \triangleq [V_1, V_2, \dots, V_m]^T \text{ and } \mathbf{V}_b \triangleq [V_{m+1}, V_{m+2}, \dots, V_n]^T:$$

$$\dot{\mathbf{V}}_a = \mathbf{f}_a(\mathbf{V}_a, \mathbf{V}_b) + \mathbf{D}\nabla^2 \mathbf{V}_a \quad (1)$$

$$\dot{\mathbf{V}}_b = \mathbf{f}_b(\mathbf{V}_a, \mathbf{V}_b) \quad (2)$$

where

$\mathbf{f}_a(\mathbf{V}_a, \mathbf{V}_b) \in \mathbb{R}^m$ denotes the first m components $f_1(\mathbf{V}_a, \mathbf{V}_b)$, $f_2(\mathbf{V}_a, \mathbf{V}_b)$, \dots , $f_m(\mathbf{V}_a, \mathbf{V}_b)$ of the kinetic term,
 $\mathbf{f}_b(\mathbf{V}_a, \mathbf{V}_b) \in \mathbb{R}^m$ denotes the remaining $(n-m)$ components $f_{m+1}(\mathbf{V}_a, \mathbf{V}_b)$, $f_{m+2}(\mathbf{V}_a, \mathbf{V}_b)$, \dots , $f_n(\mathbf{V}_a, \mathbf{V}_b)$,

\mathbf{D} denotes an $m \times m$ diagonal matrix defined by $\mathbf{D}_{\sigma\sigma} = D_\sigma$, and

$\nabla^2 \mathbf{V}_a \in \mathbb{R}^m$ denotes a $m \times 1$ vector defined by the m discrete Laplacian Operators $\nabla^2 V_\sigma$, $\sigma = 1, 2, \dots, m$.

The state variables \mathbf{V}_a and \mathbf{V}_b pertain to only one isolated cell located at the lattice coordinate $\mathbf{r} = (j, k, l)$ or to a point $\mathbf{r} \in \mathbb{R}^3$ in the continuous case. Any dynamical system has some tunable control parameters $\boldsymbol{\mu} = [\mu_1 \mu_2 \dots \mu_\rho]^T$ associated with changing conditions of the system. Hence, the kinetic term in the reaction-diffusion equation for each cell at location $\mathbf{r} = (j, k, l)$ is described by the following cell kinetic equations:

$$\dot{\mathbf{V}}_a = \mathbf{f}_a(\mathbf{V}_a(\mathbf{r}), \mathbf{V}_b(\mathbf{r}); \boldsymbol{\mu}) \quad (3)$$

$$\dot{\mathbf{V}}_b = \mathbf{f}_b(\mathbf{V}_a(\mathbf{r}), \mathbf{V}_b(\mathbf{r}); \boldsymbol{\mu}) \quad (4)$$

For an $N \times N \times N$ cubic lattice, there are N^3 identical cells, each one described these cell kinetic equations. Since only the first m state variables $\mathbf{V}_a(\mathbf{r}) = [V_1(\mathbf{r}), V_2(\mathbf{r}), \dots, V_m(\mathbf{r})]^T$ of each cell can interact with the neighboring cells via the

diffusion term $D\nabla^2 \mathbf{V}_a(\mathbf{r})$, only the energy and matter associated with the first m state variables can flow into a neighbor cell. Henceforth, the state variables $V_1(\mathbf{r}), V_2(\mathbf{r}), \dots, V_m(\mathbf{r})$ in $\mathbf{V}_a(\mathbf{r})$ are called port variables in analogy with the transfer of goods between islands. The remaining state variables $V_{m+1}(\mathbf{r}), V_{m+2}(\mathbf{r}), \dots, V_n(\mathbf{r})$ in $\mathbf{V}_b(\mathbf{r})$ are called non-port variables. The concept of local activity will be defined in terms of only port variables.

Since the diffusion term can play only a dissipative and hence stabilizing role with $D_i > 0$ in the reaction-diffusion equations, the origin of any complex phenomenon exhibited by these equations can only come from the cell kinetic equations. It can rigorously be proved that if the cell kinetic equations are not locally active for a fixed control parameter $\boldsymbol{\mu} = \boldsymbol{\mu}_0$, the reaction-diffusion equations cannot exhibit any complexity regardless of the choices of the diffusion coefficients $D_i > 0$. Moreover, explicit mathematical criteria can be given for testing any cell kinetic equation for local activity. With these criteria, one can identify the active parameter domain \mathcal{A} of the parameter space $\boldsymbol{\mu} \in \mathbb{R}^\rho$ where a cell kinetic equation is locally active. Since the complement $\mathcal{P} = \mathbb{R}^\rho \setminus \mathcal{A}$ can only lead to homogeneous solution of the reaction-diffusion equations, it is called the passive parameter domain.

Since complexity can occur only for parameters located in the active parameter region \mathcal{A} , it follows that local activity is indeed the origin of complexity. A locally-active cell kinetic equation can exhibit complex dynamics such as limit cycles or chaos, even if the cells are uncoupled from each other by setting all diffusion coefficients to zero. It is not surprising that coupling such cells could give rise to complex spatio-temporal phenomena. What is surprising and counter-intuitive is that there exists a proper subset ε of the active parameter domain \mathcal{A} , called the edge of chaos where the uncoupled cell kinetic equation is asymptotically stable.

To illustrate this state biologically, a cell is inert or dead in the sense that the concentrations of its enzymes achieved a constant equilibrium. In interaction, however, the cellular system pulses or become alive in the sense that the concentrations of the enzymes in each cell will oscillate indefinitely. By coupling these dead cells via a dissipative diffusion environment, it may be possible for the reaction-diffusion equation to exhibit non-homogeneous patterns and other spatio-temporal phenomena for appropriate diffusion coefficients. The criteria for the edge of chaos correspond mathematically to Prigogine's instability of the homogeneous and Turing's properties of instability as origin of morphogenesis.

Since local activity is defined only with respect to m port variables in $\mathbf{V}_a = [V_1, V_2, \dots, V_m]^T$, and since it does not involve the diffusion coefficients D_1, D_2, \dots, D_m in the reaction-diffusion equation, an interaction term $\mathbf{I}_a \triangleq D\nabla^2 \mathbf{V}_a$ can be defined as port input vector at the diffusion-driven ports. By substituting this input term into the compact vector form of the reaction-diffusion equations, we get the cell kinetic equations

$$\dot{\mathbf{V}}_a(\mathbf{r}) = \mathbf{f}_a(\mathbf{V}_a(\mathbf{r}), \mathbf{V}_b(\mathbf{r}); \boldsymbol{\mu}) + \mathbf{I}_a \quad (5)$$

$$\dot{\mathbf{V}}_b(\mathbf{r}) = \mathbf{f}_b(\mathbf{V}_a(\mathbf{r}), \mathbf{V}_b(\mathbf{r}); \boldsymbol{\mu}) \quad (6)$$

with control parameter $\boldsymbol{\mu} \in \mathbb{R}^\rho$. They are called forced cell kinetic equations, because $\mathbf{I}_a \in \mathbb{R}^m$ can be physically interpreted as an external force applied at the m diffusion ports. Since all the cells are identical, this equation represents the state dynamics of an isolated cell driven at its diffusion ports by an external force $\mathbf{I}_a \in \mathbb{R}^m$ representing the external world.

The equilibrium states of an isolated cell can be obtained by setting the state change to zero with $\dot{\mathbf{V}}_a = 0$ and $\dot{\mathbf{V}}_b = 0$, namely

$$0 = \mathbf{f}_a(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu}) + \mathbf{I}_a \quad (7)$$

$$0 = \mathbf{f}_b(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu}), \quad (8)$$

and solving these equations for $\mathbf{V}_a \in \mathbb{R}^m$ and $\mathbf{V}_b \in \mathbb{R}^{n-m}$ for each fixed parameter $\boldsymbol{\mu} \in \mathbb{R}^\rho$. In general, there are multiple equilibrium points for each input $\mathbf{I}_a \in \mathbb{R}^m$.

Let $\bar{\mathbf{V}}_a$ and $\bar{\mathbf{V}}_b$ denote the coordinates of any cell equilibrium point \mathcal{Q} , where \mathcal{Q} depends on the constant input $\bar{\mathbf{I}}_a \in \mathbb{R}^m$ and control parameter $\boldsymbol{\mu} \in \mathbb{R}^\rho$. In the case of an infinitesimal change $\mathbf{i}_a(t)$ of the constant input \mathbf{I}_a , we consider infinitesimal deviations $\mathbf{v}_a(t)$ and $\mathbf{v}_b(t)$ in the neighborhood of the equilibrium point \mathcal{Q} with coordinates $\bar{\mathbf{V}}_a$ and $\bar{\mathbf{V}}_b$, namely

$$\mathbf{V}_a(t) \triangleq \bar{\mathbf{V}}_a + \mathbf{v}_a(t) \quad (9)$$

$$\mathbf{V}_b(t) \triangleq \bar{\mathbf{V}}_b + \mathbf{v}_b(t) \quad (10)$$

$$\mathbf{I}_a(t) \triangleq \bar{\mathbf{I}}_a + \mathbf{i}_a(t). \quad (11)$$

Linearized Cell State Equations. In order to approximate the forced cell kinetic dynamics at the cell equilibrium point with constant input $\mathbf{I}_a = \bar{\mathbf{I}}_a$, we use the Taylor series expansion of $\mathbf{f}_a(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu})$ and $\mathbf{f}_b(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu})$ about the cell equilibrium point $\mathcal{Q}(\mathbf{V}_a = \bar{\mathbf{V}}_a, \mathbf{V}_b = \bar{\mathbf{V}}_b)$. In general, a Taylor series is a representation of a function as an infinite sum of terms that are calculated from the values of the function's derivatives at a single point. It is usual to approximate a function by using a finite number of terms of its Taylor series. Taylor's theorem gives quantitative estimates on the error in this approximation. Any finite number of initial terms of the Taylor series of a function is called a Taylor polynomial. The Taylor series of a function is the limit of that function's Taylor polynomials, provided that the limit exists. If we delete the higher-order terms of the Taylor series, we obtain linearized cell state equations. They can be interpreted as the cell dynamics along a tangent plane at the cell equilibrium point \mathcal{Q} which depends on the input \mathbf{I}_a and the control parameter value $\boldsymbol{\mu}$, namely

$$\frac{d\mathbf{v}_a(t)}{dt} = \mathbf{A}_{11}(\mathcal{Q})\mathbf{v}_a(t) + \mathbf{A}_{12}(\mathcal{Q})\mathbf{v}_b(t) + \mathbf{i}_a(t) \quad (12)$$

$$\frac{d\mathbf{v}_b(t)}{dt} = \mathbf{A}_{21}(\mathcal{Q})\mathbf{v}_a(t) + \mathbf{A}_{22}(\mathcal{Q})\mathbf{v}_b(t) \quad (13)$$

where

$$\mathbf{A}_{11}(\mathcal{Q}) \triangleq \frac{\partial \mathbf{f}_a(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu})}{\partial \mathbf{V}_a} \big|_{\mathbf{V}_a = \bar{\mathbf{V}}_a, \mathbf{V}_b = \bar{\mathbf{V}}_b}, \quad (14)$$

$$\mathbf{A}_{12}(\mathcal{Q}) \triangleq \frac{\partial \mathbf{f}_a(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu})}{\partial \mathbf{V}_b} \big|_{\mathbf{V}_a = \bar{\mathbf{V}}_a, \mathbf{V}_b = \bar{\mathbf{V}}_b} \quad (15)$$

$$\mathbf{A}_{21}(\mathcal{Q}) \triangleq \frac{\partial \mathbf{f}_b(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu})}{\partial \mathbf{V}_a} \big|_{\mathbf{V}_a = \bar{\mathbf{V}}_a, \mathbf{V}_b = \bar{\mathbf{V}}_b}, \quad (16)$$

$$\mathbf{A}_{22}(\mathcal{Q}) \triangleq \frac{\partial \mathbf{f}_b(\mathbf{V}_a, \mathbf{V}_b; \boldsymbol{\mu})}{\partial \mathbf{V}_b} \big|_{\mathbf{V}_a = \bar{\mathbf{V}}_a, \mathbf{V}_b = \bar{\mathbf{V}}_b}. \quad (17)$$

In the linearized cell state equation, $\mathbf{A}_{11}(\mathcal{Q})$ is an $m \times m$ matrix, $\mathbf{A}_{12}(\mathcal{Q})$ is an $m \times (n-m)$ matrix, $\mathbf{A}_{21}(\mathcal{Q})$ is an $(n-m) \times m$ matrix, and $\mathbf{A}_{22}(\mathcal{Q})$ is an $(n-m) \times (n-m)$ matrix. They are constant real matrices whose elements depend on the constant input $\mathbf{I}_a \in \mathbb{R}^m$, the control parameter $\boldsymbol{\mu} \in \mathbb{R}^\rho$, and the cell equilibrium point $\mathcal{Q}(\mathbf{I}_a, \boldsymbol{\mu})$.

We are now able to define local activity at a cell equilibrium point \mathcal{Q} . Given any continuous input function of time $\mathbf{i}_a(t)$ for $t \geq 0$ and assuming zero initial conditions $\mathbf{v}_a(0) = 0, \mathbf{v}_b(0) = 0$, a solution of the linearized cell state equations about cell equilibrium point \mathcal{Q} is an infinitesimal cell state in the neighborhood of the cell equilibrium point \mathcal{Q} , denoted by $\mathbf{v}_a(t)$ and $\mathbf{v}_b(t)$ for $t \geq 0$. Let us define the local power flow $p(t) \triangleq \mathbf{v}_a(t) \cdot \mathbf{i}_a(t)$ as rate of change of energy at time t at cell equilibrium point $\mathcal{Q}(\mathbf{V}_a = \bar{\mathbf{V}}_a, \mathbf{V}_b = \bar{\mathbf{V}}_b)$. Mathematically, the term $p(t)$ denotes the scalar (dot) product between the two vectors $\mathbf{v}_a(t)$ and $\mathbf{i}_a(t)$.

The principle of local activity is based on the idea that when operating in an infinitesimal neighborhood of a cell equilibrium point \mathcal{Q} , a locally-active cell must behave like a unit (e.g., a transistor in technology) operating at an active operating point whereby a small (low-power) input can be converted into a large (high-power) output at the expense of an energy supply (e.g., a battery in the case of a transistor). In general, a cell is said to be locally active at an equilibrium point \mathcal{Q} if it is possible to find a local (i.e., infinitesimal) input $\mathbf{i}_a(t)$ such that by applying the global input $\mathbf{I}_a(t) \triangleq \bar{\mathbf{I}}_a + \mathbf{i}_a(t)$, we can extract more infinitesimal energy at \mathcal{Q} over some time interval $0 < T < \infty$ than what the cell has taken from its external environment which consists of the coupling spatial grid of all the other cells.

Let $w(t)$ be the total energy (i.e., the “infinitesimal sum” or integral of power flow $p(t) \triangleq \mathbf{v}_a(t) \cdot \mathbf{i}_a(t)$) accumulated since the initial time $t = 0$ until $t = T$. It is convenient though arbitrary to distinguish the reference direction of the total energy entering and leaving the cell at $t = T$. If $w(t) > 0$, then there is a net total energy accumulated since the initial time $t = 0$ entering the cell at

$t = T$. Conversely, if $w(t) < 0$, then at $t = T$, the cell is actually delivering energy to the external circuit. In this case, at $t = T$, the cell behaves like a local source of energy, rather than a sink.

Definition of Local Activity. A cell is said to be locally active at a cell equilibrium point \mathcal{Q} if and only if there exists a continuous input time function $\mathbf{i}_a(t) \in \mathbb{R}^m, t \geq 0$, such that at some finite time $T, 0 < T < \infty$, there is a net energy flowing out of the cell at $t = T$, assuming the cell has zero energy at $t = 0$, namely

$$w(t) = \int_0^T \mathbf{v}_a(t) \cdot \mathbf{i}_a(t) dt < 0, \quad (18)$$

where $\mathbf{v}_a(t)$ is a solution of the linearized cell state equation about \mathcal{Q} with zero initial state $\mathbf{v}_a(0) = \mathbf{0}$ and $\mathbf{v}_b(0) = \mathbf{0}$.

Definition of Local Passivity. A cell is said to be locally passive at a cell equilibrium point \mathcal{Q} if and only if it is not locally active at \mathcal{Q} , namely

$$w(t) = \int_0^T \mathbf{v}_a(t) \cdot \mathbf{i}_a(t) dt > 0, \quad (19)$$

for all continuous input time functions $\mathbf{i}_a(t)$ and for all $t \geq 0$, under zero initial states $\mathbf{v}_a(0) = \mathbf{0}$ and $\mathbf{v}_b(0) = \mathbf{0}$.

Definition of Locally Active Reaction-Diffusion Equations. Reaction-diffusion equations are called locally active if and only if its associated cells are locally active at some cell equilibrium point. Otherwise, they are said to be locally passive.

Understanding the Local Activity Principle. In the definition of local activity, we need the assumption of zero energy at $t = 0$, because otherwise the cell may have some stored energy $t = 0$ and it could be discharging it to the outside circuit even though it is locally passive. The cell's ability to act as a source of small-signal energy implies that it can amplify an initially small input signal into a larger-energy signal. The increase in energy must, of course, come from some external energy supply, such as a kind of external pump or battery if the cell is a transistor, or "glucose" if the cell is a neuron. According to the conservation principle of energy, there is nothing coming from nothing, or, in economic terms, there is "no free lunch".

Mathematically, the signal must be infinitesimal small in order that we can model the cell by only the linear terms in its Taylor series expansion. This in turn allows us to apply well-known linear mathematics and derive explicit analytical criteria for the cell to be locally active at the equilibrium point where the Taylor series expansion is computed. This also proves that complexity originates from infinitesimal small perturbations, notwithstanding the fact that the complete system is typically highly nonlinear.

Intuitively, a cell is locally-active if it is endowed with some excitable "innate" potential, such that under certain conditions, it can become "mathematically alive", capable of exhibiting oscillation and chaos. The deepest and counter-intuitive

property of local activity is that a “mathematically dead” but locally active cell can become explosive, even if it is interfaced with a locally-passive load, or “sink”. That can never happen with a locally-passive cell, whose entropy must increase continuously.

Complexity Function and Complexity Matrix. In order to prove that a cell is locally-active at an equilibrium point \mathcal{Q} , the definition of local activity requires that an input time function $\mathbf{i}_a(t)$ must be found which initiates a positive energy flow $\int_0^T \mathbf{v}_a(t) \cdot \mathbf{i}_a(t) dt$ out of the cell at some finite time $0 < T < \infty$, assuming $\mathbf{v}_a(0) = \mathbf{0}$ and $\mathbf{v}_b(0) = \mathbf{0}$. The definition is intuitively clear and mathematically precise, but misses a constructive procedure whether such an input time function exists or not. Therefore, computationally practical necessary and sufficient conditions must be found to test the local activity of some cell at an equilibrium point. In natural and engineering sciences, Laplace transforms are used for analysis of linear time-invariant systems (e.g., electrical circuits, harmonic oscillators, optical devices, and mechanical systems). In this analysis, the Laplace transform is sometimes interpreted as a transformation from the time-domain, in which inputs and outputs are functions of time, to the frequency-domain, where the same inputs and outputs are functions of complex angular frequency (in radians per unit time). In any way, given a simple mathematical or functional description of an input or output to a system, the Laplace transform provides an alternative functional description that often simplifies the process of analyzing the behavior of the system, because it converts a system of linear differential equations to a system of linear algebraic equations. Therefore, we consider the Laplace transforms of each component of the vectors $\mathbf{v}_a(t)$, $\mathbf{v}_b(t)$, and $\mathbf{i}_a(t)$.

By this procedure, a complexity function $Y_{\mathcal{Q}}(s)$ can be derived for complex number $s = a + ib$ with $\text{Re}[s] = a$ and $\text{Im}[s] = b$, in short, $s = \text{Re}[s] + i \text{Im}[s]$. The complexity function can be illustrated as mapping on a complex plane. It is said to be a positive-real function iff (1) $Y_{\mathcal{Q}}(s)$ is a real number whenever s is a real number, (2) $\text{Re}[Y_{\mathcal{Q}}(s)] \geq 0$ for all s with $\text{Re}[s] \geq 0$ where $Y_{\mathcal{Q}}(s)$ is not singular. Since $Y_{\mathcal{Q}}(s)$ is assumed to be a rational function, condition (1) is always satisfied. In this case, $Y_{\mathcal{Q}}(s)$ is a positive-real function iff the closed right-half s -plane is mapped into the closed right-half $Y_{\mathcal{Q}}$ -plane.

In the simplest case with only one diffusion coefficient ($m = 1$), one port state variable $\hat{\mathbf{v}}_a(s)$ and one non port variable $\hat{\mathbf{v}}_b(s)$ ($n = 2$), the complexity function $Y_{\mathcal{Q}}(s)$ reduces to a scalar rational function. For $m = 1$ and $n = 2$, we get a rational function of complex variable s . For $m > 1$, we get a $m \times m$ complexity matrix $Y_{\mathcal{Q}}(s)$ whose elements are rational functions of complex variable s .

In the scalar case (one port state variable), the Local Passivity Principle can be proved ([7], 3442):

The Local Passivity Theorem. A uncoupled cell with one port state variable $m = 1$ is locally passive at a cell equilibrium point $\mathcal{Q}(V_1 = \bar{V}_1)$ if, and only if, the complexity function $Y_{\mathcal{Q}}(s)$ is a positive real function.

It follows from the Local Passivity Theorem that a cell is locally passive if, and only if, the closed right-half s -plane maps into the closed right-half Y_Q -plane. These insights deliver a practical test for local passivity ([7], 3443).

Test for Local Passivity. A cell with one port state variable is locally passive at equilibrium point $Q(V_1 = \bar{V}_1)$ if, and only if, all four conditions are satisfied:

- (i) $Y_Q(s)$ has no poles in the open right plane $\text{Re}[s] > 0$.
- (ii) $Y_Q(s)$ has no multiple poles on the imaginary axis.
- (iii) If $Y_Q(s)$ has a simple pole $s = i\omega_P$ on the imaginary axis, then $K_Q(i\omega_P) \triangleq \lim_{s \rightarrow i\omega_P} (s - i\omega_P)Y_Q(s)$ must be a positive real number.
- (iv) $\text{Re}[Y_Q(i\omega)] \geq 0$ for all $\omega \in (-\infty, \infty)$ where $s = i\omega$ is not a pole.

Since all four conditions must be satisfied for $Y_Q(s)$ to be locally passive, the negation of any one of these conditions gives us the desired

Test for Local Activity of Complexity Function $Y_Q(s)$. A cell with one port state variable is locally active at equilibrium point $Q(V_1 = \bar{V}_1)$ if, and only if, any one of the following conditions is true:

- (i) $Y_Q(s)$ has a pole in the open right plane $\text{Re}[s] > 0$.
- (ii) $Y_Q(s)$ has a multiple pole on the imaginary axis.
- (iii) $Y_Q(s)$ has a simple pole $s = i\omega_P$ on the imaginary axis and $K_Q(i\omega_P) \triangleq \lim_{s \rightarrow i\omega_P} (s - i\omega_P)Y_Q(s)$ is either a negative real number, or a complex number.
- (iv) $\text{Re}[Y_Q(i\omega)] \geq 0$ for some $\omega \in (-\infty, \infty)$.

By the same procedure, one can prove the general test for local activity of complexity matrix $\mathbf{Y}_Q(s)$ for any $m \geq 2$.

The complexity matrix $\mathbf{Y}_Q(s)$ depends not only on the cell equilibrium point $Q(\mathbf{V}_a = \bar{\mathbf{V}}_a)$, but also on the cell control parameters $\boldsymbol{\mu} \in \mathbb{R}^p$. For each cell state $(Q, \boldsymbol{\mu})$, we can test whether any one of the four conditions in our Local Activity Test is satisfied. This explicit procedure can be derived analytically in simple cases, or numerically by a computer. We can partition therefore the parameter space into a locally-passive domain \mathcal{P} and a locally-active domain \mathcal{A} , over all possible cell equilibrium points corresponding to $\mathbf{V}_a \in \mathbb{R}^m$ with $\mathcal{P} \cup \mathcal{A} = \mathbb{R}^m$.

It can be proven that no reaction-diffusion equation can exhibit complexity if its cell parameters lie in the locally-passive domain \mathcal{P} ([7] 3447). The larger the size of the locally-active domain \mathcal{A} , the more chances are for the reaction-diffusion equation to exhibit complexity. Since the number of non-state variables can be increased by setting more diffusion coefficients to zero, it follows that $\mathcal{A}_1 \subset \mathcal{A}_2 \subset \dots \subset \mathcal{A}_m \dots \subset \mathcal{A}_n$, where \mathcal{A}_m denotes the local-activity domain of a cell with m port state variables, and m is equal to the number of positive diffusion coefficients. The local activity domain \mathcal{A}_m is defined as the union of all locally-active parameter domains at cell equilibrium points corresponding to all possible port state variables $\mathbf{V}_a \in \mathbb{R}^m$.

By definition, a cell is locally passive iff it is not locally active. Therefore, to prove that local activity is the origin of complexity, it suffices to prove that the reaction-diffusion equation cannot exhibit any form of complexity if the cells are locally passive. But what does complexity mean here?

Definition of Complexity. A spatially continuous or discrete medium made of identical cells which interact with all cells located within a neighborhood (called sphere of influence) with identical interaction laws is said to manifest complexity iff the homogeneous medium can exhibit a non-homogeneous static or spatio-temporal pattern, under homogeneous initial and boundary conditions.

It follows that a reaction-diffusion medium is capable of exhibiting complexity if, and only if, the corresponding continuous reaction-diffusion partial differential equations, or their discretized version, have at least one non-homogeneous static or spatio-temporal solution for some homogeneous initial and boundary conditions. The initial condition is required to be homogeneous since otherwise, we can consider a system made of only cells which are not coupled to each other, such as a system of reactive-diffusion equations with zero diffusion coefficients. This system can exhibit a non-homogeneous static pattern by choosing the initial condition to correspond to any pattern of cell equilibrium states, assuming each cell has two or more equilibrium states.

The main result is that if the cells are strictly locally passive, then all solutions of the reaction-diffusion differential equations must converge to a unique steady state as $t \rightarrow \infty$. Since the homogeneous steady state consisting of all uncoupled cells at the same equilibrium state is one such solution, it must be the only solution due to the uniqueness property. Therefore, the corresponding medium cannot exhibit any form of complexity.

The local activity domain \mathcal{A}_m is the union of four local activity parameter subsets $\mu(I)$, $\mu(II)$, $\mu(III)$, and $\mu(IV)$ of the parameter space $\mu \in \mathbb{R}^p$, each one satisfying the local activity conditions (i) – (iv) in the test for local activity of complexity matrix $\mathbf{Y}_Q(s)$. Although the first three subsets are disjoint subsets of \mathbb{R}^p , the fourth subset $\mu(IV)$ may intersect each of the other three subsets. The subset of $\mu(IV)$ which does not intersect $\mu(I)$, $\mu(II)$, or $\mu(III)$ has poles only in the open left-half plane and hence its associated cell equilibrium points are both locally active and asymptotically stable, for all port input vectors $\mathbf{I}_a \in \mathbb{R}^m$. Although reaction-diffusion equations with cell parameters chosen from this subset may exhibit complexity, the most interesting phenomena are observed from uncoupled cells with $\mathbf{I}_a = \mathbf{0}$.

Definition of the Edge of Chaos. An uncoupled cell (with $\mathbf{I}_a = \mathbf{0}$) of a reaction-diffusion equation is said to be on the edge of chaos iff all of its cell equilibrium points are locally active but asymptotically stable. The set ε of all locally active parameters $\mu \in \mathbb{R}^p$ with this property is called the edge of chaos parameter set.

The edge of chaos parameter set ε can be expressed in terms of the restricted parameter subsets with $\mathbf{I}_a = \mathbf{0}$:

$$\varepsilon = \mu(IV) \setminus [\mu(IV) \cap \mu(I)] \cup [\mu(IV) \cap \mu(II)] \cup [\mu(IV) \cap \mu(III)] \quad (20)$$

where all subsets are restricted to the uncoupled condition $\mathbf{I}_a = 0$. The area ε which does not intersect $\mu(\text{I})$, $\mu(\text{II})$, and $\mu(\text{III})$ is the edge of chaos parameter set. The restricted local activity parameter subsets $\mu(\text{I})$, $\mu(\text{II})$, $\mu(\text{III})$, and $\mu(\text{IV})$ are calculated with $\mathbf{I}_a = 0$.

3 Local Activity and Edge of Chaos of the Brusselator Equations

The Brusselator model (named after its research group in Brussels) was one of the first systems of equations used to explain self-organizing chemical reactions of the reaction-diffusion type. Based on this model, a theory of dissipative structures operating far from thermodynamic equilibrium was developed by Ilya Prigogine [3, 19, 20]. Under stability theory techniques, Prigogine and his group derived a critical bifurcation boundary for the uncoupled cell. They studied stationary and dynamic patterns emerging in the neighborhood of this boundary. But, except for the stability boundaries, the far from thermodynamic-equilibrium theory is too coarse to predict sharper and more precise domain of emergent behavior. Especially, they ignored the relatively small subset of the edge of chaos where the emergence of complexity is most likely [21, 22].

Brusselator Equations. The mathematical model of the Brusselator is defined by two partial differential equations (PDE)

$$\frac{\partial V_1(x, y)}{\partial t} = a - (b + 1)V_1(x, y) + V_2(x, y)(V_1(x, y))^2 + D_1 \nabla^2 V_1(x, y) \quad (21)$$

$$\frac{\partial V_2(x, y)}{\partial t} = b V_1(x, y) - V_2(x, y)(V_1(x, y))^2 + D_2 \nabla^2 V_2(x, y) \quad (22)$$

with two diffusion coefficients D_1 and D_2 and two state variables V_1 and V_2 characterizing the chemical dynamics. The cell parameters are denoted by a and b , the spatial coordinates are denoted by x and y . The coupling coefficients are assumed as diffusion with $D_1 \geq 0$ and $D_2 \geq 0$.

Local Activity Test of Brusselator. A local activity test can be applied in following steps of an algorithm:

1. In a first step, the Brusselator PDE is mapped into a discrete-space version with two diffusion coefficients. For the edge of chaos, it suffices to consider the zero-input current case $I_1 = I_2 = 0$.
2. In the second step, the equilibrium points Q_i with $I_1 = I_2 = 0$ are determined. In the case of a Brusselator, there is a unique equilibrium point Q_1 .
3. In the third step, the Jacobian matrix of the discretized Brusselator equations at the equilibrium point Q_1 is determined.

4. In the fourth step, the input data of the Jacobian matrix are used to classify each cell parameter point (a, b) at the equilibrium point Q_1 with a test algorithm into one of the three disjoint categories:
 - (a) *Locally Active and Stable* $S(Q_1)A(Q_1)$: Because there is only one equilibrium point of a Brusselator, this region coincides with the edge of chaos domain. The edge of chaos domain is defined as the region in the cell parameter space where the isolated cell is locally active and stable at least at one equilibrium point.
 - (b) *Locally Active and Unstable* $A(Q_1)U(Q_1)$: This region corresponds to the oscillatory or unstable region of an isolated cell.
 - (c) *Locally Passive* $P(Q_1)$: This is the region in the cell parameter space where complex phenomena are unlikely to occur in reaction-diffusion systems.

Pattern formation. Most of the parameter points found by Prigogine as examples of self-organization are located nearby the bifurcation boundary separating the stable from the unstable region. The local activity test is a general check and also identifies the ignored domain of local passivity $b \leq 1$. The boundary $b = 1$ between the local passivity and the local activity domain cannot be determined via Prigogine's linear stability analysis, because cells with parameters in both $S(Q_1)A(Q_1)$ and $P(Q_1)$ are always stable. The various static and dynamic patterns generated by the Brusselator are totally predicted by the local activity principle. Static patterns are not reduced to Turing's stationary patterns with both diffusion coefficients non zero, but also mean the emergence of patterns with only one diffusion coefficient. Some of them are located in the edge of chaos domain.

4 Local Activity and Edge of Chaos of the Gierer-Meinhardt Equations

The local activity theory is applied, again, to the Gierer-Meinhardt-equations to illustrate the emergence of complexity. On the basis of autocatalysis and lateral inhibition, Gierer and Meinhardt proposed a mathematical model [12] to explain pattern formation (morphogenesis) in living systems. Using numerical integration, they were able to produce a number of patterns relevant to the formation of biological structures. But, it is still too coarse to predict the precise domain in the cell parameter space where emergent behavior may occur. The local activity theory offers a rigorous and effective tool for sharpening existing results in the sense that it can identify more precisely those regions in the cell parameter space which are capable of emergent behaviors, and also in fine tuning such regions into a relatively small subset called the edge of chaos where the emergence of complex phenomena is most likely [14].

Gierer-Meinhardt Equations. The Gierer-Meinhardt model is described by the following system of partial differential equations (PDE) with two diffusion coefficients D_1 and D_2

$$\frac{\partial V_1(x, y)}{\partial t} = a + \frac{(V_1(x, y))^2}{V_2(x, y)} - bV_1(x, y) + D_1 \nabla^2 V_1(x, y) \quad (23)$$

$$\frac{\partial V_2(x, y)}{\partial t} = (V_1(x, y))^2 - V_2(x, y) + D_2 \nabla^2 V_2(x, y) \quad (24)$$

where V_1 and V_2 are the two state variables characterizing the cell dynamics. The cell parameters are denoted by a , and b , and the spatial coordinates are denoted by x and y . In keeping with the physical meaning of diffusion we will assume the coupling coefficients $D_1 \geq 0$ and $D_2 \geq 0$.

Local Activity Test of Gierer-Meinhardt Model. A local activity test can be applied, again, in following steps of an algorithm:

1. The Gierer-Meinhardt PDE is mapped into a discrete-space version with two diffusion coefficients. For the edge of chaos, it suffices to consider the zero-input current case $I_1 = I_2 = 0$.
2. In the second step, the equilibrium points Q_i with $I_1 = I_2 = 0$ are determined. In the case of the Gierer-Meinhardt model, it is sufficient to determine the equilibrium point Q_1 with $V_1(Q_1) = \frac{a+1}{b}$ and $V_2(Q_1) = \left(\frac{a+1}{b}\right)^2$. An equilibrium point Q_2 with $V_1(Q_2) = V_2(Q_2) = 0$ is also a solution of the system, when $I_1 = I_2 = 0$, $a = 0$ and for zero derivatives. It can easily be checked that this equilibrium is always unstable, independent of cell parameters.
3. In the third step, the Jacobian matrix of the discretized Gierer-Meinhardt equations at the equilibrium point Q_1 is determined.
4. In the fourth step, the input data of the Jacobian matrix are used to classify each cell parameter point (a, b) at the equilibrium point Q_1 with a test algorithm into one of the three disjoint categories:
 - (a) *Locally Active and Stable S* (Q_1) $A((Q_1))$: The edge of chaos domain is defined as the region in the cell parameter space where the isolated cell is locally active and stable at least at one equilibrium point.
 - (b) *Locally Active and Unstable A* (Q_1) $U(Q_1)$: This region corresponds to the oscillatory or unstable region of an isolated cell.
 - (c) *Locally Passive P* (Q_1): This is the region in the cell parameter space where complex phenomena are unlikely to occur in reaction-diffusion systems. The restricted local passivity region with $I_1 = I_2 = 0$ can be partially or totally included in the local activity region which is defined for all possible equilibrium points when $I_1, I_2 \in (-\infty, +\infty)$.

Pattern Formation. The classification of a cell parameter point into one of these three categories depends on whether there is only one diffusion coefficient, or there are two nonzero diffusion coefficients. Gierer and Meinhardt only considered selected cell parameter points. All of them lie within the edge of chaos region.

Many other examples can be determined by the local activity principle. Again, the boundary between local passivity and local activity domain cannot be determined by a linear stability analysis (in the sense of Prigogine, Gierer, and Meinhardt). The dynamics of associated computer simulations is displayed with respect to points of the parameter space. For the same cell parameter point different sets of coupling coefficients and/or initial states may lead to completely different behaviors.

5 Local Activity and Edge of Chaos of the Hodgkin-Huxley Equation

The long-lasting action and pace-maker potentials of the Purkinje fiber of the heart were first described by the Hodgkin-Huxley equations of the cardiac Purkinje fiber model of morphogenesis in [11]. The bifurcation diagrams of the corresponding computer simulations supply a possible explanation for why a heart with a normal heart-rate may stop beating suddenly: The cell parameter of a normal heart is located in a locally active unstable domain and just nearby an edge of chaos. The membrane potential along a fiber is simulated in a Hodgkin-Huxley model by a computer [23]. Computer simulations show that oscillatory patterns, chaotic patterns, or divergent patterns may emerge if the selected cell parameters are located in locally active domains but nearby the edge of chaos. This research demonstrates once again the effectiveness of the local activity theory in choosing the parameters for the emergence of complex (static and dynamic) patterns in a homogeneous lattice.

In the previous examples, the analytical criteria for testing the local activity of models with one and two local state variables have been presented. The criteria have been used to describe the bifurcation diagrams of the corresponding computer simulations, in particular finding the edge of chaos domains. In this section, the analytic criteria for testing the local activity of computer simulations with four state variables, and one diffusion coefficient are set up. After mapping the cardiac Purkinje fiber (CPF) equations to each cell of a computer simulation, which is then called the Hodgkin-Huxley cell, we choose the sodium equilibrium potential (denoted by E_{Na}) and the potassium equilibrium potential (denoted by E_K) as cell parameters for calculating the bifurcation diagrams since E_{Na} and E_K depend on the corresponding ionic concentrations which are not constants in vivo.

It is interesting to find that the cell parameter of a normal heart is located in the locally active unstable domain but nearby an edge of chaos domain. Roughly speaking, a computer simulation shows that as the values of E_{Na} and E_K are increased, the frequency of the heartbeat (corresponding to the periodic frequency of the membrane potential described via the CPF equations) also increases [11]. However the amplitude of the membrane potential decreases until the heart stops beating. Conversely as the values of E_{Na} and E_K are decreased, the frequency of

the heartbeat is also decreased until the heart stops beating. These phenomena can be explained well via the corresponding bifurcation diagrams. Extensive computer simulations show that if the chosen cell parameters are nearby the edge of chaos and are located in a locally active unstable region, the corresponding patterns may show chaotic, periodic, or unbounded characteristics.

Cardiac Purkinje Fiber (CPF) Equations. The cardiac Purkinje Fiber (CPF) equations introduced by [11] have been used to describe the action pacemaker potentials of the Purkinje fibers of the heart. The behavior of the equation corresponds quite well with the observed behavior of the Purkinje fibers. The original CPF equations have the form

$$\frac{dV}{dt} = -\frac{1}{C_m}((400m^3h + 0.14)(V - a) + 1.2 \exp((-V - 90/50) + 0.015 \exp((V + 90)/60) + 1.2n^4 + (V + b)) \quad (25)$$

$$\frac{dm}{dt} = \alpha_m(V)(1 - m) - \beta_m(V)m \quad (26)$$

$$\frac{dh}{dt} = \alpha_h(V)(1 - h) - \beta_h(V)h \quad (27)$$

$$\frac{dn}{dt} = \alpha_n(V)(1 - n) - \beta_n(V)n \quad (28)$$

where $a = E_{Na} = 40$, $b = -E_k = 100$, $C_m = 12$ and E_{Na} , E_k and C_m are sodium equilibrium potential, potassium equilibrium potential and membrane capacity, respectively. The other terms are defined as follows,

$$\alpha_m(V) = 0.1(-V - 48)/(\exp((-V - 48)/15) - 1) \quad (29)$$

$$\beta_m(V) = 0.2(V + 8)/(\exp((V + 8)/5) - 1) \quad (30)$$

$$\alpha_h(V) = 0.17(\exp((-V - 90)/20)) \quad (31)$$

$$\beta_h(V) = 1/(\exp((-V - 42)/10) + 1) \quad (32)$$

$$\alpha_n(V) = 0.0001(-V - 50)/(\exp((-V - 50)/10) - 1) \quad (33)$$

$$\beta_n(V) = 0.002 \exp((-V - 90)/80) \quad (34)$$

where V is equal to the membrane potential E minus the resting potential E_r . V is called the membrane potential.

Hodgkin-Huxley Discrete Computer Model. In the next step [11], the original CPF equations are mapped into a four-dimensional 4×30 Hodgkin-Huxley computer simulation, which is a discrete version of the CPF partial differential equation with one diffusion coefficient D_1

$$\begin{aligned}\dot{V}_{i,j} = & -\frac{1}{c_m}((400m_{i,j}^3h_{i,j} + 0.14)(V_{i,j} - a) + 1.2\exp((-V_{i,j} - 90/50) \\ & + 0.015\exp((V_{i,j} + 90)/60) + 1.2n_{i,j}^4)(V_{i,j} + b)) + D_1(V_{i+1,j} \\ & + V_{i-1,j} + V_{i,j+1} + V_{i,j-1} - 4V_{i,j})\end{aligned}$$

$$\dot{m}_{i,j} = \alpha_m(V_{i,j})(1 - m_{i,j}) - \beta_m(V_{i,j})m_{i,j} \quad (36)$$

$$\dot{h}_{i,j} = \gamma \alpha_h(V_{i,j})(1 - h_{i,j}) - \beta_h(V_{i,j})h_{i,j} \quad (37)$$

$$\dot{n}_{i,j} = \alpha_n(V_{i,j})(1 - n_{i,j}) - \beta_n(V_{i,j})n_{i,j} \quad (i = 1, 2, 3, 4; j = 1, 2, \dots, 30) \quad (38)$$

where parameters a and b are considered to be the relevant parameter space, and γ is an additional parameter to observe the effect of small disturbances on the bifurcation diagrams on the computer simulations.

In component form, these equations become

$$\dot{\mathbf{V}} = f_1(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) + D_1 \nabla^2 \mathbf{V} \quad (39)$$

$$\dot{\mathbf{M}} = f_2(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) \quad (40)$$

$$\dot{\mathbf{H}} = f_3(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) \quad (41)$$

$$\dot{\mathbf{N}} = f_4(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) \quad (42)$$

where

$$\begin{aligned}f_1(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = & -\frac{1}{c_m}((400\mathbf{M}^3\mathbf{H} + 0.14)(\mathbf{V} - a) + 1.2\exp((- \mathbf{V} - 90/50) \\ & + 0.015\exp((\mathbf{V} + 90)/60) + 1.2\mathbf{N}^4)(\mathbf{V} + b))\end{aligned}$$

$$f_2(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = \alpha_m(\mathbf{V})(1 - \mathbf{M}) - \beta_m(\mathbf{V})(\mathbf{M})$$

$$f_3(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = \alpha_h(\mathbf{V})(1 - \mathbf{H}) - \beta_h(\mathbf{V})(\mathbf{H})$$

$$f_4(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = \alpha_n(\mathbf{V})(1 - \mathbf{N}) - \beta_n(\mathbf{V})(\mathbf{N})$$

and ∇^2 corresponds to a 120×120 matrix.

Equilibrium Points of the Hodgkin-Huxley Equations. In the next step, the cell equilibrium points Q_i can only be solved numerically via the equations

$$f_1(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = 0 \quad (43)$$

$$f_2(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = 0 \quad (44)$$

$$f_3(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = 0 \quad (45)$$

$$f_4(\mathbf{V}, \mathbf{M}, \mathbf{H}, \mathbf{N}) = 0 \quad (46)$$

The cell coefficients $\alpha_{m,n}(Q_i)$ are defined via the corresponding Jacobian matrix.

Local Activity and Passivity of the Hodgkin-Huxley Model. According to the local activity and passivity criteria, local activity and passivity of a cell are characterized by certain conditions of its complexity function $Y_Q(s)$ at equilibrium point Q [10]. Sometimes, it is convenient to use the cell impedance $Z_Q(s) \triangleq Y_Q^{-1}(s)$:

A cell with one port state variable is locally active at equilibrium point $Q(\bar{V}_1, \bar{I}_1)$ if, and only if, any one of the following conditions is true:

- i. $Z_Q(s)$ has a pole in the open right plane $\text{Re}[s] > 0$.
- ii. $Z_Q(s)$ has a multiple pole on the imaginary axis.
- iii. $Z_Q(s)$ has a simple pole $s = i\omega_p$ on the imaginary axis and $K_Q(i\omega_p) \triangleq \lim_{s \rightarrow i\omega_p} (s - i\omega_p)Z_Q(s)$ is either a negative real number or a complex number.
- iv. $Z_Q^*(i\omega) + Z_Q(i\omega) < 0$ for some $\omega = \omega_0$, where ω_0 is any real number.

The locally passive domains with respect to the equilibrium points for the Hodgkin-Huxley model can be numerically calculated via computer programs. These domains with respect to two cell equilibrium points (denoted by Q_1 and Q_2) with different cell parameters were calculated numerically and can be shown in computer simulations. Since the equilibrium points Q_1 and Q_2 cannot be expressed analytically, they are obtained via simulation tools, taking different initial iterate values.

Although the results are not analytically exact, it can be concluded from the bifurcation diagrams: The cell parameter value (40, 100) of a normal heart is located in the locally active (unstable) domain with respect to the cell equilibrium points Q_1 and Q_2 , and nearby the edge of chaos with respect to the equilibrium point Q_2 . For any fixed parameter value, the bifurcation diagrams with respect to the equilibrium points Q_1 and Q_2 are quite different if the parameter b is larger than 100. Roughly speaking, our computer simulation shows that the cell equilibrium points which are located in the locally passive domains or in the edge of chaos domains with respect to the equilibrium point Q_1 seem to be globally attractive. This result may explain why a heart with an approximately normal rate can stop suddenly. A small perturbation of the parameter γ ($\pm 1.5\%$) does not cause the bifurcation diagrams to be noticeably changed. The same conclusion also holds for the case where the parameter C_m is disturbed. These facts may be interpreted as an indication of the robustness of the heart.

Applications to Cardiology. Based on the four conditions for local activity of cells, analytical criteria for systems with four state variables and one diffusion coefficient can be presented. The criteria can be easily implemented by a computer program to produce bifurcation diagrams for the corresponding Hodgkin-Huxley model. Although no chaotic phenomenon is observed, the cell parameters which cause the heart to stop beating are always located nearby the edge of chaos domains. It can be shown that the changes in the sodium equilibrium potential E_{Na} (corresponding to the parameter a) cause greater changes to the frequency of the

heartbeat than those of the potassium potential E_k (corresponding to the parameter $-b$). The parameter value $(a, b) = (58, 100)$ seems to play an extraordinary role. At this value, the frequency of the heartbeat is about 80 beats/min. However, if the parameter is changed to $(a, b) = (58.5, 100)$, the heart will stop beating. This computer simulation seems to be able to provide some insight into why it is possible that a patient with non-normal electrocardiogram but approximate normal frequency of the heartbeat might suddenly die without warning.

So-called smoothed circuit equation and the corresponding computer simulations exhibit bifurcation diagrams which show that no locally passive domain exists. Oscillatory patterns, convergent (static) patterns, and divergent (unbounded) patterns can be obtained if the parameter sets are chosen on the edge of chaos domain. In particular, emergence of complex patterns may exist if the corresponding cell parameters are chosen in the locally active unstable domain but nearby the edge of chaos domains. In summary, this research confirms once again that the local activity theory provides a practical and explicit analytical tool for determining a subset of the cell parameter space where complexity may emerge [24].

6 The Local Activity Principle of Brains and Artificial Minds

Artificial Brain of Memristors. On the horizon of future chip technology is the vision of neuromorphic computers, mimicking the human brain with billions of neurons and synaptic connections. Brains are considered complex networks with local activities of cells such as CNNs and CAs. A technical unit modeling a living neuron with synapses needs features of memories, digital circuits, and a form of analog information processing. A strong candidate fulfilling all these requirements is the memristor, a new circuit element, which was suggested [25, 26] more than 40 years ago. Modern technology assumes that the memristor will bring a new wave of innovation in electronics, packing more bits into smaller volumes and equipped with a non-volatile memory preventing the loss of data.

The memristor device has generated immense interests among both device researchers and the memory-chip industry alike [27]. These interests were due to the high potential economic impact of the HP (Hewlett-Packard) breakthrough. Since the titanium-dioxide HP memristor could be scaled down to about 1 nm and is compatible with current IC technology, many industry experts are predicting that nano memristor devices would eventually replace both flash memories and DRAMS (Dynamic Random Access Memory). Indeed, a PC with no booting time, and which remembers all data prior to unplugging the power, could become standard features within a few years.

Memristor is an abbreviation for “memory resistor” and was predicted as the fourth missing circuit element with respect to the basic equations of electric circuits [27]. These equations are defined for the four quantities voltage (v), current

(i), charge (q), and magnetic flux (φ). Each equation determines a relation between two of these variables. The simplest relation between voltage and current is Ohm's law $v = Ri$, meaning that voltage is proportional to current. The constant of proportionality is given by the resistance R . If a current of I amperes flows through a resistance of R ohms, then the voltage with respect to the resistance is v volts. Geometrically, the graph of current versus voltage for a resistor is a straight line with slope R .

There are 6 possible pairings of 4 variables. The 2 pairs (v, φ) and (i, q) are already related by definition $v = \frac{d\varphi}{dt}$ and $i = \frac{dq}{dt}$. The 3 pairs (v, i), (φ, i), and (q, v) define a resistor, inductor, and capacitor, respectively. The missing equation relating charge q and magnetic flux φ is called memristor. Obviously, the memristor was found by arguments of symmetry and completeness. There are two classes of memristors, namely, locally-passive memristors and locally-active memristors. The conventional resistor, capacitor, inductor, and the locally-passive memristor are passive circuit elements, which must be distinguished from active devices, such as transistors, which can amplify signals and inject power into circuits. However, locally-active memristors are active devices and hence can also amplify signal, with a power supply, just like transistors. The memristor is a nonlinear device defined by the graph of a curve in the flux vs. charge plane, whose slope, called the memristance, varies from one point to another [28].

A transistor is a three-terminal device with three connections to a circuit. It acts as a switch or amplifier, with a voltage applied to one terminal controlling a current flowing between the other two terminals. Although a locally-active memristor has only two terminals, it can also realize these functions. On the other hand, locally-passive memristors can be used to build both memory and digital logic without the need for a power supply. This is why they are called non-volatile memories. Metaphorically speaking, the memristor has a built-in sense of history. A signal applied at one moment can affect another signal that travels the same path later. The first signal realizes this control by setting the internal state of the memristor to high or low resistance.

Therefore, in a neuromorphic computer, locally-passive memristors would not totally supplant a transistor, but supplement them in memory functions and logic circuits. Memristors could play the role of synapses. In biological neural networks, each nerve cell communicates with other cells through thousands of synapses. An important mechanism of learning is realized by adjusting the strength of the synaptic connections. In an artificial neural network, synapses must be small, but effective structures. Locally-passive memristors satisfy all the needed requirements. They change their resistance in response to the currents that flow through them. This operation suggests a direct way of modeling the adjustment of synaptic strength.

Recall that there are two qualitatively distinct kinds of memristors, namely locally passive memristors and locally active memristors. The HP memristor is locally passive because it does not require a power supply, and is said to be

non-volatile. The potassium and sodium ion channels in the classic Hodgkin-Huxley nerve membrane circuit model can be considered locally-active memristors, powered by a sodium and a potassium pump whose energy derives from ATP molecules. In contrast, synapses are locally passive memristors capable of retaining their synaptic efficacies over long periods of time without consuming any power.

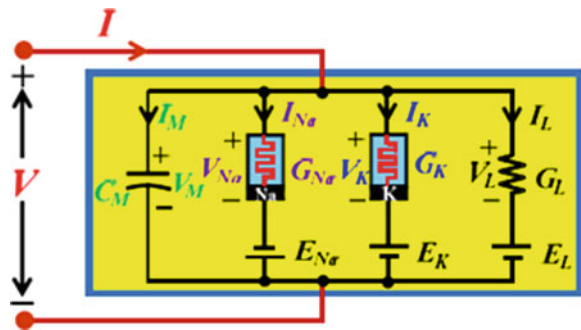
Since our brains process information using only synapses and axons, it follows that circuits made of both types of memristors should be able to emulate higher brain functions as well. The long-term potentiation (LTP) phenomenon associated with long-term memory can also be emulated by a memristor. Many associative memory phenomena, such as the Pavlovian dog behavior, can be emulated by a memristor circuit. If brains are made of memristors, then we can expect that electronic circuits made of both locally passive and locally active memristors may someday emulate human minds [29]. The key to this fundamental process is to uncover how local activity could lead to the emergence of complex patterns from a mass of homogeneous brain tissues. Formally, the local activity principle is realized in any cellular automaton. In neurons and memristors, the local activity principle is not only a formal model, but biological and technical reality.

Hodgkin-Huxley Electrical Circuit Model. The Hodgkin-Huxley electrical circuit model of a squid giant axon membrane and its associated Hodgkin-Huxley equations (henceforth referred to as HH equations) has stood the test of time and has served as a classic reference in neurophysiology and brain science research for 70 years. The squid was chosen by Hodgkin and Huxley because they are endowed with enormous axons, the largest of them in a large Atlantic squid (*Loligo pealii*) being as much as one millimeter in diameter ([30, 31, 32, 33, 34]).

In the Hodgkin-Huxley electrical circuit model, I , I_{Na} , I_K , and I_l denote the external axon membrane current, the sodium ion current, the potassium ion current, and the leakage current, respectively. Likewise, E , E_{Na} , E_K , and E_l denote the membrane capacitor voltage, the sodium ion battery voltage, the potassium ion battery voltage, and the leakage battery voltage, respectively. The basic assumption Hodgkin and Huxley made is that the squid giant axon can be modeled by a distributed circuit consisting of a line of identical 2-terminal electrical devices (henceforth referred to as HH cells) described by the HH Circuit Model, and coupled by identical passive resistors. By assuming the HH cells to be physically small and taking the limit as the length $\Delta x \rightarrow 0$, the dissipative couplings tend to a Laplacian, thereby modeling the standard diffusion mechanism. The equations describing the entire system then tends to a system of reaction diffusion equations in one spatial variable.

The HH Circuit Model [31, 35] contains 7 circuit elements that must be specified before it is possible to formulate the equations governing the circuit. These elements and their parameter values must be determined by meticulous experiments. For convenience in their measurement setups, Hodgkin and Huxley had opted to measure all voltages with respect to the resting potential E_r , whose value depends on the axon specimen and measurement temperature.

Fig. 1 Memristive Hodgkin-Huxley axon circuit model [10]



But, the time-varying resistances R_K and R_{Na} (resp., time-varying conductances g_K and g_{Na}) assumed by Hodgkin and Huxley are fundamentally wrong from a circuit-theoretic and scientific perspective. In particular, the two elements R_K and R_{Na} (resp., g_K and g_{Na}) in their HH axon Circuit model belong to the fundamentally different class of time-invariant circuit elements of memristors. After Hodgkin-Huxley's mis-identification error is corrected, all of the anomalies and confusions ([8, 23]) concerning anomalous inductances, rectification, frequency-dependent parameters, and the fundamental mechanisms responsible for the generation of action potentials can be clearly resolved in a simple and rigorous manner.

Memristive Hodgkin-Huxley Axon Circuit Model. Substituting the Hodgkin-Huxley time-varying potassium resistance R_K , and sodium resistance R_{Na} by the potassium ion-channel memristor and the sodium ion-channel memristor, respectively, we obtain the memristive Hodgkin-Huxley Axon Circuit Model. Observe that this circuit model contains only well-defined time-invariant circuit elements, as expected of any realistic physical model of the axon. With basic circuit theory, the Hodgkin-Huxley DC circuit model in Fig. 1 can be derived by simply deleting the axon membrane capacitor C_M , since $I_M = C_M \frac{dV_M}{dt} = 0$ at DC. For each $V = V_m = V_m(Q)$ at an equilibrium point $Q(V_m, I_m)$, where $V = V_m$ and $I = I_m$ denote the axon membrane voltage and membrane current in Fig. 1, respectively, we can substitute the potassium ion-channel memristor by its small-signal circuit model, about the corresponding DC equilibrium point at $V_K = V_m(Q) - E_K = V_m(Q) - 12 \text{ mV} \triangleq V_K(Q_K)$. Similarly, we can substitute the sodium ion-channel memristor by its small-signal circuit model, about the corresponding DC equilibrium point at $V_{Na} = V_m(Q) + E_K = V_m(Q) + 115 \text{ mV} \triangleq V_{Na}(Q_{Na})$.

By recognizing that the two circuit elements R_K and R_{Na} in the Hodgkin-Huxley axon circuit model are not time-varying, but are rather time-invariant memristors, we were able to provide a firm circuit-theoretic foundation for analyzing, interpreting, and explaining various anomalous phenomena and paradoxes reported in the literature more than 70 years ago and which had remained unresolved [31]. For example, Hodgkin [31, 32] was quite shocked to find the small-signal impedance they measured from the axon membrane of squids had exhibited a positive reactance that suggested the presence of a gigantic inductance and an enormous

magnetic field in the squid axon. This inexplicable phenomenon had since been referred to in the literature as an anomalous impedance [8]. But the terms in the Hodgkin-Huxley equations pertaining to the time varying potassium conductance G_K is in fact a first-order memristor. Similarly, the terms pertaining to the time-varying sodium conductance G_{Na} is in fact a second-order memristor. The historical Hodgkin-Huxley axon circuit model should therefore be replaced henceforth by the memristive Hodgkin-Huxley axon circuit model shown in Fig. 1.

Since the two memristors in the Hodgkin-Huxley Axon Circuit Model are time-invariant nonlinear circuit elements, we can exploit the local activity theory to uncover the nonlinear dynamical potentials of these two circuit elements. Classic circuit-theoretic concepts as small-signal admittance, small-signal impedance, pole-zero diagrams, etc. can be applied. All of these intrinsic linear circuit characterizations and their explicit analytical formulas can be derived from the memristive Hodgkin-Huxley axon circuit model. The circuit-theoretic properties represent definitive characterizations of the Hodgkin-Huxley Axon. They play a fundamental role in the research on the dynamics of ion channels.

The main theorem [16] asserts that the zeros of the scalar function $Y(s, V_m) \triangleq \frac{1}{Z(s, V_m)}$ called the small-signal admittance of the Hodgkin-Huxley memristor circuit model, are identical to the eigenvalues of the 4×4 Jacobian matrix of the HH equation, calculated at the equilibrium point $V_m = V(I_{ext})$ of the HH equations, for each constant DC excitation current I_{ext} . Here $s = \sigma + i\omega$ denotes the complex variable associated with the Laplace transform $\hat{v}(s)$ of a time function $v(t)$ [16] and I_{ext} denotes an external current source applied to the Hodgkin-Huxley axon circuit model shown in Fig. 1. In chapter 2 it was explained that this theorem is valid not only for the 4-dimensional HH equation, but for any system of n differential equations. This theorem is a powerful tool because instead of calculating the eigenvalues of a high dimensional $n \times n$ matrix, one only has to calculate the roots (zeros) of a scalar polynomial equation of a single variable s , for any integer n .

Most deep insights concerning local activity and edge of chaos can be uncovered from an analysis of the linearized differential equations about the equilibrium points of its associated nonlinear dynamical system. The edge of chaos is typically a very small subset of the local activity domain. So small and yet so profound is the edge of chaos domain that we often dramatize its significance by dubbing it the pearl of local activity. Indeed, edge of chaos is the source of life, and we will show that neurons are poised near this pearl of local activity. It is rather enigmatic that while all complexity phenomena, including the generation of spikes, require strongly nonlinear dynamics, yet the mathematical genesis of such global phenomena is strictly local. The theory of local activity and edge of chaos is based entirely on linearized differential equations about an equilibrium point. Testing an equilibrium point Q for local activity in general, or edge of chaos in particular, involves examining the linearized Hodgkin-Huxley equations about Q .

Linearized Hodgkin-Huxley Equations. For each equilibrium point Q corresponding to a DC excitation current I_{ext} , let us superimpose an infinitesimally

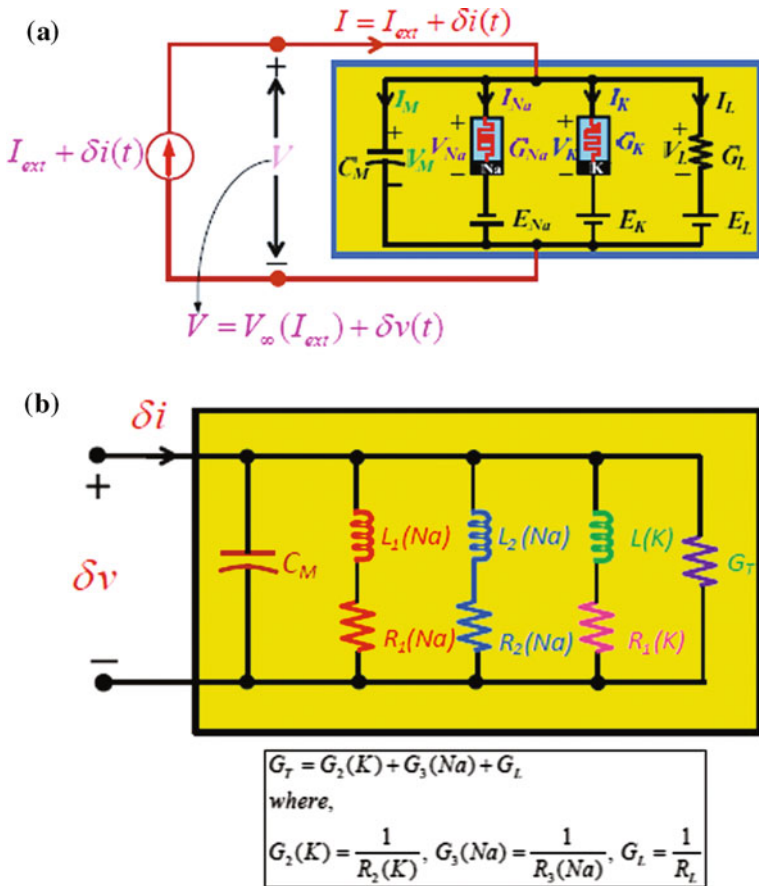


Fig. 2 Hodgkin-Huxley axon circuit model (a) and its linearized small-signal equivalent Hodgkin-Huxley circuit (b) [8]

small current signal $\delta i(t)$ and apply the composite signal $I_{ext} + \delta i(t)$ to the HH axon circuit model Fig. 2a. Whether a system (in this case the Hodgkin-Huxley axon) is locally active at Q or not is completely determined by the response $\delta v(t)$ to an infinitesimally small sinusoidal testing signal $\delta i(t) = A \sin \omega t$, where A denotes the amplitude, and $\omega = 2\pi f$ denotes the angular frequency. It follows from elementary circuit theory that the response $\delta v(t)$ to any small-signal current excitation $\delta i(t)$ can be predicted analytically from a small-signal equivalent circuit whose elements are calculated explicitly from the Jacobian matrix of the associated nonlinear differential equations, evaluated at the equilibrium point Q . Such an equivalent circuit is presented in Fig. 2b.

Complexity Function of Hodgkin-Huxley Equations. In general (chapter 2), the complexity function $C(s)$ for a single-input single-output system is defined by

the ratio between the Laplace transform of the output variable $\hat{y}(t)$ and the Laplace transform $\hat{u}(s)$ of the input variable $u(t)$, namely,

$$C(s) = \frac{L(y(t))}{L(u(t))} = \frac{\hat{y}(s)}{\hat{u}(s)}. \quad (47)$$

In Fig. 2a, $u(t) = \delta i(t)$ and $y(t) = \delta v(t)$, respectively. Testing for local activity and edge of chaos of an equilibrium point Q of the HH axon circuit in Fig. 2a at any $I = I_{ext}$ requires that we examine the complexity function defined by

$$Z(s) = \frac{\hat{v}(s)}{\hat{i}(s)}, \quad (48)$$

where $\hat{v}(s) = L(\delta v(t))$ and $\hat{i}(s) = L(\delta i(t))$ denote the Laplace transform of $\delta v(t)$ and $\delta i(t)$, respectively. The complexity function $Z(s)$ is called the impedance function in circuit theory. The impedance functions $Z(s)$ for the small-signal equivalent HH circuit in Fig. 2b has been derived in [16] and is reproduced below:

$$Z(s, V_m) = \frac{a_3 s^3 + a_2 s^2 + a_1 s + a_0}{b_4 s^4 + b_3 s^3 + b_2 s^2 + b_1 s + b_0}. \quad (49)$$

The formulas for calculating the 4 coefficients a_0, a_1, a_2, a_3 in the numerator and the 5 coefficients b_0, b_1, b_2, b_3, b_4 in the denominator of $Z(s)$ are listed in [16].

According to the test criteria of local activity and edge of chaos in previous chapter, we extract and rephrase only the key aspects that are essential for Hodgkin-Huxley model [8]:

Local Activity Theorem. It is impossible to generate a spike train unless the memristive Hodgkin-Huxley one-port in Fig. 2a is locally active at some equilibrium point.

Edge of Chaos Theorem. A locally asymptotically stable-equilibrium point Q of the Hodgkin-Huxley equation is poised on the edge of chaos if, and only if, $\text{Re } Z(i\omega, V_m(Q)) < 0$ at some frequency ω . $\text{Re } Z$ denotes the real part of the complex number Z . $Z(i\omega, V_m(Q))$ is the impedance function calculated at $s = 0 + i\omega$.

From action potentials to mental states. In general, it is assumed that all mental states are correlated to corresponding cell assemblies. But, they are not only sets of firing neurons, but hierarchical systems of neural subsystems of subsystems with different depth and degrees of complexity. Research hypothesis means that the corresponding cell assemblies must empirically be identified by observational and measuring instruments. In brain reading, for example, active cell assemblies correlated with words and corresponding objects can be identified. A single neuron is not decisive and may differ among different persons. There are typical distribution patterns with fuzzy shapes which are represented in computer simulations. Brain research is still far from observing the activities of each neuron in a brain. Nevertheless, the formal hierarchical scheme of dynamics allows the explanation of complex mental states like, for instance, consciousness. Conscious

states mean that persons are aware of their activities. Self-awareness is realized by additional brain areas monitoring the neural correlates of these human activities (e.g., perceptions, feeling, or thinking). Thus, even consciousness is no mysterious event, but observable, measurable, and explainable in this research framework. In the next step, the formal hierarchical model offers the opportunity to build corresponding circuits and technical equipments for technical brains and robots with these abilities.

Traditional terms “intelligence”, “mind”, “consciousness” etc. are historically overloaded with many meanings depending on different point of views, experience and historical positions. Therefore, their meaning depends on our definitions. Concerning intelligence, a simple working definition is suggested which does not depend on “human intelligence” (in the sense of Turing’s AI-test). A system is called “intelligent” depending on its ability to solve problems. In that sense, a tick has a certain degree of intelligence, because it can solve the problem of finding blood. But, simple technical systems (e.g., a chip) can also have certain degrees of “intelligence”, because there can solve certain problems. Thus, in philosophical terms, this position sympathizes with the pluralism of Leibniz who distinguished degrees of intelligence in nature instead of Descartes’ dualism who believed in a “substance” called “intelligent mind” which was reserved to human beings. In that sense, there are already many intelligent functions of, e.g., robots [8].

Obviously, patterns of cell assemblies in the brain are not identical with our perceptions, feeling, and thinking. But, it is well confirmed in modern brain research that neural patterns of firing cells are correlated with mental states. “Mental states” can be defined and computationally modeled in state and parameter spaces with associated dynamical systems which allow us to test our models. With the technology of brain reading, an analysis of cell assemblies even allows us to recognize their represented meaning (e.g., pictures, words, phrases): Of course, there are only the first steps of research, but it seems to be possible at least in principle. Concerning computer science, semantics is technically realized in first steps and to certain degrees in a restricted and well-defined sense.

Motory, cognitive, and mental abilities are stored in synaptic connections of cell assemblies. A hard core of synaptic network is already wired, when a mammal brain is born. But many synaptic connections are generated during growth, experience and learning phase of mammals. Firing states of neurons with repeated action potentials enforce synaptic connections. Thus, during a learning phase, a cell assembly of simultaneously firing neurons creates a synaptic network storing the learnt information. Learning phases can be modeled computationally by learning algorithms [8]. As we all know, the learnt information can be forgotten, when learning is not repeated and the synaptic connections decay. Thus, on the microlevel, brain dynamics is determined by billions of firing and not firing neurons, and, on the macrolevel, by emerging and changing cell assemblies of neural networks coding different neural information.

The efficiency of neural networks depends on their number of hierarchical layers. They enable the brain to connect different neural states of, e.g., visual, haptic, and auditive information. But, there are also layers monitoring perceptual

procedures and generating visual consciousness: A person is aware and knows that she perceives something. Even our emotions depend on specified neural networks which are connected with all kinds of brain activity. It is a challenge of brain research to identify the involved layers and networks of the brain during all kinds of mental and cognitive activities.

From action potentials to semantic understanding. Semantic understanding is made possible by hierarchical layers and learning procedures of brain dynamics. In formal semantics, a formal language gets its meaning by mapping its formal expressions onto expressions of another formal language. In computer science, several layers of symbolic languages are also used to generate semantic meaning and digital procedures. Natural languages of users refer to an advanced computer language. In a next step, a kind of behavioral language describes the functions that are intended to be executed in the system. They relate their arguments typically like mathematical functions. Then, an architectural description is needed that describes the available resources, the communication and the control. The relation between the behavioral description and the architecture is done by binding elementary functions to resources and scheduling them in time (a task that can be done by a compiler or an assembler). Finally, the architecture gets realized by a register transfer layer which is close to digital circuits that in term are realized by electrical components, voltages and currents. Each such layer has its own descriptive language, dynamics and syntactic rules. Semantics is provided by interaction between layers. The digital circuit provides the semantics for the electrical circuit (vice versa, the circuit layer provides timing information to the digital), the register transfer to the digital layer, the architecture to the register transfer layer and the behavioral description sits on top of that all, at least five layers of semantic abstractions, each expressed in a dedicated formal syntax. It is only because engineers can make such a strong taxonomy and abstraction of the semantic layers, that a digital system is understandable for the human user and the digital machine. The communication between man and machine is realized in an intermediate transfer process between layers.

The human brain has a much more complex architecture which, until nowadays, cannot be completely reconstructed in clearly distinguished layers. Obviously, the brain is not the design of an architect or engineer with specific technical purposes, but the result of a more or less random and blind evolution during millions of years. Thus, several functions of tissues and networks are still unknown, although topological layers can be identified and used for explanations of semantic processes. In the end, the brain should be totally scanned and modeled from its single neurons, synapses, and action potentials to cell assemblies, networks, and layers, in order to model the whole dynamics on the micro- and macrolevel. The “machine level” is already well known and described by the digital behavior of firing and non-firing neurons with emerging action potentials. The edge of chaos domains could clearly be identified in parameter spaces of the Hodgkin-Huxley equations. They are the origin of all kind of brain dynamics with attractors of neural states correlated with human cognitive and intelligent activities.

Compared with human brains, technical systems may be restricted, but they are sometimes much more effective with their specific solutions of cognitive and intelligent tasks. In computer science, semantic webs and i-phones can already understand questions to some extension and even answer in natural languages. The technology of applied (speech analysis) algorithms may be different from biological procedures which were developed during evolution. But, they solve the problem to some degree with their computer power, high speed, parallelism and storage which can be improved in the future. Human beings are hybrid complex systems with additional abilities (e.g., imaginations, feelings) which are always involved in our semantic acting. But, it cannot be excluded that these abilities could also be managed in a future technology.

7 Local Activity and Computational Universe

Can pattern formation in our universe be understood in the analytic framework of mathematical equations or is it too complex? Instead of the traditional analytical approach of mathematical physics, Steven Wolfram declared computer experiments with pattern formation of cellular automata as “new kind of science” (NKS). It is obviously a great merit of NKS to highlight the experimental approach in the computational sciences [6]. But we claim that even in the future quasi-empirical computer experiments are not sufficient [10]. Cellular automata must be considered complex dynamical systems in the strictly mathematical sense with corresponding equations and proofs. In short, we also need analytical models of cellular automata, in order to find precise answers and predictions in the universe of cellular automata. In this sense, the analytical approach goes beyond Wolfram’s NKS.

In the analytical approach, cellular automata (CA) are defined as complex dynamical systems. Their (difference or differential) equations allow precise definitions of a complexity index and universal symmetries. It can be proved that the 256 one-dimensional cellular automata are classified by local and global symmetry classes of cellular automata. There is an exceptional symmetry group with universal computability which we called the “holy grail” in the universe of cellular automata [10]. Many analytical concepts of complexity research (e.g., attractors, basin of attractors, time series, power spectrum, fractality) are defined for cellular automata. The local activity of cells leads to pattern formation which can be analytically determined and completely classified in a rigorous mathematical manner.

Summing up all these insights, we are on the way to conceive the universe as a computational and dynamical system. The success of this research program depends on the digitization of physics. The question “*Is the Universe a computer*” leads to the question: How far is it possible to map the laws of physics onto computational digital physics? [36] Digitization is not only exciting for answering philosophical questions of the universe. Digitization is the key paradigm of modern research and technology. Nearly all kind of research and technical

innovation depend on computational modeling. The emerging complexity of nature and society cannot be handled without computers with increasing computational power and storage.

In order to make this complex computational world more understandable, cellular automata are an educational tool. NKS and the analytical approach show that many basic principles of the expanding universe and the evolution of life and brain can be illustrated with cellular automata. The emergence of new structures and patterns depends on phase transitions of complex dynamical systems in the quantum, molecular, cellular, organic, ecological, and societal world [1, 37]. Cellular automata are recognized as an intuitive modeling paradigm for complex systems with many useful applications [38]. In cellular automata, extremely simple local interactions of cells lead to the emergence of complex global structures. The local principle of activity is also true in the world of complex systems with elementary particles, atoms, molecules, cells, organs, organisms, populations, and societies [8, 10]. Although local interactions generate a complex variety of being in the universe, they can be mathematically reduced to some fundamental laws of symmetry [39]. From a philosophical point of view, we cannot be sure in principle that computational and digitalized physics described the world completely, but its explanatory and predictive power is, until nowadays, overwhelming.

References

1. K. Mainzer, *Thinking in Complexity. The Computational Dynamics of Matter, Mind, and Mankind*, 5th edn. (Springer, Berlin, 2007)
2. E. Schrödinger, *What is Life? The Physical Aspect of the Living Cell & Mind and Matter* (Cambridge University Press, Cambridge, 1948)
3. I. Prigogine, *From Being to Becoming* (Freeman, San Francisco, 1980)
4. H. Haken, *Synergetics, An Introduction*, 3rd edn. (Springer, New York, 1983)
5. C.G. Langton, Computation at the edge of chaos. Phase transitions and emergent computation. *Physica D* **42**, 12–37 (1990)
6. S. Wolfram, *A New Kind of Science* (Wolfram Media, Champaign, 2002)
7. L.O. Chua, Local activity is the origin of complexity. *Inter. J. Bifurcat. Chaos* **15**(11), 3435–3456 (2005)
8. K. Mainzer, L.O. Chua, *Local Activity Principle. The Cause of Complexity and Symmetry Breaking* (Imperial College Press, London, 2012)
9. A.M. Turing, The chemical basis of morphogenesis. *Philos. Trans. Roy. Soc. London, B* **237**, 37–72 (1952)
10. K. Mainzer, L.O. Chua, *The Universe as Automaton. From Simplicity and Symmetry to Complexity* (Springer, Berlin, 2011)
11. D. Noble, A modification of the Hodgkin-Huxley equations applicable to Purkinje fibre action and pacemaker potentials. *J. Physiol.* **160**, 317–352 (1962)
12. A. Gierer, H. Meinhardt, A theory of biological pattern formation. *Kybernetik* **12**, 30–39 (1972)
13. H. Haken, H. Olbrich, Analytical treatment of pattern formation in the Gierer-Meinhardt model of morphogenesis. *J. Math. Biol.* **6**, 317–331 (1978)
14. R. Dogaru, L.O. Chua, Edge of chaos and local activity domain of the Gierer-Meinhardt CNN. *Inter. J. Bifurcat. Chaos* **8**(12), 2321–2340 (1998)

15. A. Mauro, Anomalous impedance, a phenomenological property of time-variant resistance. An analytic review. *Biophys. J.* **1**, 353–372 (1961)
16. L.O. Chua, V. Sbitney, and H. Kim, Neurons are poised near the edge of chaos. *Inter. J. Bifurcat Chaos* (forthcoming) (2012)
17. S. Smale, A mathematical model of two cells via Turing's equation. *Lect. Appl. Math.* **6** (American Mathematical Society) 15–26 (1974)
18. L.O. Chua, Passivity and complexity. *IEEE Trans. Circ. Syst.* **46**(1), 71–82 (1999)
19. I. Nicolis, *Prigogine. Exploring Complexity* (W.H. Freeman, New York, 1989)
20. R.J. Field, R.M. Noyes, Oscillations in chemical systems IV. Limit cycle behavior in a model of a real chemical reaction. *J. Chem. Phys.* **60**, 1877–1884 (1974)
21. R. Dogaru, L.O. Chua, Edge of chaos and local activity domain of the Brusselator CNN. *Inter. J. Bifurcat. Chaos* **8**(6), 1107–1130 (1998)
22. L. Min, K.R. Crounse, L.O. Chua, Analytical criteria for local activity and applications to the Oregonator CNN. *Inter. J. Bifurcat. Chaos* **10**(1), 25–71 (2000)
23. K.S. Cole, *Membranes, Ions and Impulses* (University of California Press, Berkeley, 1972)
24. L. Min, K.R. Crounse, L.O. Chua, Analytical criteria for local activity of reaction-diffusion CNN with four state variables and applications to the Hodgkin-Huxley equation. *Inter. J. Bifurcat. Chaos* **10**(6), 1295–1343 (2000)
25. D.B. Strukov, G.S. Snider, D.R. Duncan, R.S. Williams, The missing memristor found. *Nature* **453**, 80–83 (2008)
26. L.O. Chua, Memristor—the missing circuit element. *IEEE Trans. Circ. Theor.* **18**, 507–519 (1971)
27. B. Hayes, The memristor. *Am. Sci.* **9**(2), 106–110 (2011)
28. L.O. Chua, Resistance switching memories are memristors. *Appl. Phys. A* **102**(4), 765–783 (2011)
29. J. Mullins, Memristor minds: The future of artificial intelligence. *New Scientist* **7**(2009)
30. A.L. Hodgkin, A.F. Huxley, Currents carried by sodium and potassium ions through the membrane of the giant axon of Loligo. *J. Physiol.* **116**, 449–472 (1952)
31. A.L. Hodgkin, A.F. Huxley, B. Katz, Ionic currents underlying activity in giant axon of the squid. *Arch. Sci. Physiol.* **3**, 129–150 (1949)
32. A.L. Hodgkin, R.D. Keynes, Experiments on the injection of substances into squid giant axons by means of microsyringe. *J. Physiol. London* **131**, 592 (1956)
33. R. FitzHugh, Mathematical models of excitation and propagation nerve, in *Biological Engineering*, ed. by H. Schwan (McGraw-Hill, New York, 1969), pp. 1–85
34. J.Z. Young, Structure of nerve fibres and synapses in some invertebrates. *Cold Spring Harb. Symp. Quant. Biol.* **4**, 1–6 (1936)
35. R. Dogaru, L.O. Chua, Edge of chaos and local activity domain of the FitzHugh-Nagumo equation. *Inter. J. Bifurcat. Chaos* **8**(2), 211–257 (1998)
36. D. Deutsch, Quantum theory, the church-turing principle and the universal quantum computer. *Proc. Roy. Soc. London A* **400**, 97–117 (1985)
37. K. Kaneko, *Life: An Introduction to Complex Systems Biology* (Springer, Berlin, 2006)
38. A.G. Hoekstra, J. Kroc, P.M.A. Sloot (eds.), *Simulating Complex Systems by Cellular Automata* (Springer, Berlin, 2010)
39. K. Mainzer, *Symmetries in Nature*, (De Gruyter, Berlin, 1996) (German: *Symmetrien der Natur*, De Gruyter, Berlin, 1988)

How Nature Works

Complexity in Interdisciplinary Research and
Applications

Zelinka, I.; Sanayei, A.; Zenil, H.; Rössler, O. (Eds.)

2014, VIII, 290 p., Hardcover

ISBN: 978-3-319-00253-8