Quasilocality Effect in Active Media

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Abstract—In this paper, we consider the problem of living tissue self-regulation as an example of general self-organization phenomena during adaptation and functioning of active hierarchical systems of different nature without common control center. A model of living tissue self-regulation, including two aspects, is developed. The first is the hierarchically organized data self-processing via matter conservation laws in blood flows. The second is the mechanism of the response of individual vessels to a corresponding information part expressed by the activator concentration in blood. It was shown that the necessary property of the self-regulation process, i.e., the locality of the living tissue response to an external stimulus, is mainly a consequence of the hierarchical system of blood vessels and appears at a sufficiently general form of the dependence of the vessel response to the activator concentration.

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1. Introduction. Vital functions of highly organized biological systems are accompanied by continuous consumption of nutrients necessary for maintaining and functioning of their elements. These nutrients are mostly supplied to the system in a centralized way; before they reach each element, their flow is multiply branched in a multilevel hierarchical transport network. In this case, the transport network mission is to supply only such a nutrient amount that is necessary for efficient functioning of a given element under varying conditions. This is precisely the essence of the self-regulation process whose mechanism is still not unambiguously clear. The point is that there is no common control center in many such systems, which could be responsible for nutrient distribution. Therefore, the fundamental problem is the realization of this distribution on the basis of some self-organization processes during the cooperative interaction of all transport network elements.

Let us consider a concrete example of the process under discussion, which is the living tissue self-regulation. Cells are supplied with nutrients, oxygen and are freed from waste products via the blood vessel network; blood also controls the tissue temperature. In other words, in the case of nutrient deficiency or under critical conditions of any tissue element, functioning is normalized exactly by local changes in blood flows (see, e.g., [1]). In particular, during local heating of tissue, the blood flow in the heating region can ten times increase; in other regions, an increase in the blood flow will not be so significant [2]. It should be noted that cooperative self-regulation (i.e., the self-regulation without a common control center) plays a key role in the formation, evolution, and adaptation of not only living organisms, but also in a wide class of ecological [3–6], economic [7–9], social, and technological [10–12] systems.

In [13–16], the idea of the mechanism of cooperative self-regulation was formulated and the existence of the so-called ideal self-regulation was found. The latter means that in principle there exist such systematic features in the system response to changes in external conditions, that its transport network can ideally control states of elements, i.e., supply nutrients in such amounts and there where it is required, not disturbing vital functions of neighboring elements.

The proposed self-regulation mechanism includes two components. The first is the self-processing of the data on the system state on the basis of conservation laws of matter flows over the transport network. The second is the model of the response of individual elements of the transport network to a corresponding information part. Such a mechanism of living tissue self-regulation will be concretized below. Unfortunately, the ideal self-regulation model requires such properties of the response of transport
network elements, which cannot be implemented in practical systems. This study is therefore devoted to self-regulation in active hierarchical systems whose transport network is not ideal. We present the model of a peripheral blood network in which the blood flow distribution is described by hydrodynamic laws, and the response of vessels to a change in the living tissue state is based on an increase in the concentration of special chemical substances, i.e., activators, in blood (such a response mechanism is described in detail and substantiated in [17]). The goal of this study is to demonstrate the fact that the ideality of the response of living tissue vessels is not a necessary condition for localizing the system response in the direct stimulus region.

2. Living tissue and its spatial organization model. The considered spatial organization of the living tissue is based on the following assumptions. The individual topological structure of arterial and venous beds is shaped as a tree, i.e. a circuit-free graph. The vessel branching is symmetric, i.e., characteristic parameters of e.g. smaller arteries derived in branching of a larger artery, are identical. Vessels of each hierarchy level are uniformly distributed in a considered living tissue region. Moreover, arterial and venous channels are formally considered as mirror reflections of each other in properties and states of corresponding arteries and veins. Thus, the blood pressure at the level of capillaries (the lowest vessels of these beds), i.e., the pressure at the arterial bed output and at the venous bed input is constant. This assumption is accepted to simplify the mathematical description but yields an adequate result for the actual blood circulation system [17]. Figure 1 schematically shows the accepted model of the system of blood vessels and their hierarchical description.

The blood flow \( \{v\} \) over the vessel network shown in Fig. 1 is described by the following set of variables. The blood flow through the vein \( v \) of level \( n \) is characterized by its intensity \( J_v \) (blood flow) and the activator concentration \( \theta_v \). The state of a given vein is controlled by its hydrodynamic resistance \( R_n(\theta_v) \) as a function of \( \theta_v \). The appearance of this dependence and its shape are, respectively, a consequence and a mathematical description of the local self-regulation of living tissue at the level of an individual vessel.

The blood flow distribution over the vein network is controlled by the pressure \( \{P_k\} \) at vein confluence nodes \( \{k\} \); i.e., the vein blood flow intensity \( J_v \) in vein \( v \) is related to the blood pressure at the vein input \( P_{\text{in}} \) and output \( P_{\text{out}} \) by the Poiseuille law,

\[
J_v R_n(\theta_v) = P_{\text{in}} - P_{\text{out}}. \tag{1}
\]

At nodes \( \{k\} \), the blood and activator substance conservation laws are satisfied,

\[
J_{\text{in},1} + J_{\text{in},2} = J_{\text{out}}, \tag{2}
\]

\[
J_{\text{in},1} \theta_{\text{in},1} + J_{\text{in},2} \theta_{\text{in},2} = J_{\text{out}} \theta_{\text{out}}. \tag{3}
\]

These equations are also illustrated in Fig. 1. Equations (1)–(3) are complemented by “boundary” conditions. The pressure at the inputs to veins of the last level \( N \) (venules) is supposed to be set and constant \( P \), and the pressure at the output of the zero-level vein (root vein) is supposed to be zero,

\[
P_{\text{in}|v_N} = P, \quad P_{\text{out}|v_0} = 0. \tag{4}
\]

The living tissue self-regulation is performed via a change in the hydrodynamic resistance \( \{R_n(\theta_v)\} \) of vessels (an increase or decrease in their diameter), depending on the activator concentration \( \{\theta_v\} \) in blood in these vessels.

We will use the dependence

\[
R_n(\theta_v) = R_{0,n} \cdot \phi(\theta_v), \tag{5}
\]

where \( R_{0,n} \) is the hydrodynamic resistance of vessels of level \( n \) under normal conditions of living tissue functioning, and the function \( \phi(\theta_v) \) describing the vessel response \( v \) is, for simplicity, supposed identical for all vessels. The dependence of the hydrodynamic resistance \( R_{0,n} \) of vessels on the hierarchy level \( n \) under normal conditions is set from the fact that pressure difference over the peripheral blood circulation system beds is distributed relatively uniformly over all hierarchy levels [1]. Considering this feature, we will use the following dependence

\[
R_{0,n} = (2\zeta)^n R_{0,n=0}. \tag{6}
\]